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THE ANNALS OF THORACIC SURGERY

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Thirty-eighth Annual Meeting of the Southern Thoracic Surgical Association
November 7–9, 1991, Hyatt Regency Grand Cypress, Orlando, Florida (Page 586)

Proceedings of CURRENT CONTROVERSIES AND TECHNIQUES IN CONGENITAL HEART SURGERY *Meeting (Page 591)*

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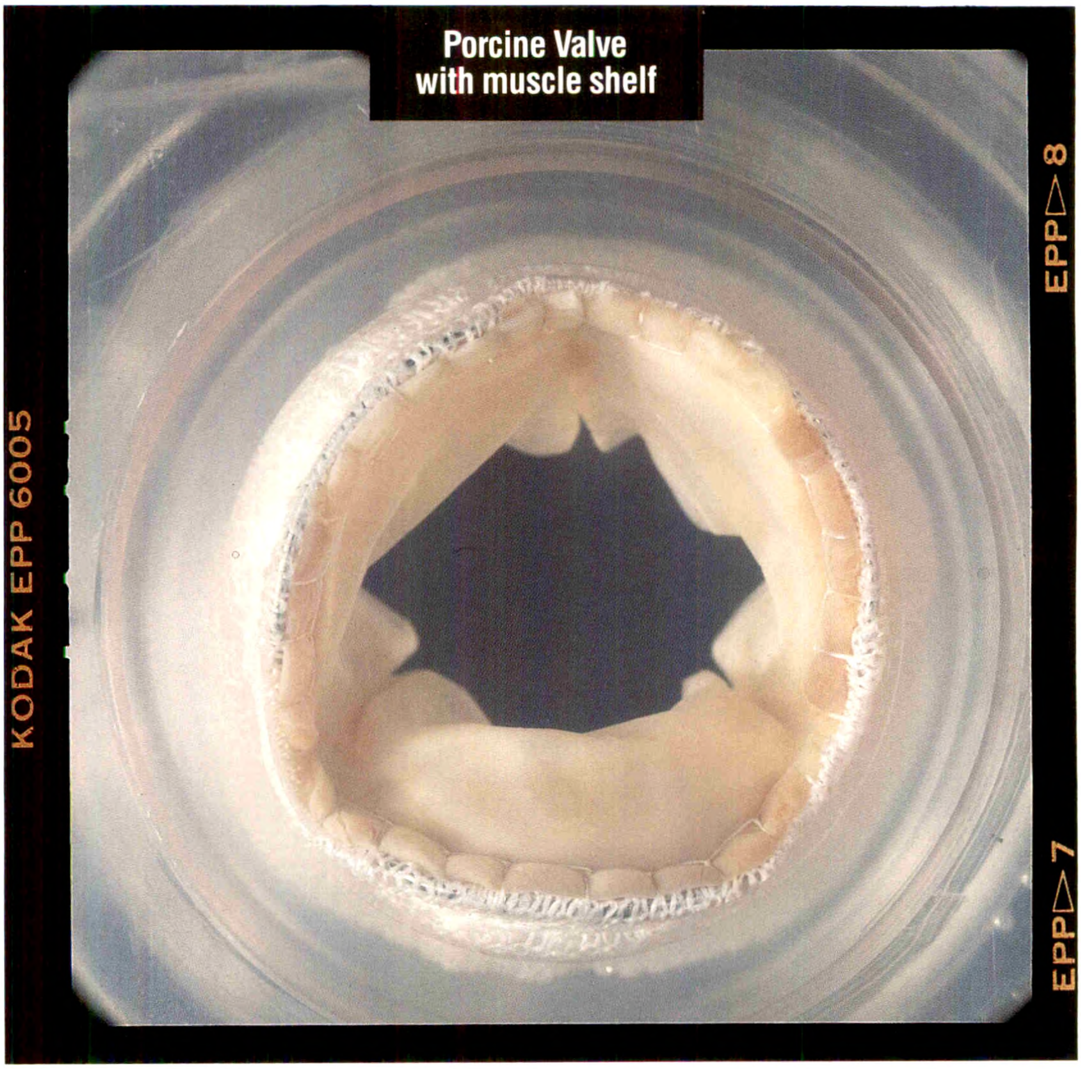

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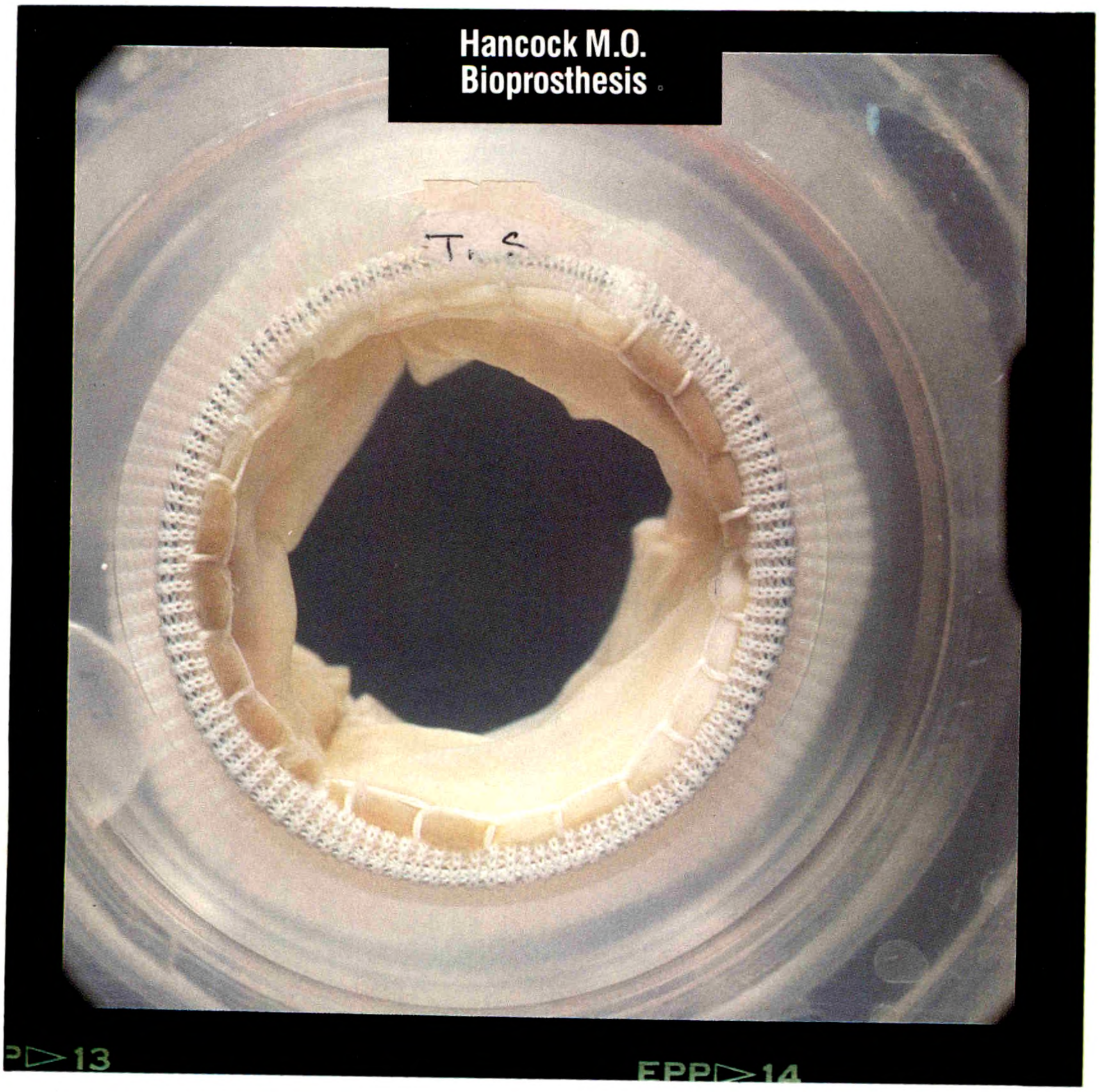

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Reference: 1. Kahn SS, Mitchell RS, Derby GC, Oyer PE, Miller DC. Differences in Hancock and Carpentier-Edwards Porcine Xenograft Aortic Valve Hemodynamics: Effect of Valve Size. *Circulation.* 1990; 82(suppl 4):117-124.

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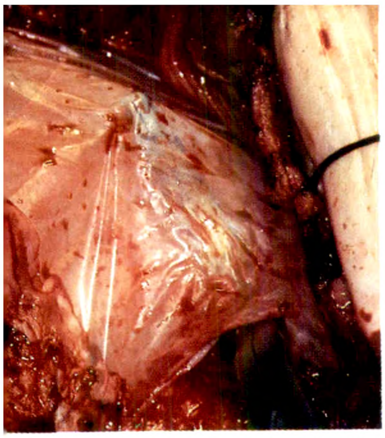

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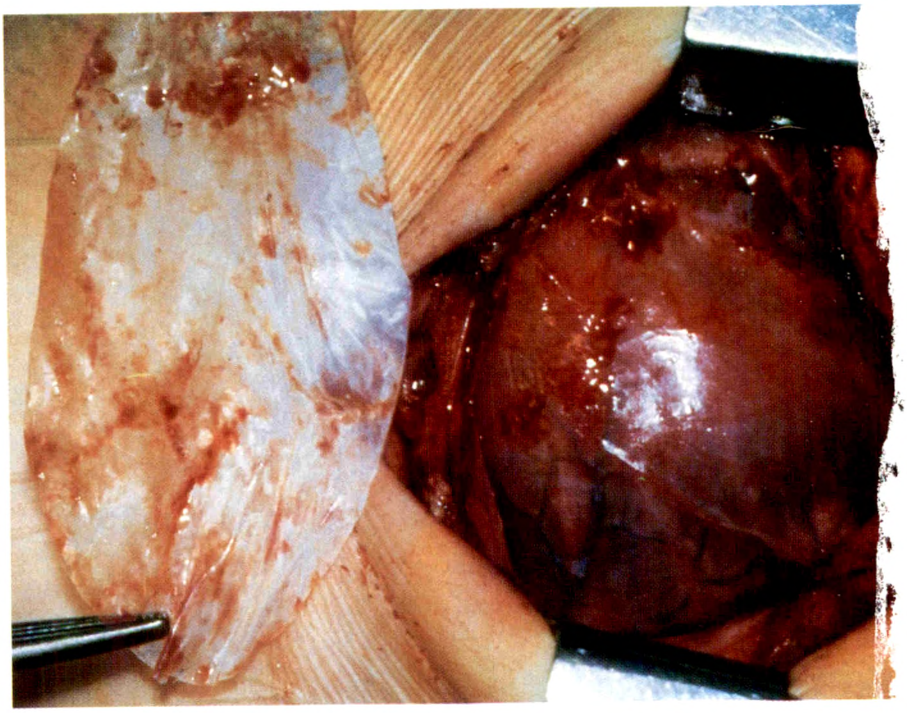

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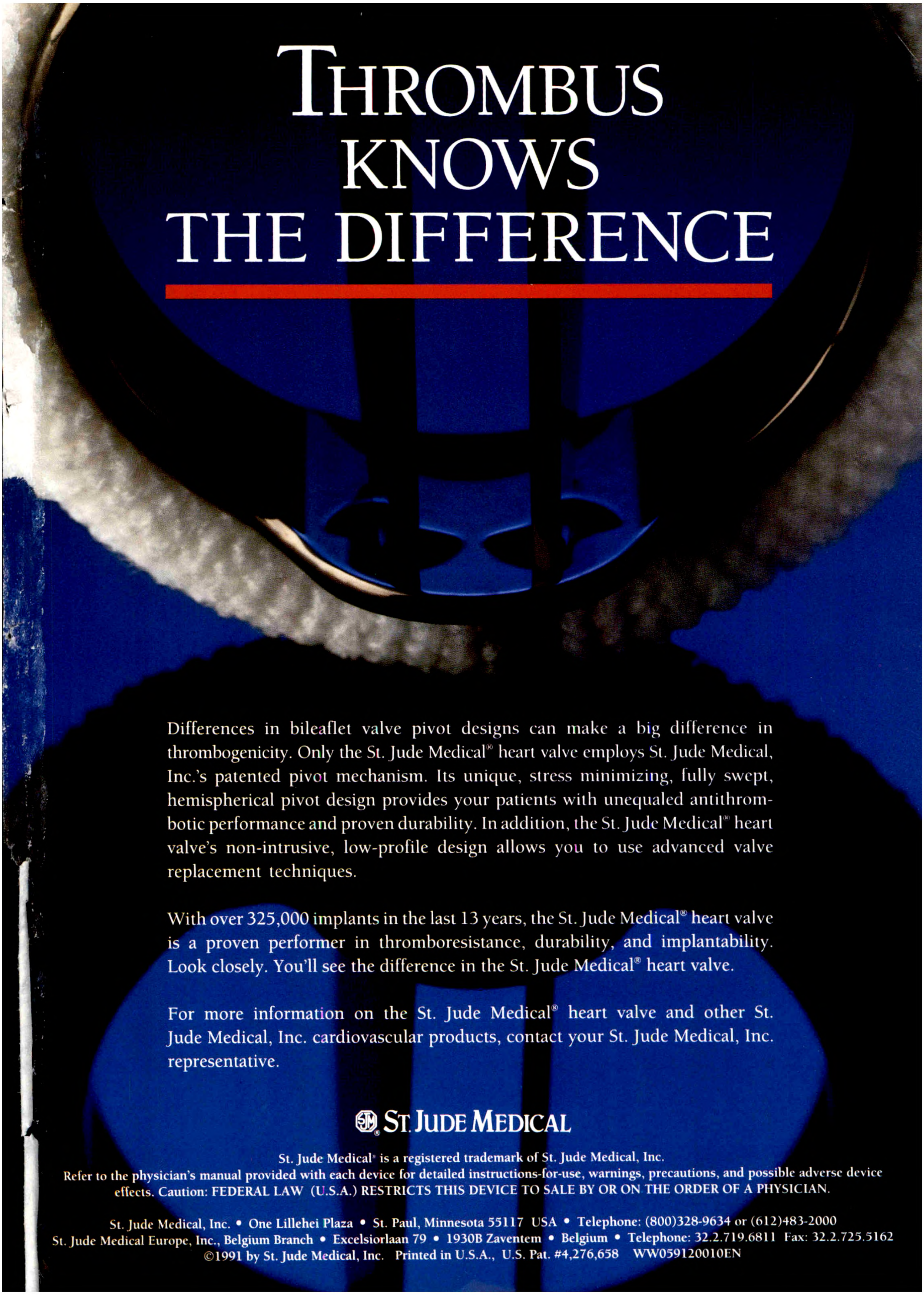

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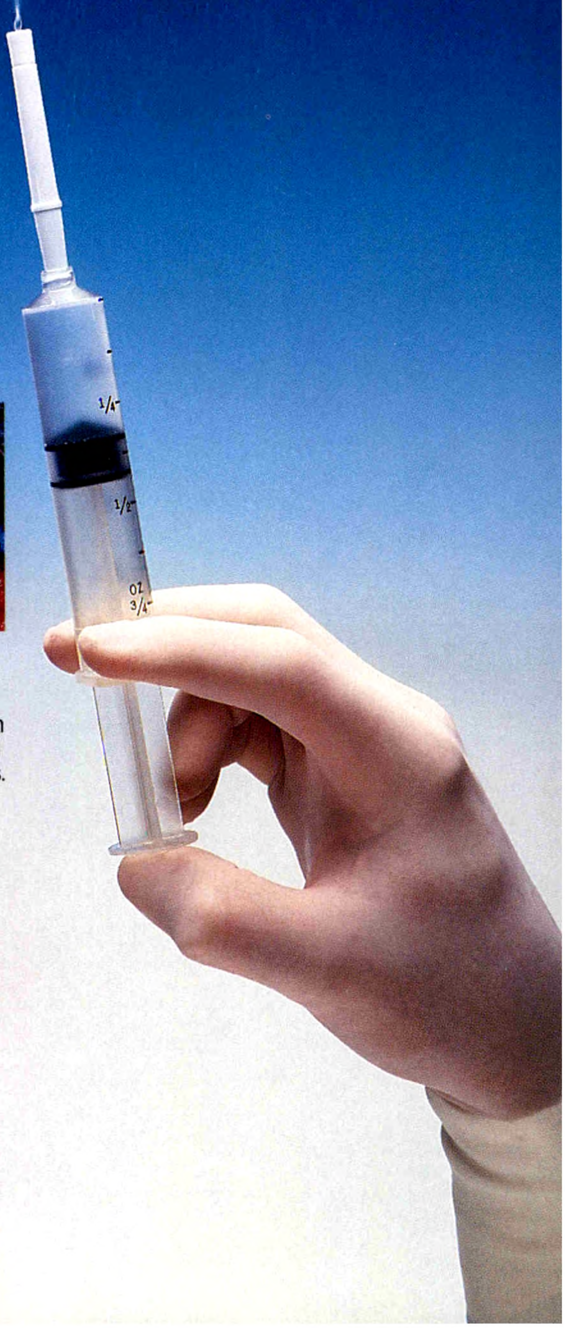

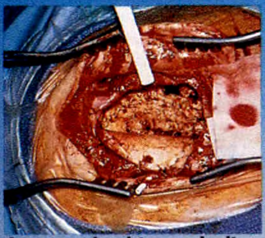

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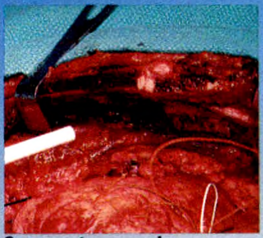

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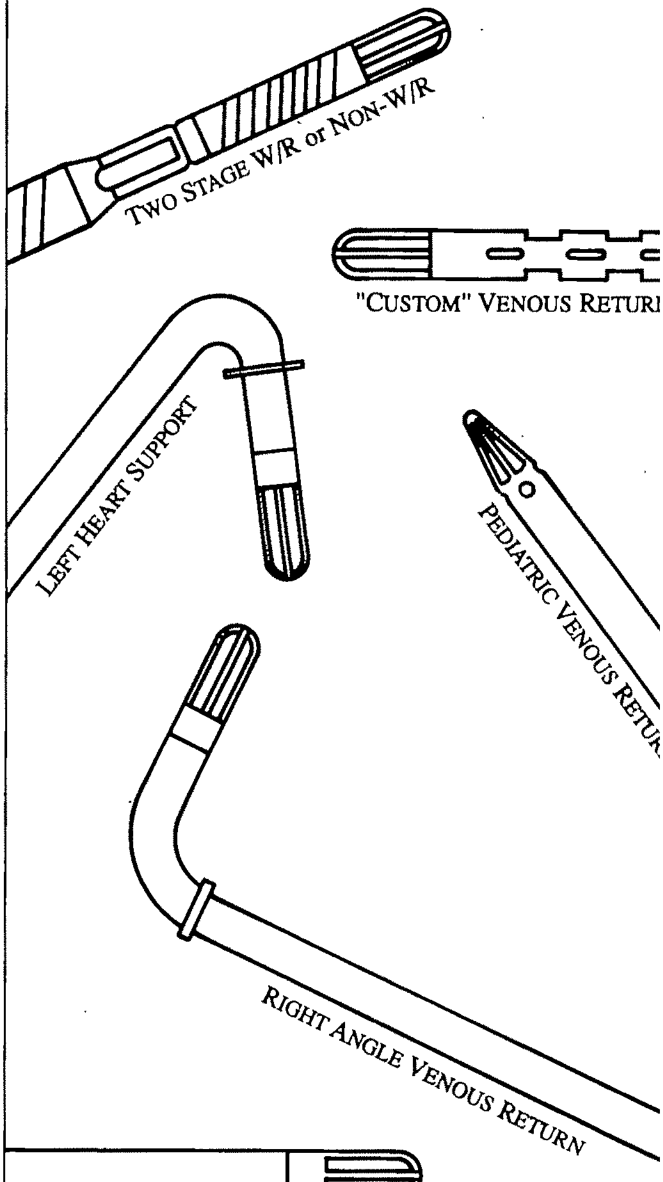

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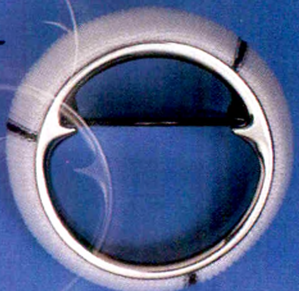

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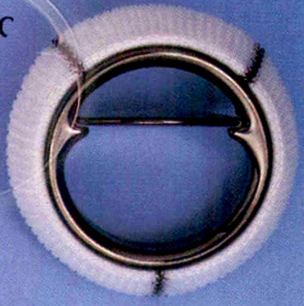

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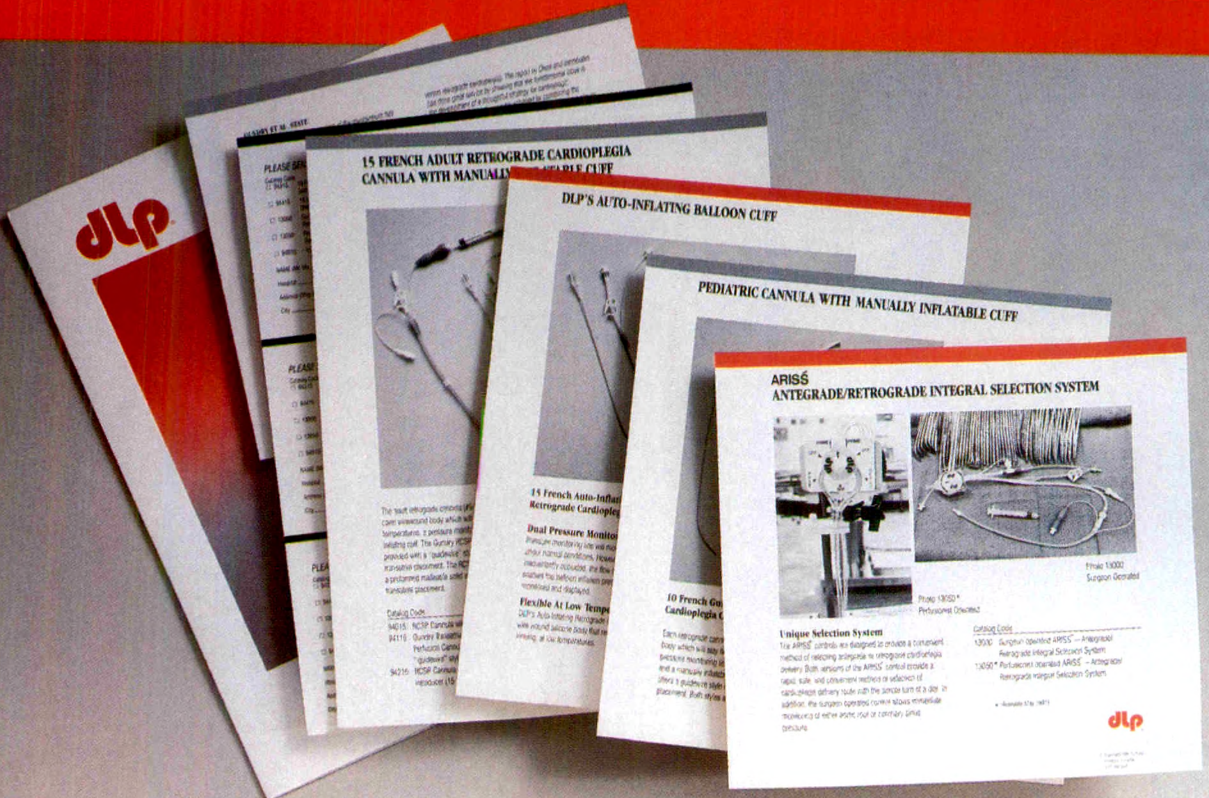
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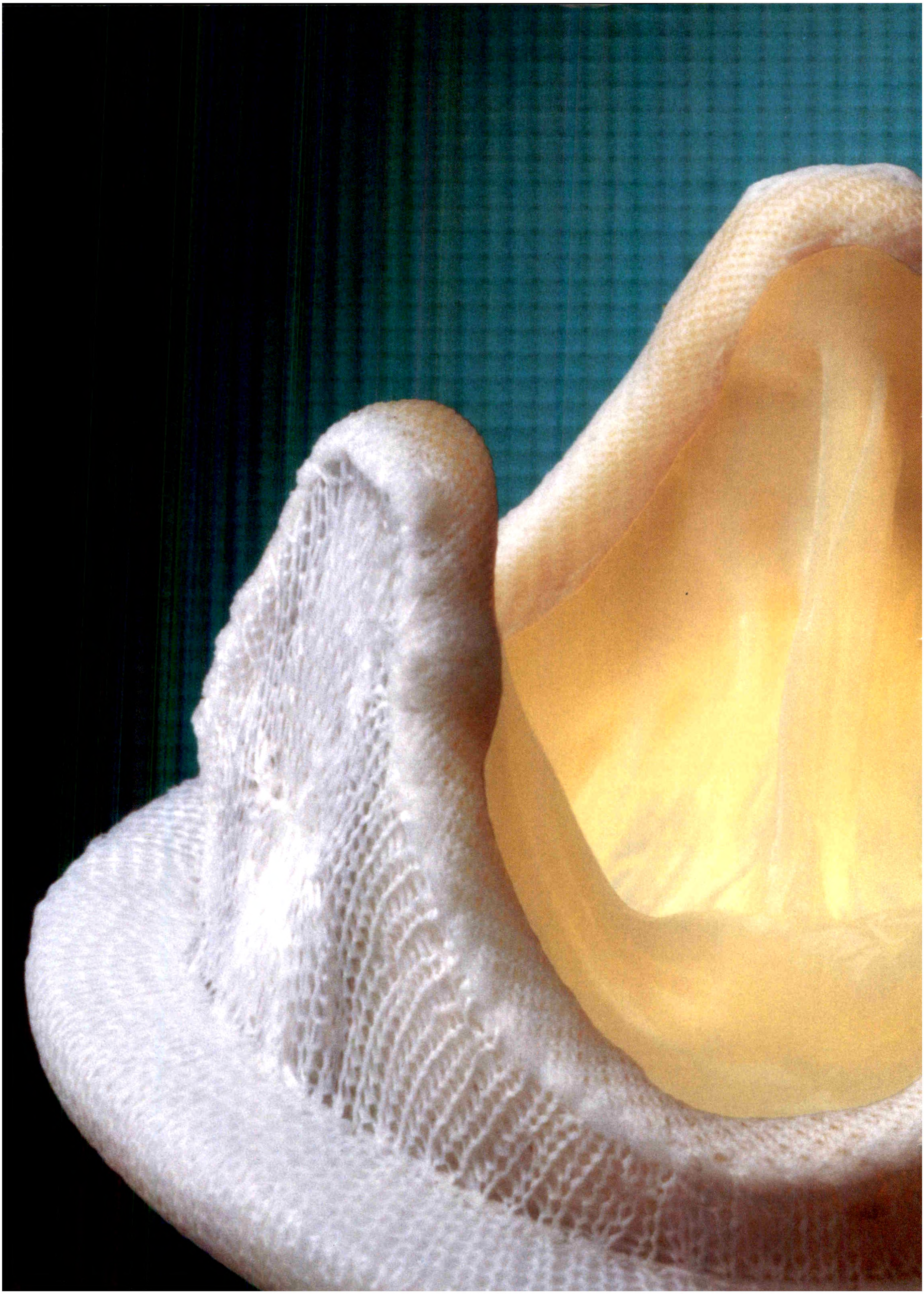

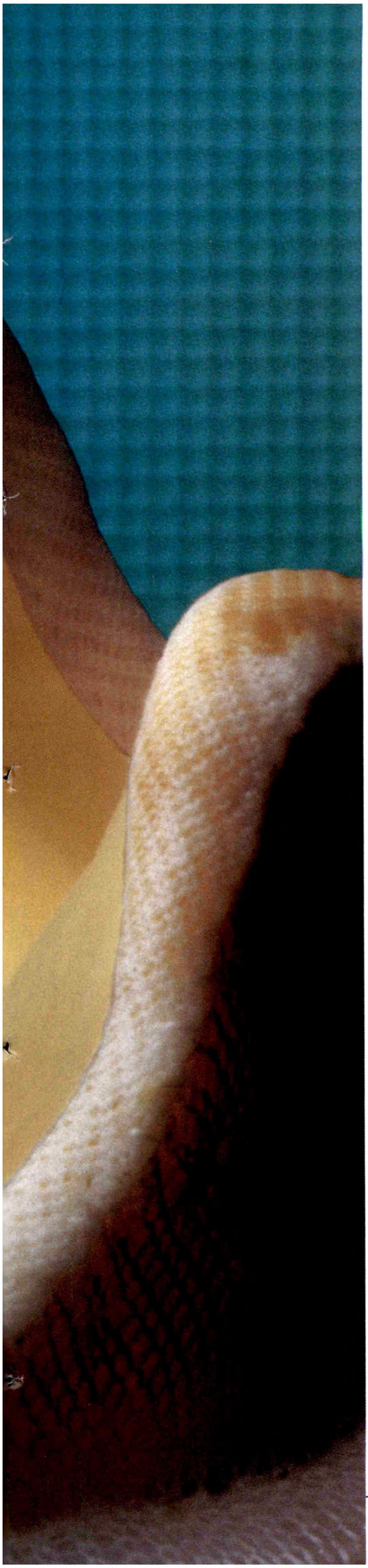

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The Carpentier-Edwards bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of their natural valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis. **Warnings:** For Single Patient Use Only. In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, in the presence of severe systemic hypertension, and in patients who are on maintenance hemodialysis. The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed. Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are sterile. The outside of the jar is not sterile and must not be placed in the sterile field. Furthermore, the valve and container should never be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions:** 1. When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. 2. Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. 3. Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. 4. The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. 5. When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. 6. Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. 7. As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. 8. The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. 9. In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. 10. A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. 11. Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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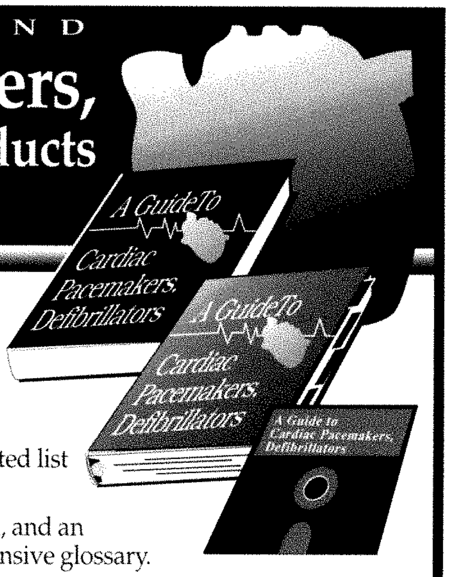
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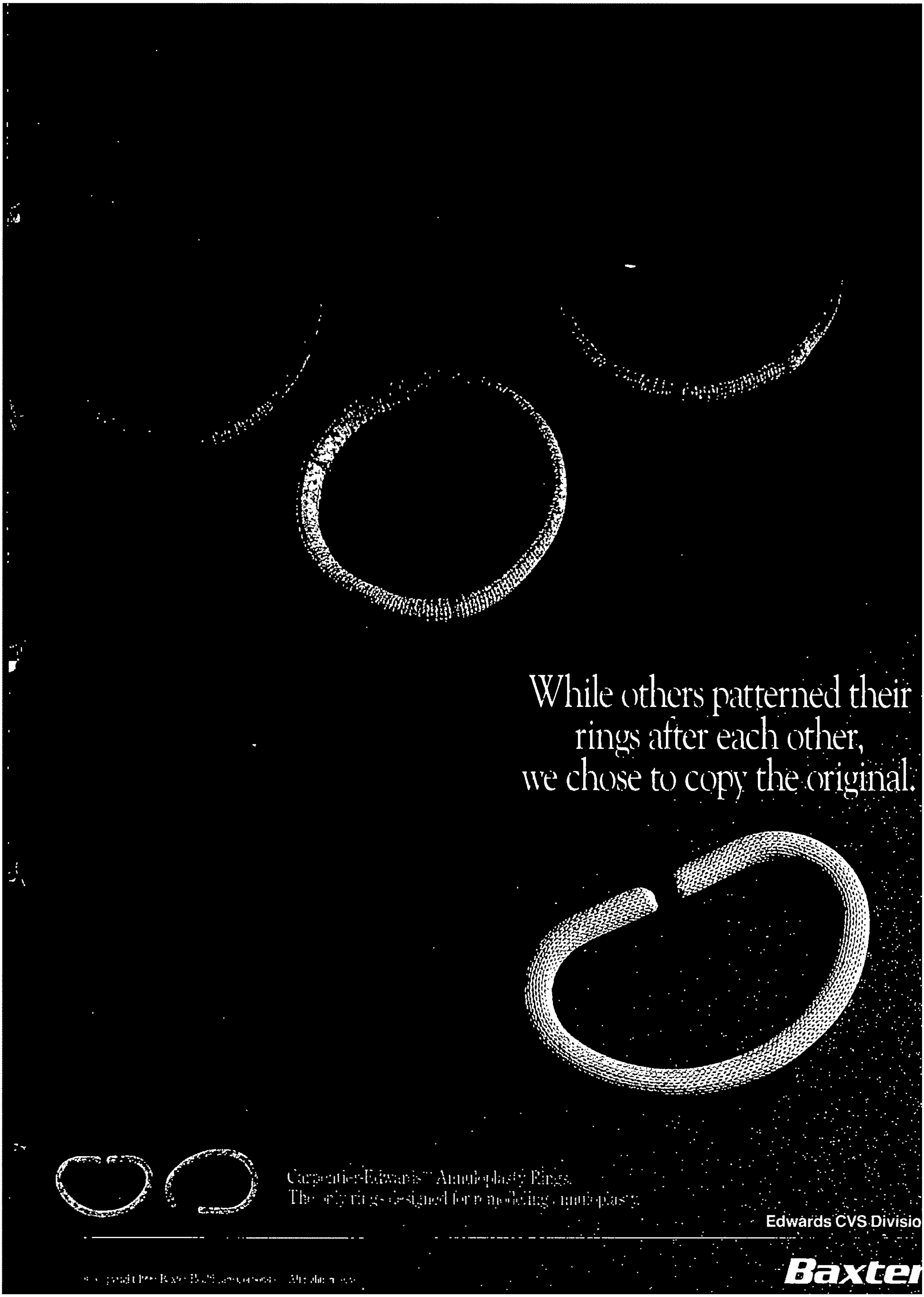

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Michel N. Ilbawi, MD, Serafin Y. DeLeon, MD, William R. Wilson, Jr, MD, Jose A. Quinones, MD, David A. Roberson, MD, Tarek S. Husayni, MD, Otto G. Thilenius, MD, PhD, and Rene A. Arcilla, MD

Angioscopic Evaluation of Intravascular Morphology After Coronary Endarterectomy
Bruce E. Keogh, MD, FRCS, Benjamin P. Bidstrup, FRACS, FRCS, Kenneth M. Taylor, MD, FRCS, and Ralph N. Sapsford, ChM, FRCS

Extended Vertical Transatrial Septal Approach to the Mitral Valve
Gerard M. Guiraudon, MD, John G. Ofiesh, MD, Raj Kaushik, MD, Douglas G. McLellan, MA, and Donna M. Cade, AHT

Fibrin Sealant Improves Surgical Results of Type A Acute Aortic Dissections
J. R. Séguin, MD, J.-M. Frapier, MD, P. Colson, MD, and P. A. Chaptal, MD

Lung Cancer Model for Study of the Metastatic Process
William G. Hammond, MD, Raymond L. Teplitz, MD, and John R. Benfield, MD

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Douglas J. Mathisen, MD, Hermes C. Grillo, MD, John C. Wain, MD, and Alan D. Hilgenberg, MD

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Todd K. Rosengart, MD, Nael Martini, MD, Pierre Ghosn, MD, and Michael Burt, MD, PhD

Multiple Reoperative Coronary Artery Bypass Grafting
Kevin D. Accola, MD, Joseph M. Craver, MD, William S. Weintraub, MD, Robert A. Guyton, MD, and Ellis L. Jones, MD

Repair of Truncus Arteriosus in Infancy
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Ultrasonic Surgical Aspirator for Lung Resection
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Jeongryul Lee, MD, Yongjin Kim, MD, Yongsoo Yoon, MD, Joonryang Rho, MD, and Kyungphill Suh, MD

Cardiac Transplantation in a Patient With a Single Ventricle and Transposition of the Great Vessels
Wayne E. Richenbacher, MD, Shreekanth V. Karwande, MD, Robert E. Shaddy, MD, Dale G. Renlund, MD, Michael R. Bristow, MD, John B. O'Connell, MD, and William A. Gay, Jr, MD

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John A. Rousou, MD, Richard M. Engelman, MD, Joseph E. Flack III, MD, and David W. Deaton, MD

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John A. Meyer, MD

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Cardiac Tumors: Clinical Experience and Surgical Results in 74 Patients
Albert Miralles, MD, Luis Bracamonte, MD, Halim Soncul, MD, Roberto Diaz del Castillo, MD, Rama Akhtar, MD, Valeria Bors, MD, Alain Pavie, MD, Iradj Gandjbackhch, MD, and Christian Cabrol, MD

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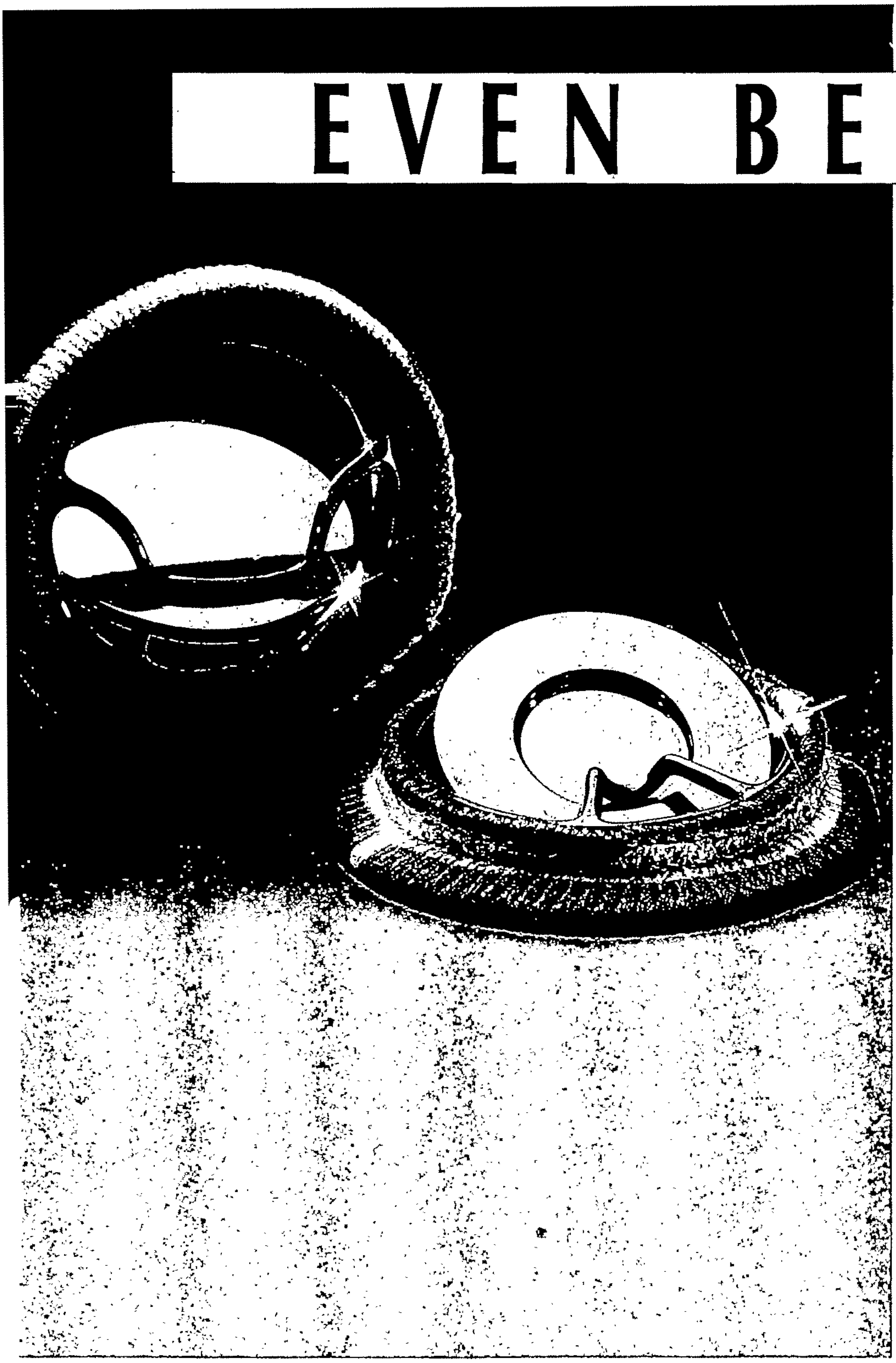

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Arterial Switch for Palliation of Subaortic Stenosis in Single Ventricle and Transposition: No Mean Feat!

Robert M. Freedom, MD, FRCP(C), and George A. Trusler, MD, FRCS(C)

Departments of Pediatrics and Surgery, University of Toronto Faculty of Medicine, and Divisions of Cardiology and Cardiovascular Surgery, The Hospital for Sick Children, Toronto, Ontario, Canada

The clinical and pathological evidence is now persuasive that subaortic stenosis is frequently a consequence of palliative pulmonary banding for double-inlet left ventricle (and other forms of univentricular atrioventricular connection of left ventricular type) and transposition of the great arteries [1–4]. Although subaortic stenosis usually follows pulmonary artery banding, clearly subaortic stenosis can and does complicate the clinical course in a few patients with double-inlet left ventricle and transposition of the great arteries and naturally occurring pulmonary outflow tract stenosis. In hearts with double-inlet left ventricle (or classic tricuspid atresia) and concordant or normal ventriculoarterial connections, the morphological substrate for subaortic stenosis is remarkably diminished (though occasionally still present).

See also page 420.

But whether pulmonary artery banding in the patient with double-inlet left ventricle and transposition is causal as a primary event, or whether this procedure merely accelerates the tendency of the moderate-sized muscular ventricular septal defect to spontaneous diminution in size is not the issue [3, 4]. Rather there is now compelling evidence that myocardial hypertrophy is a risk factor for Fontan operation, and the two factors commonly promoting myocardial hypertrophy in these hearts are banding of the pulmonary arterial trunk and its usual consequence: subaortic stenosis [5–7]. Data from The Toronto Hospital for Sick Children indicated that in the majority of patients dying after a Fontan procedure, abnormal myocardial hypertrophy was a conspicuous feature [7]. Seliem and colleagues [6] have measured left ventricular wall mass in a cohort of patients undergoing Fontan operation. Those patients with a good clinical course after Fontan operation had a wall mass significantly less than those patients whose Fontan operation ended in death, Fontan takedown, or chronic heart failure.

We warned some time ago that pulmonary artery banding should be used with caution in those patients with double-inlet left ventricle and transposition of the great arteries because of the very high association with progressive narrowing of the ventricular septal defect and subaortic stenosis [1, 3, 4, 7, 8]. Franklin and colleagues [9] have now extended these caveats, suggesting that pulmonary artery banding is contraindicated in those neonates and young infants with double-inlet ventricle, discordant ventriculoarterial connections, and an obstructive anomaly of the aortic arch as subaortic stenosis is an inevitable consequence of this procedure. If one eschews pulmonary artery banding for the neonate with double-inlet left ventricle, transposition of the great arteries, and excessive pulmonary blood flow and pulmonary artery hypertension, palliative algorithms concluding in Fontan operation have included either a Norwood-like approach or enlargement of the restrictive ventricular septal defect and other procedures [8, 10]. Clearly both of these maneuvers can and will effectively treat subaortic stenosis. However, enlargement of the ventricular septal defect is more hazardous in the neonate and the young infant, and despite important information about the course of the conduction tissue in these hearts, surgical heart block is still a reality. Furthermore, malattachment of atrioventricular valve tissue about the margins of the ventricular septal defect may make surgical enlargement impossible or hazardous to atrioventricular valve function. The fate of the pulmonary valve and potential for growth of the proximal aortopulmonary connection are concerns of the Norwood approach, although the extensive experience of Norwood and colleagues in the treatment of the hypoplastic left heart syndrome has addressed these issues. Regurgitation through the pulmonary valve is usually very mild and of little clinical significance, and there is every indication that growth of the proximal aortopulmonary anastomosis occurs. Yet the Mayo Clinic has abandoned the Damus-Kaye-Stansel connection because of distortion of the pulmonary valve and the resulting semilunar insufficiency [11].

A new and innovative approach to the palliation of the neonate with double-inlet left ventricle, transposition of the great arteries, and excessive pulmonary blood flow with pulmonary artery hypertension is demonstrated effectively by Karl and associates in this issue of *The Annals* [12]. The arterial switch maneuver has been used previously in combination with a right atrium to right ventricle valved conduit to treat somewhat older patients with complex forms of univentricular heart, including double-inlet left ventricle, transposition of the great arteries, banded pulmonary trunk, and real or potential subaortic stenosis [13]. The application of the arterial switch operation to the standard form of double-inlet left ventri-

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Table 1. Palliative Operations for Subaortic Stenosis

Type of Palliation	Potential Complication
PA–aortic connection (Damus-Kaye-Stansel)	Distortion of pulmonary valve; pulmonary incompetence
Enlargement of VSD	Heart block; potential distortion of AV valve tissue; recurrence of VSD
Arterial switch operation	Pulmonary arterial obstruction; supraaortic obstruction; myocardial ischemia reflecting distortion of coronary artery
LV–aortic conduit	Ventricular dysfunction

AV = atrioventricular; LV = left ventricle; PA = pulmonary artery; VSD = ventricular septal defect.

cle or to the patient with tricuspid atresia, both forms with discordant ventriculoarterial connections, excessive pulmonary blood flow, and pulmonary artery hypertension, creates templates where subaortic stenosis is far less common. Indeed, subaortic stenosis is almost unknown in the patient with tricuspid atresia and concordant ventriculoarterial connections. The application of a technique currently used in neonates with transposition of the great arteries with or without a ventricular septal defect to this particular group of patients is no mean feat. This is not a definitive operation; rather it is one that hopefully provides a safer pathway to the Fontan operation, a path not jeopardized by the development of subaortic stenosis and myocardial hypertrophy. These patients will still require the surveillance of all patients who have undergone the arterial switch operation, focusing on the potential for pulmonary or (far less commonly) supraaortic obstruction, growth of the coronary arterial anastomoses, and the development of myocardial ischemia. Most of these patients palliated with the arterial switch operation will require further augmentation of pulmonary blood flow as well, as illustrated by the patients of Karl and associates [12]. This and other approaches (excluding pulmonary artery banding) to palliation of subaortic stenosis of the neonate and infant are shown in Table 1.

Finally, one should ask: Is there still a role for banding of the pulmonary trunk in double-inlet left ventricle and transposition of the great arteries [4, 9]? In a rare patient the ventricular septal defect will be unequivocally large, and one would certainly be tempted to band such a patient to protect the pulmonary vascular bed for a later Fontan operation. But in the majority of patients with this anatomy undergoing pulmonary arterial banding, subaortic stenosis is a predictable sequela [3, 4]. And in those requiring both banding of the pulmonary trunk and repair of an associated coarctation of the aorta, acquired subaortic stenosis approaches the inevitable [9]. Data from Fontan and associates [14] combining the experience of both Bordeaux and Birmingham showed a combined early and late mortality (or Fontan takedown) for patients undergoing a Fontan operation but palliated with previous banding of the pulmonary trunk in the range of 45% (13 of 29). This does not include those patients who died before or those not considered candidates for Fontan operation. Thus one will have to be very cautious in supporting the role of banding in the palliation of babies with the potential for subaortic stenosis. The accomplishment of Karl and colleagues presents us with an alternative palliative approach, and this, no mean feat, should be taken seriously.

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Assessing the Effect of Cardiopulmonary Bypass on the Brain

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Over the past three decades cardiac surgical outcome has dramatically improved, largely a result of improved surgical techniques, anesthetic drugs, and cardiopulmonary bypass (CPB) management. As the incidence of perioperative death (usually cardiac in origin) has declined over time, postoperative neuropsychological dysfunction has emerged as an increasingly important aspect of cardiac surgical morbidity. The focus has shifted from mere survival to quality of life after a cardiac operation. Because the brain is the organ most sensitive to the effects of ischemia and because brain injury after cardiac operations can be devastating, investigations of the central nervous system and CPB are of great interest. Investigative groups are systematically examining different techniques of cardiopulmonary bypass management (eg, alpha-stat versus pH-stat blood gas management, circulatory arrest versus continuous low-flow CPB) and their impact on the central nervous system. This examination of the cerebral effects of CPB is especially important because of the recent surgical trends toward operating at the extremes of age, the elderly and the very young, where physiological compensatory responses are limited and the risks of brain injury are higher. In these patients, surgeons are concerned with protection of the entire body and not just the heart.

See also page 479.

In their experimental study, Johnston and colleagues [1] examine the controversial area of CO_2 management during CPB and the effects on the brain. The theoretical advantages of alpha-stat management (temperature-uncorrected blood gases) include decreased emboli and the maintenance of cerebral blood flow/metabolism coupling [2]. The hypothetical disadvantages would include cerebral hypoperfusion due to lower arterial carbon dioxide tension. Alternatively, pH-stat management (temperature-corrected arterial blood gases) has the theoretical advantage of increasing cerebral perfusion, enhancing brain cooling, and the disadvantages of raising intracranial pressure and increasing brain edema, as well as the uncoupling of flow/metabolism. The study by Johnston and colleagues [1] addresses the impact of these two different blood gas management techniques on cerebral blood flow (CBF), on intracranial pressure and brain edema, and on retinal histopathology. The methods and findings in this study warrant further examination and should be put within the context of current knowledge regarding CPB and its effect on the central nervous system.

As with many studies examining the effects of CPB on the brain, the Johnston study lacks a true control group. To accurately assess the effect of CPB, a controlled group of animals undergoing median sternotomy without CPB is essential. Regrettably, no experimental or clinical studies to date have included this fundamental aspect of study design.

It is surprising and somewhat disconcerting that Johnston and associates found CBF to be depressed after rewarming from a relatively short period (150 minutes) of hypothermic CPB. This finding alone leads us to question the validity of their model. There are ample human data that document return of CBF to normal after equally long periods of CPB, provided there has been no period of circulatory arrest or very low CPB flow [3–5]. Reduction of CBF after a period of CPB can reflect a period of cerebral ischemia and is a usual accompaniment to perfusion strategies that use a period of total circulatory arrest. Not only have these findings been repeatedly reproduced in human subjects, experimental investigations by others [6–8] and in our own laboratory (unpublished data) further support this pattern—CBF follows temperature and cerebral metabolism and routinely returns to normal after a period of CPB when perfusion has been maintained at adequate levels, but tends to remain depressed if there has been a period of cerebral hypoperfusion. Furthermore, this depression of CBF is directly related in a dose-related fashion to the length of cerebral ischemia at a given temperature. Therefore, for Johnston and colleagues to make conclusions on a model that demonstrates depression of CBF after hypothermic CPB suggests that they have a system or model that provides inadequate perfusion or rewarming, and this may alter the interpretation of their data.

There are several possible explanations for their findings of cerebral hypoperfusion. They use both surface (rectal) and core (esophageal) temperatures in assessing temperature conditions during hypothermic and normothermic CPB. It is possible that brain temperature, which was not measured in the Johnston study, was still reduced even though rectal and esophageal temperatures were normal after reperfusion. Under these circumstances, it is likely that the decrease in CBF documented after rewarming could be accounted for by a change in brain temperature; that is, the brain is still cool at the end of the procedure despite "rewarming of core temperature." We believe that this may be an important explana-

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tion for their finding of cerebral hypoperfusion after CPB. In experimental animal models, small changes in brain temperature (2° to 3°C) can significantly confound results [9].

Alternatively, the animals were perfused at a rate of only 80 mL/kg. This may be too little flow, despite the fact that the mean arterial pressures seemed acceptable. By their own acknowledgement, the pressures were maintained by an increase in cerebral vascular resistance, and this clearly results in diminished CBF. The arterial cannulas used by their methods were exceptionally large (24F or 28F) given the size of their animals and perhaps impeded CBF. Finally, baseline and concluding measurements were taken during CPB rather than before and after weaning from CPB. It is important to assess the animals' CBF off CPB. In so doing, Johnston and colleagues might have discovered that they were producing inadequate flow during baseline (compared with what the animal really was used to) and that prolonged exposure to this inadequate flow, and not to their system of CO_2 management, is what actually produced the alterations they eventually observed.

Interpretation of CBF data in the absence of metabolic indices can be limiting. Under the study conditions where CBF is reduced it is important to know whether oxygen extraction is increased. The normal compensatory response in the presence of reduced flow is increased oxygen extraction, supporting oxygen consumption. We are unable to determine this in the Johnston study because oxygen metabolism was not examined. Cerebral hypoperfusion becomes pathological only when extraction is high, cerebral metabolic rate becomes fixed, and oxygen supply is dependent on oxygen delivery.

All of these issues demonstrate a very important point. It is essential that surgeons clearly and carefully try to understand the impact that their commonly used perfusion strategies have on altering the physiology to which the organism is accustomed. By trying to produce systems that most closely reproduce normal physiology, detrimental effects from CPB on the brain (and the body as a whole) should be limited.

Arterial carbon dioxide tension is a known potent cerebral vasodilator, even under the circumstances of hypothermic CPB [10]. In their study, Johnston and colleagues [1] showed no increase in CBF despite raising the arterial carbon dioxide tension with pH-stat management. Several studies, including previous studies from the same group [11, 12] have demonstrated both experimentally and clinically the maintenance of CO_2 responsivity during hypothermic CPB [3, 5, 13]. Why this was not observed in the present study is perplexing and may be unique to the model or experimental conditions. More importantly, because the CO_2–CBF response curve was flat during hypothermic CPB, one would not expect to see an increased incidence of microembolization or brain edema with either blood gas management technique. The fundamental concern of pH-stat management is the effect of increased CBF due to the addition of CO_2 with this type of management. Because the study did not achieve differential CBF levels, one would not anticipate different outcomes. Indeed, Johnston and associates demonstrated no difference in retinal histopathology or brain weight between groups. Unfortunately, the study conditions were inadequate to test the fundamental hypothesis.

In their methods, Johnston and colleagues use study conditions that could both favorably and unfavorably affect the incidence of microembolization. The use of a bubble oxygenator increases the incidence of gaseous emboli compared with use of a membrane oxygenator [14]. Cross-clamping of the aorta and opening of the left side of the heart increase the risk of microembolization during cardiac operations. The current study, similar to other experimental studies examining central nervous system injury, has excluded this important causative factor when assessing the effect of CPB on the brain. The inclusion of this study condition would greatly enhance the study of embolization during cardiac operations. Nonetheless, Johnston and colleagues' use of retinal histopathology to examine embolization during CPB is an elegant method. They observed an alarmingly high incidence (67%) of retinal emboli during rewarming [1]. In human studies using fluorescein retinography, a similar incidence of embolization has been observed associated with CPB [15]. Future investigations should address the size and distribution of microembolization.

The current mechanisms of brain injury due to CPB are thought to be related to three factors: (1) hypoperfusion (2) macroemboli or microemboli, or (3) brain injury secondary to the systemic inflammatory responses stimulated by CPB. Johnston and associates [1] have suggested evidence for two of these potential mechanisms of neuropsychological dysfunction in their animal model, ie, microembolization and hypoperfusion. The methodologic limitations of this study do not permit a more conclusive answer with regard to the issue of alpha-stat versus pH-stat management, and futher investigation is warranted.

In conclusion, investigations such as those by Johnston and colleagues are heading in the right direction with regard to a better understanding of the pathophysiological responses of the central nervous system to CPB. During the next decade it is anticipated that this line of investigation will lead to overall improvement in the quality of life for our patients.

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Operations for Subaortic Stenosis in Univentricular Hearts

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Optimal prevention and treatment of subaortic stenosis (SAS) in the univentricular heart with subaortic outlet chamber and high pulmonary blood flow remains controversial, especially when complicated by aortic arch obstruction. Herein we analyze our surgical results. Group 1 consisted of 11 infants (mean age, 10 days) with univentricular heart and SAS. Ten required repair of interrupted aortic arch (n = 7) or coarctation with hypoplastic arch (n = 7). Four patients had relief of SAS by either Damus-Kaye-Stansel connection (n = 2) or aortopulmonary window (n = 2), with three operative deaths and one late death. Six had one-stage arterial switch and atrial septectomy with arch repair (5/6) with one operative death and one late death. Two survivors have progressed to bidirectional cavopulmonary shunt, a third has had a Fontan operation, and a fourth awaits Fontan. In group 2, 11 children required operation for acquired SAS after pulmonary artery banding. Nine have progressed to Fontan operation with either staged (n = 3) or concurrent (n = 6) relief of SAS by Damus-Kaye-Stansel connection or subaortic resection. Fontan mortality was 11% (70% confidence limits, 2% to 32%). Group 3 consisted of 3 patients without pulmonary artery banding who had SAS diagnosed at Fontan evaluation. All 3 survived Fontan operation and relief of SAS by Damus-Kaye-Stansel connection or subaortic resection. Group 4 consisted of 1 patient with previous pulmonary artery banding (no SAS) who underwent Fontan operation but required Damus-Kaye-Stansel connection 30 months later for SAS. We conclude that arterial switch, arch repair, and atrial septectomy effectively palliate newborns with univentricular heart and SAS, although ultimate Fontan suitability is yet to be established. Both Damus-Kaye-Stansel connection and subaortic resection provide good long-term relief of SAS in select patients, and previous or concurrent treatment of SAS does not preclude successful Fontan operation in otherwise suitable patients.

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Subaortic stenosis (SAS) in the univentricular heart (UVH) remains problematic. Whether naturally occurring or secondary, SAS and its frequent sequelae, ventricular hypertrophy and dysfunction, have been identified as incremental risk factors for poor outcome after Fontan operations [1–7]. Although it is now widely accepted that pulmonary artery banding (PAB) can cause or accelerate the development of SAS in various types of UVH with discordant ventriculoarterial connections, a universally acceptable alternative for control of pulmonary blood flow in infancy has not been proposed [5, 8–10]. In this study we analyze our own experience with various palliative and definitive operations for SAS in the UVH. Long-term and short-term outcome, effect of palliation on Fontan suitability, and risks relative to patients with UVH without SAS are examined retrospectively. We also report our initial experience with a new form of palliation for newborns with UVH, SAS, and aortic arch obstruction, the subset of patients in which severe SAS is most likely to develop after PAB [4, 11].

For editorial comment, see page 415.

Material and Methods

All patients undergoing surgical procedures for SAS in the setting of UVH at the Royal Children's Hospital were identified using our database (1979 to present). Hospital and departmental records were examined by us, including operative notes, inpatient/outpatient entries, reports of cardiac catheterization and echocardiographic studies, and where appropriate the echocardiographic video tapes and angiograms. Autopsy data were either obtained from pathologist's report or when possible from personal examination of specimens by one of us. Follow-up information was obtained by us directly and from our referring cardiologists. No survivors were lost to follow-up. Analysis of data was done using standard statistical methods (when population size warranted). Seventy percent confidence intervals were calculated using continuity correction for upper and lower limits.

Patients and Operative Results

Twenty-six patients are included in this study, mean age at initial operation for SAS being 45 months (median, 13

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Table 1. Clinical Profile of 11 Newborns With UVH Who Required Surgical Treatment of SAS

Patient No.	Diagnosis	SAS Criteria	Operation (Age)	Outcome
1	DILV, TGA, type A IAA	Small BVF (echo)	a. Switch + atrial septectomy + arch repair + PAB (2 d) b. Fontan (25 mo)	NYHA I 3 mo post Fontan, free of SAS
2	Left AVV atresia, TGA, CoA, hypoplastic arch	Subaortic muscle bands, small BVF (echo)	Arch repair + atrial septectomy + PAB + APW (10 d)	Operative death
3	DILV, TGA, CoA, hypoplastic arch	Small BVF (echo)	Arch repair + enlargement BVF, PAB (1 d)	Well at 38 mo, awaits Fontan, free of SAS
4	DILV, CoA, hypoplastic arch, duct-dependent circulation	Small BVF (echo)	a. Switch + arch repair + atrial septectomy (15 d) b. Right MBTS (5 wk)	Well at age 13 mo, awaits Fontan, free of SAS
5	Tricuspid atresia, TGA, CoA + hypoplastic arch, duct-dependent circulation	Subaortic muscle bands + small BVF (echo)	a. Switch + arch repair + atrial septectomy (4 d) b. Right MBTS (3 mo)	Late death (3½ mo, probable recoarctation)
6	Left AVV atresia, TGA, type B IAA	Small BVF (echo)	Arch repair + atrial septectomy + APW + right MBTS (5 d)	Late death (3 mo, 50-mm Hg at APW)
7	Tricuspid atresia, TGA, CoA, hypoplastic arch, duct-dependent circulation	Small BVF (echo)	Switch + arch repair + atrial septectomy (2 wk)	Operative death (neoaortic valve incompetence)
8	DILV, TGA, type B IAA, duct-dependent circulation, single coronary	Small BVF (echo)	Arch repair + DKS + right MBTS (2 d)	Operative death (? coronary compression by DKS)
9	DILV, TGA, situs inversus, bilateral SVC	Subaortic muscle bands + small BVF (echo)	Switch + atrial septectomoy + PAB	Well at 30 mo, awaits Fontan, free of SAS
10	DILV, TGA, CoA, hypoplastic arch	Small BVF (echo)	a. Switch + arch repair + atrial septectomy (4 wk) b. MBTS (4 mo) c. BCPS + division MPA (19 mo)	Well at 25 mo, awaits Fontan, free of SAS
11	DILV, TGA, hypoplastic arch	Small BVF (echo)	Arch repair + atrial septectomy + PAB (6 d)	Operative death

APW = aortopulmonary window; AVV = atrioventricular valve; BCPS = bidirectional cavopulmonary shunt; BVF = bulboventricular foramen; CoA = coarctation of the aorta; DILV = double-inlet left ventricle; DKS = Damus-Kaye-Stansel connection; echo = echocardiography; IAA = interrupted aortic arch; MBTS = modified Blalock-Taussig shunt; MPA = main pulmonary artery; NYHA = New York Heart Association; PAB = pulmonary artery banding; SAS = subaortic stenosis; SVC = superior vena cava; TGA = transposition of the great arteries; UVH = univentricular heart.

months). The initial anatomical diagnoses are listed in Tables 1 through 3. The majority of patients had double-inlet left ventricle (or atrioventricular valve atresia) with a subaortic outlet chamber, discordant ventriculoarterial connection, and restrictive bulboventricular foramen (BVF). Patients were grouped according to age when first seen, type of operation for relief of SAS, and relation to previous operation, if any.

Group 1

Eleven patients had SAS recognized and treated surgically in infancy at a mean age of 10 days. The diagnosis was established by echocardiography in all. The criteria for diagnosis of SAS were a BVF significantly smaller (less than half diameter) than the aortic valve annulus or a measurable gradient from dominant ventricle to aorta (Doppler estimated), or both. Ten patients in group 1 had associated aortic arch obstruction consisting of interrupted aortic arch (n = 3) or coarctation plus hypoplastic transverse arch (n = 7).

Several surgical procedures were employed in this group (see Table 1). Four patients seen early in the series had one-stage arch repair through a median sternotomy plus atrial septectomy and either creation of aortopulmonary window plus PAB (n = 2) or Damus-Kaye-Stansel connection (DKS) plus modified Blalock-Taussig shunt (n = 2). There were three operative deaths, and the fourth patient died 3 months after operation. This fourth patient had recurrent/persistent SAS with a 50-mm Hg gradient across the aortopulmonary window at catheterization just before his death.

A fifth patient in group 1, with a right-sided outlet chamber and double-inlet left ventricle, underwent arch repair plus enlargement of BVF and PAB. He is currently awaiting Fontan operation and is free of SAS.

More recently, 6 patients have been treated with one-

Table 2. Clinical Profiles of 11 Children With UVH in Whom SAS Developed After PAB

Patient No.	Diagnosis	SAS Criteria	Operation (age)	Outcome
1	AVSD, hypoplastic LV, CoA, hypoplastic arch	30-mm Hg cath gradient (LV-Ao)	a. Arch repair + PAB (1 mo) b. DKS + BCPS (14 mo) c. AVV replacement (22 mo)	Probably Fontan unsuitable 15 mo postop
2	DILV, TGA	10-mm Hg cath gradient (LV-Ao)	a. PAB (10 d) b. DKS + Fontan (40 mo)	NYHA I 16 mo postop, free of SAS
3	LAVV atresia, TGA	Small BVF (echo)	a. PAB + septostomy (30 d) b. DKS + Fontan (28 mo)	NYHA I 19 mo postop, free of SAS
4	DILV, TGA, CoA	100-mm Hg cath gradient (LV-Ao)	a. PAB + CoA repair (3 d) b. Subaortic resection (trans-RV) (2 mo) c. Repair RV aneurysm (3 mo) d. Fontan (34 mo)	NYHA I 46 mo postop, free of SAS
5	DILV, TGA	Small BVF (echo)	a. PAB (10 d) b. PA reconstruction + central shunt (71 mo) c. Fontan (41 mo), subaortic resection (trans-Ao) (81 mo)	NYHA I 4 mo postop, 15 to 30-mm Hg SAS (echo)
6	Left AVV atresia, TGA, CoA	Small BVF (echo) + 15-mm Hg cath gradient (LV-Ao)	a. PAB + CoA repair (7 d) b. DKS + BCPS septectomy (13 mo) c. Fontan (35 mo)	NYHA I 25 mo postop, free of SAS
7	Single ventricle (indeterminate morphology); common AVV, LA isomerism, interrupted IVC, CoA	40-mm Hg cath gradient (Vent-Ao)	a. PAB + CoA repair (5 d) b. DKS + BCPS + common AVV replacement (19 mo)	Operative death
8	Tricuspid atresia, TGA	20-mm Hg cath gradient (LV-Ao)	a. PAB (8 d) b. Subaortic resection (trans-Ao) + BCPS (37 mo) c. Fontan (45 mo)	NYHA I 8 mo postop, free of SAS
9	Large VSD, hypoplastic right ventricle straddling tricuspid valve	40-mm Hg cath gradient (LV-Ao)	a. PAB + septectomy (5 mo) b. Fontan + subaortic resection (trans-RV) (62 mo) c. Subaortic resection (trans-Ao) (15 y)	Operative death
10	DILV, TGA	70-mm Hg cath gradient (LV-Ao) induced with isoproterenol	a. PAB (3 y) b. Fontan + DKS (82 mo)	Operative death
11	DILV, TGA	15-mm Hg cath gradient	a. PAB (5 mo) b. Fontan + subaortic resection (trans-Ao) (7 y)	NYHA I 18 mo postop, Free of SAS

Ao = aorta; cath = catheterization; IVC = inferior vena cava; LA = left atrial; LV = left ventricle; PA = pulmonary artery; RV = right ventricle; VSD = ventricular septal defect; other abbreviations as in Table 1.

stage arterial switch, aortic arch repair (5/6), and atrial septectomy, with one operative death (17%, 70% confidence limits, 2% to 46%). Three of 5 survivors required modified Blalock-Taussig shunt at a mean interval of 10 weeks from the time of arterial switch. There has been one sudden late death in this latter group, occurring at 5 months, probably related to recoarctation.

Of the 4 long-term survivors, 2 have undergone successful bidirectional cardiopulmonary shunts (BCPSs) with division of the main pulmonary artery (PA). A third patient has had a Fontan operation, and a fourth awaits BCPS or Fontan. All survivors are free of SAS and expected to be Fontan suitable.

Group 2

Eleven children, median age 10 days, who did not have SAS underwent PAB. All subsequently required surgical treatment for SAS 2 to 199 months after PAB (mean, 52 months; standard deviation, 56 months). Four of 11 (36%) required a coarctation repair at the time of PAB. There were no operative deaths related to PAB. The diagnosis of SAS after PAB was established by two-dimensional Doppler echocardiography or cardiac catheterization or both. Echocardiographic criteria were the same as in group 1, and any directly measured ventricular-aortic gradient was considered clinically significant.

Table 3. Clinical Profiles of 3 Children With UVH and SAS Who Had Concurrent Fontan Operation and Relief of SAS and 1 Child With UVH Who Required Surgical Treatment of SAS After Fontan Operation

Patient No.	Diagnosis	SAS Criteria	Operation (age)	Outcome
Group 3				
1	DILV, TGA, mild PS and sub-PS	30-mm Hg cath gradient (LV-Ao)	Fontan + DKS (137 mo)	NYHA I 15 mo postop, 10-mm Hg SAS by Doppler echo
2	DILV, TGA, PS	50-mm Hg gradient (LV-Ao) intraop	Fontan + resection SAS (240 mo)	NYHA I 17 mo postop, free of SAS
3	DILV, TGA, PS	10-mm Hg cath gradient (LV-Ao) and small BVF (echo + angio)	Fontan + DKS + pulmonary valvotomy (34 mo)	NYHA I 9 mo postop, free of SAS, mild AI
Group 4				
1	DILV, TGA, left-sided outlet chamber (3ASV), CoA	50-mm Hg cath gradient (LV-Ao)	a. CoA repair + PAB (newborn) b. Fontan (34 mo) c. DKS (64 mo)	NYHA I 30 mo after DKS, free of SAS

AI = aortic insufficiency; angio = angiography; Ao = aorta; cath = catheterization; echo = echocardiography; LV= left ventricle; PS = pulmonary stenosis; 3ASV = type 3A single-ventricle (Van Praagh); other abbreviations as in Table 1.

Treatment of SAS in this group consisted of either DKS or subaortic resection. In general, the former procedure was favored when the subaortic chamber was left sided (as in levotransposition of the great arteries), whereas direct resection was used for patients with right-sided outlet chamber.

The fate of patients in whom SAS developed after banding is outlined in Table 2. Nine of 11 ultimately progressed to Fontan operations. Of these 9 patients the SAS was dealt with at staging procedures in 3 (subaortic resection with or without BCPS, n = 2; DKS + BCPS, n = 1). The remaining 6 patients had Fontan operation plus concurrent subaortic resection (n = 3) or Fontan operation plus concurrent DKS (n = 3). There was one operative death in the Fontan group (11%; 70% confidence limits, 2% to 32%), occurring in a patient who had concomitant DKS. Postmortem examination showed severe myocardial hypertrophy and interstitial fibrosis.

Long-term follow-up of the patients who underwent Fontan operation revealed recurrence of SAS in 1 patient 10 years after transventricular subaortic resection. She underwent reoperation to relieve a 40-mm Hg ventricular-aortic gradient and could not be separated from cardiopulmonary bypass. She was bridged to cardiac transplantation with a ventricular assist device but died postoperatively of gram-negative sepsis. Postmortem examination revealed severe myocardial hypertrophy. The remaining 7 patients who underwent Fontan operation are well 12 to 46 months (mean, 21 months) after relief of SAS, although 1 has a detectable ventricular-aortic gradient by two-dimensional Doppler echocardiography.

Two children in group 2 have had DKS plus BCPS and common atrioventricular valve replacement, with one operative death due to pulmonary vascular disease. The survivor has no SAS 23 months after operation but may not be a Fontan candidate due to elevated upper body systemic venous and PA pressures.

Group 3

Three patients aged 34 months to 20 years, with no previous PAB, were discovered to have SAS at the time of preoperative evaluation for Fontan operation (n = 2) or during the operation (n = 1) (see Table 3). Two patients (with leftward outlet chambers) had Fontan + DKS and the third (with rightward outlet chamber) had transaortic subaortic myectomy. There were no operative deaths, and all 3 patients are in New York Heart Association class I at 9 to 17 months' follow-up. One has a Doppler signal compatible with a 10-mm Hg ventricular to aortic gradient, as compared with a resting 25-mm Hg mean gradient at preoperative catheterization, at which time the heart appeared volume loaded with a dynamic component of obstruction.

Group 4

A single patient who underwent PAB and coarctectomy in the newborn period underwent Fontan operation at 34 months of age. Neither catheterization nor two-dimensional Doppler echocardiography suggested SAS at that time. Thirty months later, however, SAS was documented with a 50-mm Hg ventricular-aortic gradient at catheterization. A DKS was successfully constructed at that time using the previously transected and oversewn PA. The patient remains free of SAS and is in New York Heart Association class I at 30 months' follow-up (see Table 3).

Surgical Techniques

Pulmonary Artery Banding

Our newborn banding technique employed median sternotomy whenever possible, the exception usually being a requirement for concomitant coarctation repair. In this latter case, the PAB was applied through a left thoracotomy.

Through a sternotomy, the upper pericardium was incised in the midline and the PA encircled with silicone elastomer–impregnated Teflon tape. The initial circumference of the band was usually 25 mm for a 3-kg baby, and adjustment was made to ensure a 10-mm Hg increase in mean arterial pressure with banding. Proximal and distal PA pressures were not measured. An arterial hemoglobin saturation of greater than 0.75 with satisfactory appearance of the ventricle and no acute increase in central venous pressure were additional requirements for a satisfactory band. The pericardium was closed whenever possible after the band was secured to the proximal PA with multiple sutures. If a future DKS procedure was a possibility, then high fixation of the band was employed to preserve as much length of PA as possible.

The sternotomy approach allowed a more precise placement of the PAB and in our opinion reduced the risk of late problems with the branch pulmonary arteries. Resternotomy after newborn PAB generally was not difficult, and the siliconized bands were easily separated from PA and left atrial appendage.

Damus-Kaye-Stansel Connection

This technique, originally described as part of an operation for transposition of the great arteries and later adapted for relief of SAS, involves end-to-side connection of the proximal transected PA to the proximal aorta [12–16]. Using hypothermic cardiopulmonary bypass and cardioplegic arrest, we routinely excised a triangular portion of aortic wall large enough to create an unobstructed PA-aortic connection. In most cases the connection was made without the addition of prosthetic material, even after PAB. We have used this connection in a patient with valvar pulmonary stenosis (combined with pulmonary valvotomy) and after previous division and oversewing of the proximal PA, with good relief of stenosis in both cases.

Bidirectional Cavopulmonary Shunt

All cavopulmonary shunts were performed during cardiopulmonary bypass with a beating heart. If concomitant Fontan operation was not performed, the superior vena cava was not routinely cannulated, and superior vena caval return was collected with a pump sucker during anastomosis. Cavopulmonary anastomoses were performed with continuous and interrupted fine polypropylene sutures, using a pulmonary arteriotomy of greater diameter than the superior vena cava to avoid narrowing the anastomosis.

Subaortic Resection

Enlargement of the BVF in hearts with right-sided outlet chambers was performed during hypothermic cardiopulmonary bypass with cardioplegic arrest, through either aorta (n = 4) or anterior ventricle (n = 2). In either case the resection was performed to enlarge the BVF in a superior and anterior direction. The end point was to create a BVF or subaortic channel equal in size to the aortic valve. There were no episodes of heart block using either the transaortic or transventricular approach. An aneurysm at the ventriculotomy developed in 1 patient and required early reoperation.

Arterial Switch Plus Aortic Arch Reconstruction

Repair of hypoplastic or interrupted aortic arch was performed from an anterior approach using profound hypothermia and circulatory arrest, in 3 cases with concurrent isolated myocardial perfusion [17]. All ductal tissue was excised and the coarctation and the hypoplastic arch segments (if present) were resected. Oblique end-to-end or end-to-side (interrupted aortic arch) anastomoses were then performed with running polypropylene sutures, after which pump flow was resumed. Arterial switch was then performed using the Royal Children's Hospital technique previously described, with atrial septectomy performed during a brief second period of circulatory arrest [18]. The PA was transected as high as possible to minimize any size mismatch with the native aortic root.

Fontan Operation

The type of Fontan connection used was atriopulmonary (n = 10) or cavopulmonary (n = 3). In either case, atrial neoseptation was performed with a Gore-Tex baffle, leaving atrioventricular valves and coronary sinus to the left.

Comment

Subaortic stenosis in the UVH has been recognized worldwide as a clinical entity since the early years of the Fontan experience [10, 12]. The key anatomical feature in SAS in the UVH is the BVF, which may be a slitlike opening with a completely muscular rim, somewhat smaller than the aortic annulus itself [2]. As such, there is a tendency for spontaneous closure, a process that is undoubtedly accelerated by the immediate reduction in diastolic heart volume after an effective PAB. In the longer term there is further reduction in BVF size due to PAB-related myocardial hypertrophy [19, 20]. Other anatomical causes of SAS may also exist in the UVH and may or may not be related to the presence of PAB [12, 21]. The time course for development of SAS after PAB for patients in whom it is not recognized at birth appears to be variable. Subaortic stenosis can become apparent very early in the postoperative period and is said to nearly always be evident by 2.5 years [5, 11, 20]. The mean time from PAB to surgical treatment of SAS in our series was 52 months (standard deviation, 56 months), although it is possible that the problem might have been present (but clinically silent) earlier in some patients, skewing the distribution. Furthermore, some newborns had open palliation of SAS, eliminating part of the cohort in which more severe SAS probably would have developed early after PAB. It is also likely that the diagnosis of SAS would have been more frequently made in the latter years of this study, with wide availability of two-dimensional Doppler echocardiography.

The actual incidence of SAS after banding in patients with UVH has varied from series to series and relates to a certain extent to the underlying anatomy. The classic case is the double-inlet left ventricle (or tricuspid atresia) with aorta arising from a right ventricular outlet chamber (discordant ventriculoarterial connection) and high pulmonary blood flow [5, 9, 11]. The incidence of SAS after PAB in this anatomical subset has been estimated to be as high as 42% to 84.4% in carefully followed up infant cohorts [5, 9, 11]. Our own experience supports this. The majority of our UVH patients coming forward for treatment of SAS have likewise had left ventricular morphology (24/26) and discordant ventriculoarterial connection (23/26).

In our series, patients with any type of aortic arch obstruction were 7 times more likely to require surgical treatment of SAS in the newborn period ($p < 0.005$). The association of arch obstruction and early SAS in the UVH has been documented previously [4, 11]. It has been suggested that the presence of SAS in utero can result in subnormal aortic flow, a situation favoring development of coarctation or other types of arch obstruction [20].

The diagnosis of SAS in the UVH requires echocardiographic demonstration of a BVF that is significantly smaller than the aortic root, or direct measurement of a ventricular aortic gradient. The majority of newborns at the Royal Children's Hospital currently are diagnosed with two-dimensional Doppler echocardiography (see Tables 1–3). Because this group tends to have arch obstruction and high pulmonary blood flow, a ventricular aortic pressure gradient is not always generated before PAB and an increase in aortic flow [4, 5, 22]. Our arbitrary echocardiographic criterion for diagnosis of SAS over the period of this study has been BVF size less than half the diameter of the aortic root. For patients undergoing cardiac catheterization, any measured ventricular aortic gradient was considered to be clinically significant. We have also used isoproterenol challenge to induce a gradient in 1 patient, as recommended by others [4, 10].

Even with catheterization and echocardiographic studies, the diagnosis of SAS may be difficult to establish. One patient in our series, who was 20 years old and had double-inlet left ventricle, transposition of the great arteries, pulmonary stenosis, and no previous PAB had no gradient at catheterization and an adequate size BVF by two-dimensional Doppler echocardiography and cineangiography. At the completion of Fontan operation, hemodynamics were poor and a harsh thrill was noted at the base of the heart. A 60-mm Hg gradient was measured between ventricle and aorta. This was successfully relieved with transaortic subaortic myectomy. It is thus possible that the Fontan operation itself, by "volume unloading" the single ventricle can, under some anatomical circumstances, initiate or increase SAS. This further strengthens the argument that any systemic gradient must be considered to be important in such patients.

The development of SAS, though manageable in some cases with complex surgical procedures, is certainly an undesirable situation. Progressive ventricular hypertrophy, subendocardial ischemia, and reduced ventricular compliance are predictable consequences. Although not mentioned per se in Fontan's original exclusion criteria, ventricular hypertrophy has been identified by Kirklin and associates [7] as a risk factor for early and late mortality after Fontan operation. A reduction in ventricular compliance may primarily affect diastolic function, with elevation of ventricular end-diastolic pressures as well as PA pressures distal to a band [6]. This results in a quite unfavorable post-Fontan hemodynamic situation, and may in some cases result in Fontan unsuitability.

Prevention of SAS in patients with the anatomical substrate would therefore be highly desirable, although at present it is not always possible. Alternatives to PAB to control pulmonary blood flow in the newborn period may generate problems worse than the band itself. Procedures involving disconnection of the PA from the heart require an alternate source of pulmonary blood flow. The BCPS is not appropriate in newborns, and systemic to PA shunts may create an unstable postoperative hemodynamic situation after cardiopulmonary bypass without guarantee of preventing ventricular hypertrophy and SAS. In our view, PAB still provides the best palliation. Because the development of SAS after PAB may ultimately exclude some patients from Fontan operation, early further interim palliation may be required to prevent progression or allow regression of ventricular hypertrophy. In our institute, of 178 patients undergoing Fontan operation, 31 had previous PAB, and 10 of 31 required surgical relief of SAS before or concurrent to Fontan operation. We have not identified previous PAB or previous/concurrent treatment of SAS as a risk factor for early mortality after Fontan operation ($p < 0.32$ and $p < 0.89$, respectively). We recognize, however, that the numbers are relatively small and that the question of Fontan suitability in remaining palliated patients remains unanswered.

Optimal management of established SAS remains controversial. The newborn group presents the most difficult problem, as the majority of these infants also have aortic arch obstruction requiring urgent treatment. In the past, various forms of palliation have been employed by ourselves and others. In most centers, including our own, results with operations employing a DKS or aortopulmonary window have had disappointing results in newborns [1, 2, 20, 23, 24]. The limiting factor appears to be the unstable post–cardiopulmonary bypass state created by a systemic shunt-dependent pulmonary circulation. The problem of maintaining balanced pulmonary and systemic flows is similar to that encountered after operation for the hypoplastic left heart syndrome. Also, survivors of aortopulmonary window may experience late problems with stenosis of the connection or distortion of semilunar valves, which would make subsequent Fontan suitability less likely [4, 18, 24].

Because of these problems, our preferred treatment for newborns with UVH and established SAS is now arterial switch plus atrial septectomy, usually with concomitant arch reconstruction. Pulmonary blood flow after arterial switch was effectively limited by the restrictive BVF in all but 1 of our patients (who had no arch obstruction and required PAB after switch + septectomy). This is similar to the situation of many newborns with tricuspid atresia or double-inlet left ventricle plus subpulmonic outlet chamber and normally related great arteries, a group in which SAS rarely develops. The requirement for modified Blalock-Taussig shunt after arterial switch is consistent with the natural history of the latter anatomical situation. The initial regulation of pulmonary blood flow by a restrictive BVF avoids the problems associated with a systemic shunt after a long period of cardiopulmonary bypass with fluctuating pulmonary and systemic vascular resistance. We believe that the presence of a valve in the pulmonary circuit may be important in this respect, preventing continuous flow.

The initial operative mortality of 1/6 (17%; 70% confidence limits, 2% to 46%) appears to compare favorably with experience with other SAS procedures in the newborn, although the small number of patients so treated does not allow us to generate a meaningful nonparametric comparison with alternative operations. Also, there has been one late sudden death at 5 months postoperatively, probably related to recurrent arch obstruction (autopsy not performed). None of the survivors has a catheterization or two-dimensional Doppler echocardiographically detectable ventricular-aortic gradient or severe ventricular hypertrophy.

Arterial switch has been employed previously in the UVH. Trusler [2] has used arterial switch combined with right atrium to right ventricle Fontan connection in older patients with rudimentary subaortic right ventricle large enough to contribute stroke work to the pulmonary circulation. This would appear to be an excellent option in this older group, although we have not yet employed it in our own unit.

Treatment of acquired SAS in older patients, whether after PAB or de novo, has varied according to age at detection, severity, and Fontan suitability by other criteria (see Table 2). The DKS is an effective way to bypass SAS and appears to provide sustained relief of obstruction. We have used this connection successfully even in the presence of pulmonary stenosis (with pulmonary valvotomy) or after previous PA division. Combined with BCPS and division of main PA as interim palliation, it is possible to allow regression of hypertrophy and growth of the patient before Fontan operation. For otherwise suitable patients (without severe hypertrophy), DKS can be performed concurrent to Fontan operation with acceptable results [9, 12, 23]. Our own experience is limited to 3 patients undergoing DKS plus BCPS (1 hospital death; 33%; 70% confidence limits, 4% to 75%), 1 of whom has since had a successful Fontan operation. An additional 5 patients had DKS plus Fontan operation without interim BCPS, with one death (20%; 70% confidence limits, 3% to 52%). Finally, 1 patient in whom SAS developed late after a Fontan operation had successful relief with a DKS constructed 64 months postoperatively.

Another surgical option that we have employed for SAS is enlargement of the BVF, which sometimes must be combined with resection of additional subaortic muscle bands. Despite potential complications of heart block, coronary injury, and recurrent SAS this approach has been used extensively in many centers [1, 2, 4, 6, 21, 23]. It has also been combined with right ventricular outflow patch with good results [25]. We prefer a transaortic approach whenever possible, as aneurysm formation at the systemic pressure ventriculotomy is possible using a trans-right ventricular approach, as we have seen in 1 newborn. The main risk appears to be heart block due to proximity of the conduction axis to the inferior rim of the BVF [12, 21]. Fortunately, we have not encountered this complication in any of our patients. Obtaining adequate relief of SAS with direct resection is more difficult in patients with a left-sided subaortic outlet chamber, and we consider these patients to be generally better candidates for a DKS-type connection if the pulmonary valve and artery are adequate. Resection is probably also a better option in larger patients. Relief of SAS with resection has been good, with only 1 patient requiring reoperation 10 years after the initial procedure. We and others have used resection both as a palliative and definitive (with Fontan) procedure, with no operative deaths.

Other technical options for relief of SAS in univentricular heart include ventricular-aortic valved conduits and PA-descending aortic conduit with distal PAB, with which we have no experience [4, 20, 22, 23]. Cardiac transplantation may also be a reasonable option in severe cases.

Newborns with UVH and SAS usually have coexisting aortic arch obstruction and are often prostaglandin E_1 dependent with low cardiac output. Under the current protocol at the Royal Children's Hospital, these babies undergo resuscitation and two-dimensional Doppler echocardiographic diagnosis and are then treated with arterial switch, arch reconstruction, and atrial septectomy. A right modified Blalock-Taussig shunt is used if pulmonary blood flow becomes inadequate in the postoperative period. If further palliation is required and the child is unsuitable for Fontan operation due to age or other factors, we would consider BCPS and division of main pulmonary artery; otherwise, we would proceed directly to Fontan operation.

For infants with UVH, high pulmonary blood flow, and no SAS, transsternotomy PAB is our procedure of choice. If isolated coarctation is present (without hypoplastic arch or SAS) then a left thoracotomy is used for concomitant coarctation repair and PAB. Patients in whom SAS develops beyond infancy, with or without previous PAB, are treated with DKS plus BCPS or subaortic myectomy plus BCPS if unsuitable for Fontan operation (due to young age, severity of SAS, ventricular hypertrophy or other factors). Older children with mild SAS and without severe hypertrophy would proceed directly to Fontan plus DKS or Fontan plus transaortic subaortic myectomy, depending on anatomy (suitability of pulmonary root, position of outlet chamber and aorta).

In conclusion, arterial switch plus arch repair and atrial septectomy provides effective palliation for newborns with UVH, arch obstruction, and SAS. The operative risk will probably compare favorably with other forms of palliation. The ultimate suitability of newborns thus treated for Fontan operation appears to be good, although the small number of patients precludes firm conclusions at this time. The DKS operation and subaortic resection both provide good relief of SAS in select patients and are useful either as staging procedures or concurrent to a Fontan operation. Pulmonary artery banding, although associated with development of SAS in the UVH, does not preclude successful Fontan operation. Close follow-up is advisable after PAB to allow early detection of SAS and possible interim palliation before Fontan operation.

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DISCUSSION

DR MICHEL ILBAWI (Oak Brook, IL): I would like to congratulate Dr Karl and associates on very beautiful work. I think this is a very complex problem and the results are very outstanding. Recently we have adopted a totally different approach to the problem. At the present time we prefer to palliate these patients very early, before the development of a pressure gradient.

The procedure is pulmonary artery to ascending aorta anastomosis. In cases of aortic arch anomalies, we connect the proximal pulmonary artery directly to the arch. We use a piece of Gore-Tex patch to augment the anastomosis and prevent tension and distortion of the semilunar valves.

Out of the 13 patients we have treated in the last 8 years, 9 had the initial banding in the neonatal period, and subsequent to that, around 2 to 6 years of age, had relief of the stenosis whenever the pressure gradient became apparent. In the 4 patients who were treated more recently, the relief of stenosis was done in the neonatal period, around 10 days of age.

The evaluation is very impressive and shows a marked difference between the two groups when it comes to pulmonary vascular resistance, for example. The pulmonary vascular resistance was much higher in group I, who had the delayed relief of the subaortic stenosis, in contrast to group II, where the relief of stenosis was done in the neonatal period. The ventricular muscle mass, which is inversely proportional to ventricular compliance, was also markedly increased in the delayed group, and the ventricular mass to volume ratio, which reflects both the pathological hypertrophy and the contractile state of the ventricle, was markedly increased in group I, who had delayed relief of the obstruction.

The post-Fontan results again were markedly different. The central venous pressure was higher, takedown of the Fontan operation was necessary in 2 patients, and the hospital mortality was higher in group I because of the severe ventricular hypertrophy and noncompliant ventricle. I wonder whether Karl and associates have any similar experience when it comes to timing of the procedure. The other question relates to techniques. Most of these patients have levotransposition of the aorta, and you are recommending arterial switch. Would you like to comment on the technical details of how you would do an arterial switch on levotransposition of the aorta?

DR KARL: To answer the second question, our switch technique in univentricular heart would be identical to our switch technique in simple transposition, using medially based trapdoor flaps for translocation of the coronary arteries. We would transect the pulmonary artery somewhat higher than usual to minimize the size mismatch between the neoanastomosed vessels.

Regarding the timing of operation for subaortic stenosis, I may not have made this clear in my talk, but we would treat subaortic stenosis at the time of diagnosis, especially in the newborn group. We would not, for example, band a patient who we thought had a small bulboventricular foramen. We would opt for the treatment course that I have outlined. In children in whom subaortic stenosis develops after banding, we would intervene early, probably with a Damus-Kaye-Stansel operation or direct enlargement of the bulboventricular foramen, depending on the position of the subaortic chamber and aorta. In a young patient who might not be otherwise suitable for Fontan operation, we would perform a bidirectional cavopulmonary shunt concurrently.

DR ADNAN COBANOGLU (Portland, OR): Dr Karl, how often do you perform pulmonary artery banding now in patients with univentricular heart and unrestricted pulmonary blood flow? How often do you perform pulmonary artery banding; is it a very uncommon procedure now on your service?

DR KARL: I can say that we would perform pulmonary artery banding in such patients when we are happy about the size of the bulboventricular foramen and convinced by the Doppler echocardiographic studies that there is no subaortic stenosis. Otherwise we would proceed with arterial switch and atrial septectomy. Now, in the newborn group this is almost always a child with an obstructed aortic arch who needs an open procedure for that reason as well.

DR COBANOGLU: The problem that we have seen is the difficulty at times to decide if the bulboventricular foramen is truly restrictive or not. The morphology of the foramen is not a circle, it is more like a buttonhole, and at times it is difficult by echocardiography to decide whether you have a restrictive foramen or not.

DR KARL: Right. I think that question might better be posed to our cardiologists, but I think if the bulboventricular foramen is small, it is not a subtle thing. I mentioned that our criterion was a foramen less than half the diameter of the aortic root, and if there is an associated arch obstruction, I think one should have an even lower threshold to treat it as subaortic stenosis.

In our current protocol for newborns, if subaortic stenosis is present, we will perform the arterial switch as outlined. If subaortic stenosis is not present, then we would apply a pulmonary artery band through a sternotomy unless there is an isolated coarctation present, in which case it could be done through a left thoracotomy.

For the older child with acquired subaortic stenosis, a Damus-Kaye-Stansel connection is used when there is a left-sided outlet chamber, and subaortic resection, which is usually transaortic, is employed for a right-sided outlet chamber. Depending on other anatomical and physiologic features, a bidirectional cavopulmonary shunt or Fontan operation would be performed concurrently.

Use of the Inferior Epigastric Artery as a Free Graft for Myocardial Revascularization

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From March 1990 through January 1991, 47 patients undergoing myocardial revascularization had one (37) or both (10) inferior epigastric arteries (IEA) used as a conduit for bypass with 62 distal anastomoses. The internal thoracic artery (ITA) was used bilaterally in 41 patients and unilaterally in 6 with 100 distal anastomoses. Five patients had a single saphenous vein graft. In total, 167 anastomoses (3.55 per patient) were performed. Single IEA grafts were harvested through a paramedian incision and bilateral grafts, a midline incision. Harvest time was 36.5 minutes for IEA grafts and 29.6 minutes for ITA grafts ($p < 0.0001$). Graft length was 11.9 cm for IEA grafts and 16.5 cm for ITA grafts ($p < 0.0001$). Distal graft diameter was 2.0 mm for IEA grafts and 2.1 mm for ITA grafts ($p < 0.01$). Graft flow was 49.7 mL/min for IEA grafts and 48.7 mL/min for ITA grafts. Microscopic assessment of segments of both the IEA and ITA from 14 patients revealed similar internal elastic laminae and an equal number of fenestrations. Combined intimal and medial thickness was comparable in both conduits. Medial elastic tissue was more prominent in ITA grafts and lacking in eight of the 14 IEA grafts. Gross plaque formation was noted in the proximal 1 to 3 cm of 50% of IEA grafts, but the lumen was not compromised and microscopic thickening was minimal. An unexpected finding was medial calcifications (Mönckeberg's disease) in two of the 14 IEAs without associated atherosclerosis. There was one hospital death, one abdominal wound infection, and one instance of fat necrosis superficial to the sternum. The IEA has proved to be an acceptable conduit with minor associated morbidity. Short-term and long-term patency must be determined.

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It has been well established that the internal thoracic artery (ITA) is preferable to saphenous vein as a conduit for coronary artery bypass grafting (CABG) because of superior early and late patency [1]. The major advantage of the ITA is its freedom from progressive atherosclerosis, documented angiographically in patients up to 17 years after bypass grafting (Barner HB. Personal observations). Many surgeons now commonly use both ITAs for CABG, and many have adopted complex grafting methods such as free ITA grafts and sequential anastomoses. Despite these techniques, it has still not been possible to achieve complete revascularization with arterial conduits in the majority of patients with triple-vessel disease.

More recently, the right gastroepiploic artery has been used as a third arterial conduit [2–5]. Use of the conduit has not become routine, however, perhaps because of concerns regarding increased time of harvest, entry into the peritoneal cavity, or reports [3, 6] suggesting episodes of arterial vasospasm.

Puig and associates [7] have reported using the inferior epigastric artery (IEA) as an alternative arterial conduit. They placed 24 IEA bypass grafts in 22 patients with an overall patency of 88% (15/17) evaluated by catheterization at 8 to 10 days. Patency was 100% in the 10 patients in whom a patch of bovine pericardium or autogenous vein was used for the aortic anastomosis and 71% (5/7) with direct anastomoses. Encouraged by this report, we began using the IEA for select patients in March 1990, and our experience with this conduit since that time forms the basis for this report.

Material and Methods

Clinical data recorded prospectively on all patients included age, sex, history of prior operative revascularization, and the presence of associated medical diseases including insulin-dependent diabetes mellitus, cerebrovascular disease (defined as history of stroke, transient ischemic attacks, or carotid endarterectomy), peripheral vascular disease (history of claudication, rest pain, or prior vascular operation), and cigarette smoking. Height, weight, and body surface area were also recorded.

Data gathered at catheterization included the presence of coronary artery disease (defined as ≥70% luminal occlusion in any plane on coronary angiography) and left ventricular score (as defined by the Coronary Artery Surgery Study group).

The incidence of operative death (death at any time during hospitalization or within 30 days of operation after discharge), perioperative myocardial infarction (defined

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as the presence of a new Q wave on the postoperative electrocardiogram or postoperative alteration in the $LDH_1:LDH_2$ ratio to >1.0), and low cardiac output (defined as the use of an intraaortic balloon or inotropic support for >24 hours postoperatively) were recorded. Other complications noted included the presence of major arrhythmias (eg, those requiring pharmacological treatment or cardioversion), need of prolonged ventilator support (>48 hours), reoperation for bleeding, wound infection, pneumonia, and bronchopleural fistula.

The conduct of the operative procedure was similar in all patients. The ITA grafts were mobilized first, divided distally, and wrapped with a laparotomy pad soaked in warm saline solution containing 0.5 mg/mL of papaverine hydrochloride. Initially IEA grafts were mobilized with a midline incision from umbilicus to symphysis pubis with anterior retraction of the rectus muscle to expose the posterior IEA. In the first 2 patients, a complementary groin incision was made for exposure of the IEA origin, but in all subsequent patients, a groin incision was not used. Midway in this experience, a paramedian incision rather than a midline incision was used if a single IEA was to be harvested. Because the IEA lies at the junction of the middle and lateral thirds of the rectus muscle, the paramedian incision is placed well laterally with a corresponding incision in the anterior rectus fascia and splitting of the rectus muscle to expose the artery lying deep to the muscle. Branches were controlled with small clips. The ITA was mobilized with its two venae comitantes and a minimum of associated fatty tissue from within 1 cm of its origin to the level of the umbilicus. One surgeon usually mobilized all three or four arterial grafts sequentially, but when two surgeons were available, the ITA and IEA could be harvested simultaneously. After mobilization, IEA grafts were placed in a sponge moistened with room-temperature saline solution containing papaverine (1 mg/mL).

Before use, grafts were flushed with 10 mL of heparinized blood containing 1 mg/mL of papaverine using a plastic olive-tipped needle. Each end of the IEA graft was gently calibrated with a probe in 0.5-mm increments and its length measured. The ITA grafts were similarly sized. The time required from skin incision to proximal division of the IEA graft was recorded for all patients as was the IMA harvest time (interval from elevation of the hemisternum to division of last branch for in situ grafts). After reversal of heparin sodium with protamine sulfate, the abdominal incision was closed using running 2-0 polypropylene suture for the muscular fascia and 3-0 polyglycolic acid suture for subcutaneous fascia and subcuticular skin closure.

The remainder of the operation did not deviate from our standard practice and included a two-stage venous cannula, aortic cannulation, systemic hypothermia (25°C), and an anteroseptal temperature probe. Anterograde cold blood potassium cardioplegia (initial bolus of 10 mL/kg at 5°C containing 16 mEq/L of KCl, followed at 20-minute intervals by subsequent boluses of 5 mL/kg at 10°C containing 8 mEq/L of KCl) was used for primary operations and anterograde/retrograde cardioplegia for reoperations. Saline ice-slush was used for topical hypothermia. Standard procedure also included an infusion of 500 mL of warm unmodified cardioplegia (8 mEq/L of KCl) at 50-mm Hg pressure before cross-clamp release. For reoperations, proximal anastomoses were accomplished during a single period of aortic cross-clamping; otherwise, they were performed after cross-clamp removal using a partial occlusion clamp. All patients received intravenous nitroglycerin (33 μg/min) starting with ITA mobilization and continuing for 12 hours postoperatively.

Because of the more limited length of IEA grafts, they were preferentially placed to coronary arteries closer to the aorta (left anterior descending, diagonal, ramus marginalis, and first obtuse marginal), whereas ITA grafts were used for more distal arteries. Distal anastomoses entailed spatulation of the IEA, a 3- to 4-mm coronary arteriotomy, and a continuous suture line of 7-0 or 8-0 polypropylene. Proximal anastomoses to the aorta were accomplished with a 4- or 5-mm circular punch and 7-0 polypropylene or by end-to-side anastomosis to the hood of a vein graft. In patients undergoing repeat revascularization, the proximal anastomosis was made to the hood of an old vein graft. Inferior epigastric arteries that would not reach the aorta were anastomosed end-to-side to ITA grafts (7), to another IEA graft (1), or to a vein graft (1) using 4-mm incisions and 7-0 Prolene. Sequential IEA grafts were not used; however, five IEA grafts were suitable for natural Y grafts.

When patients were in hemodynamically stable condition after the termination of cardiopulmonary bypass, graft flows were measured. Initially this was performed using an electromagnetic flowmeter, but more recently, flows have been measured using an ultrasonic transit-time flowmeter (Transonic Systems, Inc, Ithaca, NY). In a few patients, flows were not measured either because of equipment malfunction or because of difficulty with graft access and probe placement.

In 14 patients (11 men and 3 women), excess arterial tissue permitted sampling of 3- to 5-mm segments of both the IEA and ITA for microscopic examination. The tissues were placed in 10% neutral buffered formalin and processed routinely for paraffin embedment. Sections 5 μm thick were cut from the paraffin-embedded blocks and were prepared with both a standard hematoxylin and eosin stain and a Verhoeff–van Gieson stain for the demonstration of elastic tissue, smooth muscle, and collagen. Morphometric data were obtained with the aid of a calibrated reticle placed in the ocular of an AO light microscope. The intimal thickness and the combined thickness of the arterial intima and media were determined at three randomly chosen sites. Qualitative determinations of the amount of elastin were also made.

Statistical comparison of continuous variables was accomplished with a Student's t test, and discrete variables were compared using a χ^2 test. Simple linear regression was used to assess correlation between continuous variables. A p value of less than 0.05 was considered significant.

Results

Between March 1990 and January 1991, 47 patients underwent myocardial revascularization at St. Louis University Medical Center using one or both IEAs as a free graft. There were 41 men and 6 women ranging in age from 39 to 75 years (mean age, 58.9 years). Associated medical disorders included insulin-dependent diabetes mellitus in 8 patients (17%), history of peripheral vascular disease in 6 (13%), cerebrovascular disease in 2 (4%), and history of smoking in 27 (57%). The results of preoperative cardiac catheterization are presented in Table 1. Approximately 9 of 10 patients had three diseased coronary vessels, and one quarter had moderate to severe ventricular dysfunction.

Seventeen (36%) of the 47 patients had undergone one (15 patients) or two (2 patients) prior revascularization procedures. The current revascularization was elective in 44 patients (94%). In 3 patients, operation was undertaken on an urgent basis because of continuing angina in the face of intravenous administration of heparin and nitroglycerin. No patient underwent CABG with an IEA for emergency indications (eg, cardiogenic shock, failed percutaneous transluminal coronary angioplasty, or evolving infarction).

In 36 patients a single IEA was removed. The remaining 11 patients had both IEAs harvested, but in 1 patient, the left IEA was pulseless and appeared fibrotic and withered; this vessel was discarded. Thus, in 37 patients (79%) a single IEA was used, and in 10 (21%), two IEA grafts were used for coronary anastomoses. A total of 62 anastomoses were performed with these grafts, and the sites of these anastomoses are shown in Table 2. Forty-one patients (87%) had both ITA vessels harvested, and only 6 (13%) had a single ITA used. The right ITA was harvested in 45 patients and the left, in 43 patients. A total of 100 anastomoses were performed with these ITA vessels, and the sites of those anastomoses are also shown in Table 2. The saphenous vein was harvested and used for coronary grafting in only 5 patients (11%), each of whom received a single anastomosis with this conduit. Thus, a total of 167 anastomoses were performed in 47 patients, a mean of 3.55 distal anastomoses per patient.

Table 1. Results of Cardiac Catheterization[a]

Variable	No. of Patients
Coronary artery disease	
Single-vessel disease	1 (2)
Double-vessel disease	4 (9)
Triple-vessel disease	42 (89)
Mean no. of diseased vessels	2.88 ± 0.40
LV score	
Normal (LV score = 5)	13 (28)
Mild dysfunction (6–10)	23 (49)
Moderate dysfunction (11–15)	11 (23)
Severe dysfunction (≥15)	1 (2)
Mean LV score	8.2 ± 2.8

[a] Numbers in parentheses are percentages.

LV = left ventricular.

Table 2. Anastomotic Sites

	IEA (n = 57)	LITA (n = 43)	RITA (n = 45)	SVG (n = 5)
Left				
End to end				
LAD	17	12	3	0
Ramus	8	2	0	0
D1	2	3	0	1
D2	0	0	1	0
Sequential				
LAD–D1	0	6	0	0
LAD–D1–D2	0	1	0	0
D1–D2	2	0	0	0
LAD–LAD	0	1	0	0
LAD–ramus	2	0	0	0
Circumflex				
End to end				
OM1	16	7	2	0
OM2	2	6	3	1
LPL	0	3	1	0
Sequential				
OM1–OM2	0	1	0	0
OM1–ramus	1	0	0	0
Right				
End to end				
RCA	1	1	5	0
PD	0	0	24	0
RPL	6	0	4	3
Sequential				
RCA–PD	0	0	1	0
PD–PPD	0	0	1	0

D1 = first diagonal; D2 = second diagonal; IEA = inferior epigastric artery; LAD = left anterior descending; LITA = left internal thoracic artery; LPL = left posterolateral; OM1 = first obtuse marginal; OM2 = second obtuse marginal; PD = posterior descending; PPD = premature posterior descending; Ramus = intermediate; RCA = right coronary artery; RITA = right internal thoracic artery; RPL = right posterolateral; SVG = saphenous vein graft.

The time required for graft harvest was approximately 7 minutes longer for the IEA than for the ITA (Table 3). Measurement of usable graft length before the performance of anastomoses revealed that the mean IEA length was 4.6 cm shorter than the mean ITA length. Although regression analysis revealed no correlation between the length of harvested IEA grafts and patient height, weight, or body surface area, there was significant correlation between the length of the ITA graft and both height ($R^2 = 0.218$, $p < 0.0001$) and body surface area ($R^2 = 0.079$, $p < 0.01$).

Calibration of graft diameter using 1.0-, 1.5-, 2.0-, and 2.5-mm probes revealed the ITA to be slightly larger on average (see Table 3). However, the significance achieved

Table 3. Comparison Between Inferior Epigastric Artery and Internal Thoracic Artery

Variable	IEA (n = 57)	ITA (n = 88)	*p* Value
Length (cm)	11.9 ± 2.6	16.5 ± 1.0	<0.0001
Diameter (mm)	2.0 ± 0.3	2.1 ± 0.2	<0.01
Flow (mL/min)[a]	49.7 ± 34.2	48.7 ± 30.7	NS
Harvest time (min)	36.5 ± 7.8	29.6 ± 6.9	<0.0001

[a] For this variable, n = 54 for IEA and n = 82 for ITA.

IEA = inferior epigastric artery; ITA = internal thoracic artery; NS = not significant.

in this comparison is due to the markedly low values for the standard deviation, a finding more likely reflecting the relatively gross measurement technique rather than any real difference in vessel size. Certainly vessel diameter did not appear to be of any clinical or statistical significance, as the measured flows were nearly identical in IEA and ITA grafts (see Table 3).

The most frequent postoperative complication was supraventricular tachycardia, which occurred in 8 patients (17%). Other complications included prolonged ventilator support in 4 patients (8.5%), myocardial infarction in 4 (8.5%) (new Q wave in 1 and isoenzyme change in 3), low cardiac output in 3 (6.4%), ventricular arrhythmia in 2 (4.3%), and abdominal wound infection, sternal wound complication (subcutaneous fat necrosis), pneumonia, bronchopleural fistula, and reoperation for bleeding in 1 patient each (2.1%).

A single patient died in the postoperative period, an operative mortality of 2.1%. This patient was a 67-year-old man who had undergone two prior myocardial revascularization procedures 11 and 15 years previously. He was seen with a 2-month history of rest angina that occurred daily and was refractory to maximal medical management. He was evaluated by coronary angiography and left ventriculography and was found to have severe left ventricular dysfunction (left ventricular score of 16 and ejection fraction of 0.29) and triple-vessel disease resulting from obstruction in both native and bypass graft vessels. Revascularization was undertaken, and four anastomoses were performed (2 ITAs, 1 IEA, and 1 saphenous vein graft). The patient required inotropic support and an intraaortic balloon to be separated from cardiopulmonary bypass, but on the first postoperative day, his status improved, and the intraaortic balloon was discontinued. Approximately 1 hour later, sudden ventricular tachycardia developed. Resuscitation included reexploration of the mediastinum and the institution of extracorporeal membrane oxygenator support through femoral cannulation. Although the patient regained consciousness, a coagulopathy developed and 24 hours later, extracorporeal membrane oxygenation was discontinued in light of what appeared to be adequate cardiac function supported by inotropic agents and an intraaortic balloon, which had been reinserted. Over the next 2 days there was gradual deterioration of cardiac function and mental status, and the patient died on the fifth postoperative day.

No hospital survivor was lost to follow-up. There have been no deaths or ischemic events in any patient since discharge. The longest period of follow-up has been 10 months.

Histopathological examination and direct comparison of IEAs and ITAs in a subgroup of 14 patients focused on four areas of interest: vascular calcification, the internal elastic lamina (IEL) and external elastic lamina, the amount of elastin within the tunica media, and the total thickness of the two vessels. Optical magnification (4.0 × loupes) allowed identification of mild plaque formation without luminal compromise in the proximal 1-cm to 3-cm in 50% of IEAs and 5% of ITAs. The arterial segments assessed microscopically did not have frank atherosclerotic lesions. Marked calcification was found in the inner third of the tunica media in two of the 14 IEAs (Fig 1).

All the ITA and IEA arteries studied had pronounced IELs containing an equal distribution of fenestrae. Both arteries also demonstrated external elastic laminae in the majority of vessels (Figs 2, 3).

The amount of lamellar elastic tissue within the tunica media differed considerably in the IEA and the ITA. In general, the IEA contained only scant or absent elastic lamellae within the media (see Fig 2), but in the ITA, four to six such lamellae were usually observed (see Fig 3). However, deviation from this pattern was noticeable in three of the 14 IEAs (Fig 4) and in the ITA in 2 patients (Fig 5). Marked intimal thickening, measuring 50 μm or thicker, was present in only two ITAs (Table 4).

Measurements of the combined thickness of the intima and media revealed no significant differences between the two arteries (see Table 4). Although every effort was made to embed all arteries at a 90-degree angle to the cutting surface, a variation in the angle finally obtained cannot be ruled out; moreover, the combined thickness inevitably reflected, in part, the extent of intimal thickening manifest in the majority of this group of middle-aged patients electing CABG. For these reasons, the measurements of medial plus intimal thickness may differ from those obtained in randomly selected autopsy specimen [8].

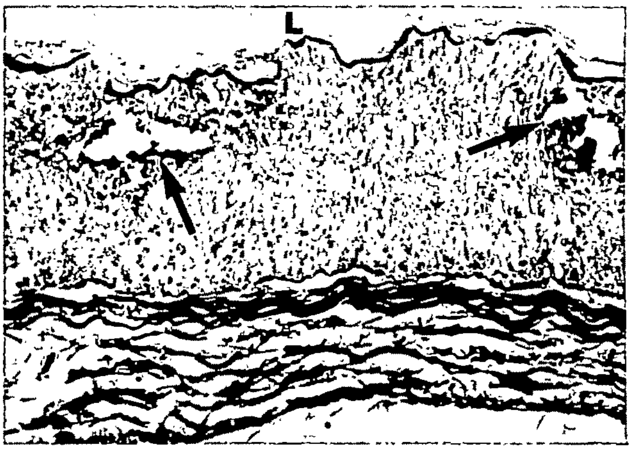

Fig 1. Inferior epigastric artery. Foci of calcification (arrows) are present in the luminal portion of the media. No inflammatory reaction is apparent. (Verhoeff–van Gieson; ×150 before 26% reduction. (L = lumen.)

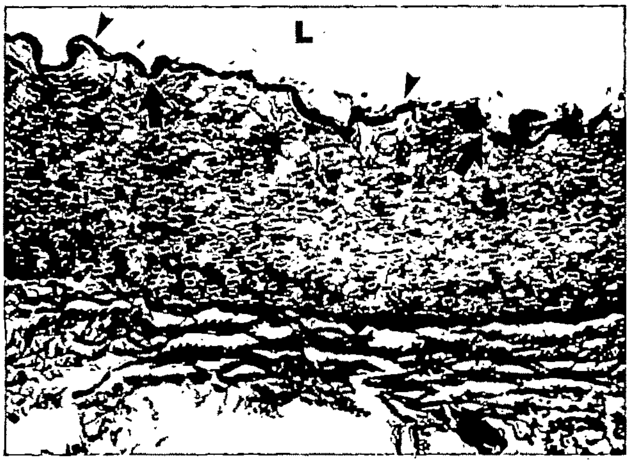

Fig 2. Inferior epigastric artery. Note prominent internal elastic lamina (arrowheads) *and fenestrations* (arrows) *within it. The media contains only sparse amounts of elastic tissue. The external elastic lamina is indicated by the* black diamond-shaped marker. *(Verhoeff–van Gieson; ×100 before 26% reduction.) (L = lumen.)*

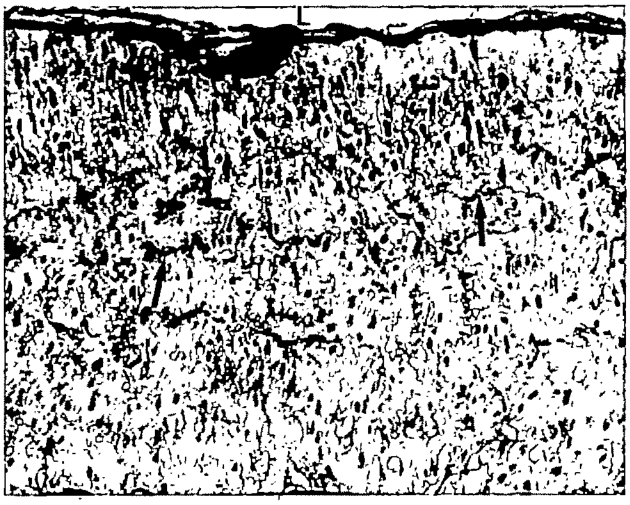

Fig 4. Inferior epigastric artery. Note moderate amounts of elastic tissue within media (arrows). *This degree of elastic tissue presence in the media was noted in a small number of inferior epigastric arteries. (Verhoeff–van Gieson; ×200 before 25% reduction.) (L = lumen.)*

Comment

This limited experience suggests that the IEA can serve as a satisfactory coronary artery conduit without excessive early morbidity or mortality. It can be argued that our patients were carefully selected to be a relatively "low risk" population. The mean age of these patients was less than that of the patient population having isolated CABG in 1989 (58.9 ± 8.5 years versus 64.7 ± 9.6 years; $p < 0.001$), and the number of urgent or emergency operations was less (6% versus 15%), although this latter difference was not significant. Left ventricular score was also not significantly different when the IEA and 1989 populations are compared (8.2 ± 2.8 and 8.9 ± 3.0, respectively). In several respects, however, these IEA patients were at similar or greater risk than our general CABG population. Reoperations have recently been demonstrated to have significantly higher morbidity and mortality than "first-time" revascularizations. More than one third of our IEA patients were undergoing a second or third revascularization, and this incidence was three times higher than that found in our 1989 population (36% versus 11%; $p < 0.0001$). In addition, more IEA patients had insulin-dependent diabetes mellitus (17% versus 8%), a condition known to predispose to substantial morbidity in patients undergoing bilateral ITA harvesting, as was the rule in our patients. The severity of coronary artery disease as measured by the mean number of diseased vessels was worse in our IEA patients (2.9 ± 0.4 versus 2.6 ± 0.7; $p < 0.01$).

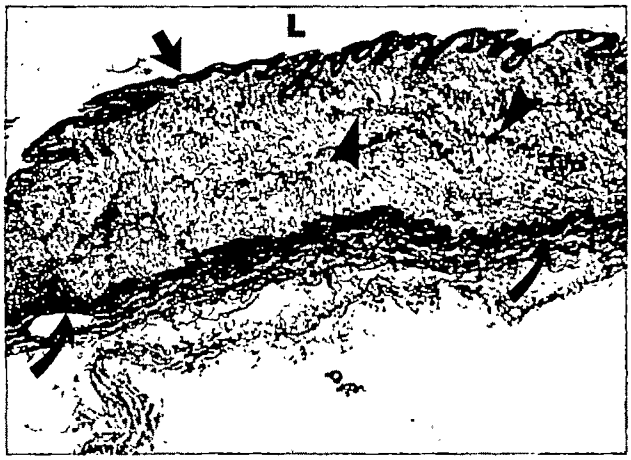

Fig 3. Internal thoracic artery. Pronounced internal (straight arrow) *and external* (curved arrows) *elastic laminae are evident. Note elastic lamellae within the media* (arrowheads). *(Verhoeff–van Gieson; ×100 before 25% reduction.) (L = lumen.)*

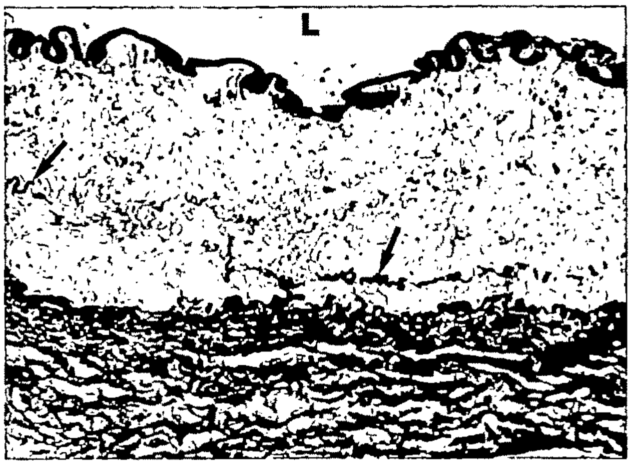

Fig 5. Internal thoracic artery. Note scant amount of medial elastic tissue (arrows), *a pattern not commonly seen in this artery. (Verhoeff–van Gieson; ×200 before 25% reduction.) (L = lumen.)*

Table 4. Morphometric Data

Variable	IEA (n = 14)	ITA (n = 14)
Degree of intimal thickening		
Absent or minimal (<10 μm)	8	8
Moderate (10–50 μm)	6	4
Marked (>50 μm)	0	2
Combined thickness of media and intima		
<200 μm	3	4
<300 μm	10	6
<400 μm	1	3
>400 μm	0	1
Presence of elastic tissue in media		
None	8	0
Minimal	6	2
Distinct	0	12

A major disadvantage of the IEA is its limited length, which is roughly the distance from the inguinal ligament to the level of the umbilicus. When a paramedian muscle-splitting incision is used, it is relatively easy to carry the dissection above the umbilicus, but the "usable" length is then limited by the decreasing diameter of the IEA as it courses cephalad. When the IEA enters the rectus muscle, dissection becomes extremely tedious and may be impossible when the approach is from a midline incision. In this instance, conversion to an anterior muscle-splitting approach may be helpful.

The shorter length of the IEA as compared with the ITA has resulted in our use of the IEA to bypass arteries closer to the aorta. However, with increasing experience, we have been more willing to place the IEA to more distal arteries with a planned proximal end-to-side anastomosis to an ITA or the contralateral IEA. One potentially useful anatomical variation is a bifurcation of the IEA usually inferior to the umbilicus, which occurs in 14% [9] to 57% [10] of patients. Although this arrangement hinders dissection and may limit the length of IEA with adequate caliber, it also provides the opportunity for a natural Y graft, which we used in five instances.

Two marked differences between the handling characteristics of the IEA and ITA are worthy of comment. First, the intimal and medial layers of the IEA seem more tightly adherent to one another than those of the ITA. The former does not easily separate into two layers as commonly occurs with the ITA when it is cut with scissors or handled with forceps, making it somewhat easier to handle. In addition, although the IEA quickly goes into profound spasm with loss of pulse during harvesting, there is rapid resolution of spasm when flow is restored, with the result that the IEA dilates to a larger caliber than the ITA and remains so dilated through the remainder of the operation. One might speculate whether endothelial-derived relaxing factor may be released as flow is restored [11] and whether the IEA endothelium is more active in this regard than the ITA endothelium.

Our complication rates in this group were similar to those reported previously by us and others for patients undergoing CABG using two ITA grafts. The only death occurred in a patient with severe left ventricular impairment undergoing a third operation for coronary artery disease. It is not possible to say whether use of one IEA graft affected this outcome. There was delayed healing in 1 diabetic man with subcutaneous fat necrosis at the lower end of the sternal incision; however, this wound healed without intervention. Within the first 10 postoperative days, several patients had minimal serous drainage from the abdominal incision, which resolved without intervention.

The one complication directly attributable to IEA use was an abdominal wound infection. It occurred in an obese woman (height, 162 cm; weight, 100 kg) undergoing her second coronary bypass procedure. The wound opened spontaneously down to the fascia and was allowed to heal by secondary intention. An incisional hernia has not yet been noted.

There are several potential complications of IEA usage, but we have not observed any of them. Rectus muscle necrosis could theoretically be a complication, especially in patients with ipsilateral ITA mobilization. However, human anatomical and clinical studies [10] suggest that the rectus muscle receives lateral collaterals through the costomarginal artery that allow survival despite distal ITA ligation and muscle mobilization; studies in the dog support this finding (Paletta CE, Freedman B; personal communication). The vas deferens passes adjacent but lateral to the IEA at the internal inguinal ring and could conceivably be injured during IEA mobilization [12]. If the venae comitantes are divided at their junction with the external iliac vein, there is the potential for inadvertent injury to the latter structure. This can be avoided by dividing the veins 1 cm to 2 cm from their junction with the external iliac vein.

There is the potential for injury to the external iliac artery, particularly if it is mobilized or directly opened during harvest, as has been suggested by some authors [13]. Our surgical approach does not include an inguinal incision, but it does allow for harvest of the IEA at a level 1 cm distal to its origin from the external iliac. The luminal diameter of the IEA at this level is as large or larger than the diameter of a free ITA graft and allows for direct anastomosis to the aorta. Thus more proximal dissection is both unnecessary and potentially harmful.

A serious limitation of this experience is the lack of angiographic follow-up. Evaluating patency is particularly important after the first year to assess the deleterious effects of harvesting trauma, the adequacy of anastomotic technique, and the proclivity for graft spasm. We do not have great concerns about the first two factors because of favorable experience using the ITA. As for spasm, this was identified in a single patient immediately after restoration of IEA flow. The spasm spanned a 5-mm segment of graft, was of moderate severity, and resolved completely after a papaverine-soaked sponge (1 mg/mL) was placed on the artery for 5 minutes.

Of greatest concern is long-term graft patency. This will probably depend on the potential for progressive atherosclerosis of the transplanted IEA graft. Early in the experience with ITA grafting, it was predicted that the ITA would gradually occlude just like the coronary vessels when subjected to the hemodynamic stresses of the coronary circulation. Freedom from progressive atherosclerosis has been demonstrated in the ITA for up to 17 years after bypass, but there is no assurance that results with the IEA will be comparable. The critical question remains whether the IEA will prove susceptible or resistant to atherosclerosis over a prolonged period.

Although only long-term follow-up will definitively answer this question, we hoped that the results of histological examination of the vessels might give some clues regarding the fate of the IEA. It is encouraging that light microscopic examination of IEA and ITA tissue evidenced few structural dissimilarities. The most outstanding observations gleaned from the direct histopathological comparison of IEAs and ITAs were (1) the similarity in the IEL, (2) the differential presence of elastic tissue within the tunica media, and (3) the detection of medial calcification in several IEAs. These findings may have major effects on the likelihood of the development of progressive atherosclerosis.

Internal Elastic Lamina

The IEL has been implicated as a factor in the development of atherosclerosis [14, 15]. This elastic layer separates the arterial intima from the media and may act as a two-way barrier protecting the media from the effect of any noxious luminal stimuli and also protecting the intima by preventing the inward migration of smooth muscle cells. It has been demonstrated that there are an increased number of fenestrations within the IEL of the coronary artery and that these may impair the protective effect of the IEL. In contrast, the ITA has fewer fenestrations, which are smaller in size and may result in a more atherosclerosis-resistant vessel [12]. In this regard, it is encouraging that there are no differences in the number of or morphological characteristics of the fenestrations within the IEL of the ITA and the IEA. It is possible that if diminished fenestration of the IEL yields a protective effect against atherosclerosis in the ITA, then a similar effect may occur in the IEA.

Elastic Layers in the Tunica Media

It has also been proposed that elastic tissue within the media may provide some protection from atherosclerosis by preventing the migration of smooth muscle cells into the intima [8]; such migration is considered to represent an early event in atherogenesis [16]. In addition, van Son and associates [8] suggested that medial elastic tissue is bradytrophic and dampens the forceful mechanical stretching of both IEL and medial smooth muscle during systole and therefore may minimize propulsion of blood components into the vessel wall. Such a mechanism might explain the delayed onset of intimal hyperplasia in elastic arteries. Van Son and colleagues have classified the ITA as just such an elastic vessel and believe this explains in part its remarkable resistance to atherosclerosis. Using this reasoning, the relative paucity of elastin in the IEA may forebode inferior patency results.

We do not consider the ITA a typical elastic artery. That term generally is reserved for the larger conducting vessels having abundant elastic lamellae within the media. Often these layers are so prominent that the IEL and the external elastic lamina are not readily distinguishable. The ITAs in our study did not fulfill these criteria. Although elastic lamellae were present within the media, they did not obscure the identification of the prominent IEL or external elastic lamina in most cases. We are inclined to believe that the number and size of the fenestrae within the IEL may play a more important role in atherogenesis by facilitating access of luminal stimuli to the media and allowing transmigration of smooth muscle cells into the intima. Thus we hope that the IEA may be as resistant to the atherosclerotic process as is the ITA, regardless of the amount of intramedial elastic tissue.

Medial Calcific Sclerosis

The foci of calcification identified within the tunica media of two of 14 IEAs examined suggests the presence of medial calcific sclerosis (Mönckeberg's arteriosclerosis). Although intraoperatively this did not interfere with suture placement or hinder use of the conduit, it is important to determine the ramifications of this lesion for long-term patency. According to the classification scheme of Silbert and co-authors [17], Mönckeberg's arteriosclerosis can progress and result in vascular occlusion if there is pathological evidence of an active inflammation, a state classified as stage III. The histological examination in our 2 patients revealed no ongoing inflammatory process, hyaline degeneration, or bone formation. The absence of these findings would result in a stage II classification, which, according to Silbert and colleagues, does not progress to thickening of the intima or blockage of the vascular lumen. Thus, these calcifications are unlikely to adversely affect the long-term results of the IEA grafts in these patients.

Summary

In summary, our short-term results support the supposition that the IEA is a satisfactory conduit for the purpose of coronary artery revascularization. Although the low operative morbidity and freedom from ischemic events after discharge are encouraging, follow-up is still quite limited. The fact that the IEA is an arterial graft that is very similar to the ITA in size, flow characteristics, and histology suggest that it will resemble the ITA with respect to its resistance to atherosclerosis and its outstanding long-term patency rates. It must be emphasized that there are no objective data to support such assumptions. Although the notion of complete revascularization using only arterial grafts is quite attractive, the durability of this free IEA graft is as yet unproved. It is certainly possible that the patency for this graft will rival that of the ITA, but long-term results could prove inferior to those achieved with saphenous vein grafts. The rationale for preferential use of IEA grafts over saphenous vein grafts currently rests on the supposition that the architectural similarities of the IEA to the ITA favor its long-term patency.

The early catheterization results of Puig and colleagues [7] documenting immediate IEA patency rates of 88% are certainly heartening. However, longer-term angiographic patency must be documented periodically as occurred in the early days of CABG when the durability of all arterial conduits was unknown. One-year, 5-year, and 10-year patency curves must be established before widespread preferential use of the IEA can be confidently advised.

Who then should receive IEA grafts? The obvious indication is found in the patient undergoing a second revascularization in whom the ITAs and remaining segments of saphenous vein provide inadequate lengths of conduit to achieve complete revascularization. It is also appropriate to employ the IEA in situations of concentrated experience with a strong commitment to close clinical and angiographic follow-up.

Addendum

On February 13, 1991, a 64-year-old man had quadruple CABG using both ITAs and IEAs. By 1 week postoperatively, there was necrosis of the skin overlying the rectus muscle from the umbilicus extending 10 cm inferiorly and having a maximum width of 8 cm. Two weeks postoperatively, the skin and subcutaneous tissue, anterior rectus fascia, and rectus muscles were debrided; the posterior rectus sheath and peritoneum were left intact. The wound was allowed to granulate, and the patient was discharged from the hospital 2 weeks later. He was readmitted to the hospital on May 8, and the skin was closed.

At the time of this complication, this patient represented 1 of 11 patients having bilateral ITA and IEA harvesting. At present, we are continuing to use this procedure, and 5 additional patients have had bilateral IEA and ITA harvesting. We plan to continue to harvest all four conduits when appropriate and to attempt to determine what conditions contribute to a critical reduction in blood flow to the abdominal wall. This patient has a 10-year history of Raynaud's phenomenon and describes "slow healing" of traumatic lesions of the hands. On this admission, the circulation to the arms was evaluated; the large vessels had normal symmetrical waveforms and the first right digit had diminished photoplethysmographic waveforms. After exposure to cold, waveform amplitude decreased in the first and the third digits bilaterally.

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DISCUSSION

DR NOEL L. MILLS (New Orleans, LA): I enjoyed the paper by Dr Barner and his associates and appreciate the opportunity to review the manuscript.

First, Barner and associates report the use of the inferior epigastric artery (IEA) in 18 patients in the abstract and in 47 patients in the manuscript. This is clear evidence of their satisfaction with the short-term use of the IEA for coronary bypass grafting. The technical aspects of using the IEA have been worked out and are straightforward. Rectus muscle necrosis has not been a problem.

Our technique has not differed substantially from that of Dr Barner. More than ever, cardiac surgeons struggle to find adequate graft for reoperations, and another arterial conduit would certainly be welcome, one that does not have the fate of saphenous vein grafts. On occasion we likewise have found the IEA capable of performing as a natural Y graft.

There is considerable comment in the manuscript comparing light microscopic details of the IEA with mammary grafts. I suspect that the difference in the amount of elastic tissue in the media of the two arteries is not as important as basic biochemical data.

One problem with this study is that there is not one early postoperative angiogram. Second, in 17 instances the IEA, an unproven graft, was used to bypass the left anterior descending coronary artery; more often than not, this is the most important artery supplying the heart. We studied seven of our first 20 IEA grafts angiographically in the first 8 days to 3 months postoperatively: two were occluded; two had string signs throughout their length, possibly the result of competition of flow or spasm; and three were widely patent.

We have concern about this artery functioning as a long-term coronary artery bypass graft, and although we applaud the pioneering efforts of Dr Barner and his associates, it is our opinion that the IEA at this time should be used to bypass coronary arteries of lesser importance.

DR BARNER: I thank Dr Mills for his comments. Dr Mills has been highly innovative in the use of different conduits for coronary bypass and has been appropriately critical in his scrutiny of new conduits. As I indicated, angiographic follow-up is needed, and we hope to obtain this at 1 year, our thought being that early angiography is somewhat premature, whereas the 1-year mark gives a better perspective. Obviously the final word is not in on this conduit.

Bilateral Sequential Lung Transplantation: The Procedure of Choice for Double-Lung Replacement

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We recently described a technique for bilateral sequential lung transplantation that replaces the en bloc double-lung operation, a procedure that was accompanied by frequent problems with airway healing. Twenty-seven patients have undergone 28 bilateral sequential lung transplantations over the past 14 months. Eighteen patients had transplantation because of end-stage emphysema; 6, cystic fibrosis; and 1 each, obliterative bronchiolitis, usual interstitial pneumonitis with pulmonary fibrosis, and bronchiectasis. Cardiopulmonary bypass was used electively in the first 5 patients until it was recognized that the procedure could be done safely without it, and in only 3 additional recipients has it been employed. Mean ischemic time for the first lung was 276 ± 43 minutes and for the second lung, 410 ± 64 minutes. There have been five deaths, three in the postoperative period (11% operative mortality) and two late. The other patients are alive and well and do not require oxygen 2 to 15 months after transplantation. Mean forced expiratory volume in 1 second rose from 16% ± 8% of predicted to 84% ± 17% at 12 weeks. Six-minute walk values increased from a mean of 251 ± 91 m to 666 ± 42 m at 24 weeks. The excellent exposure afforded to both hemithoraces by the thoracosternotomy incision and the rare need of cardiopulmonary bypass have allowed us to offer the option of transplantation to patients who formerly would have been turned down because of previous pulmonary resection or pleurectomy. On four occasions, ventilator-dependent patients underwent successful transplantation. The applicability of the procedure seemingly will be limited only by donor considerations and questions regarding how much better one lung is than two in patients other than those with chronic infection. Bilateral sequential lung transplantation can be performed with a minimum of early mortality and morbidity, which is comparable with single-lung transplantation.

(Ann Thorac Surg 1991;52:438–46)

Recently we [1] described a method for bilateral lung replacement that uses a sequential implantation technique through a transverse thoracosternotomy incision. As we have gained more experience with this procedure, its broad applicability and substantial advantages over previous techniques for double-lung transplantation have become apparent. Many patients who previously would not have been considered candidates for double-lung transplantation because of technical factors stand to benefit from this procedure.

Bilateral lung replacement is mandated in situations where infection is present, such as cystic fibrosis or bronchiectasis. Whether bilateral lung replacement provides a distinct advantage to patients with end-stage emphysema remains to be definitively determined, but it is our current practice to offer it to the younger patients (less than age 50 years) and to those with a large bullous component to their disease. This age limit, which is by no means absolute, stems in part from the higher incidence of serious coronary artery disease found in older individuals, which would be expected to contribute to perioperative problems, especially if cardiopulmonary bypass (CPB) were to be required. Any advantage conferred by bilateral lung transplantation for patients with other than infected end-stage processes depends on a comparative analysis of morbidity and mortality of single-lung and bilateral lung replacement measured against differences in overall function between the two groups over a long-term follow-up period. In this report we detail our initial experience with bilateral sequential lung transplantation in 27 patients.

Material and Methods

Patients

Between October 1989 and January 1991, 27 patients underwent 28 bilateral sequential lung replacements at Barnes Hospital, St. Louis, MO. The mean age of the patients was 41 years (range, 18 to 54 years). Seventeen were men and 10 were women. Eight patients had end-stage emphysema of unknown etiology, 10 patients had α-antitrypsin deficiency, 6 patients had cystic fibrosis, and

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1 patient each had obliterative bronchiolitis, usual interstitial pneumonitis with pulmonary fibrosis, and bronchiectasis. Recipients waited an average of 106 ± 75 days (range, 2 to 262 days) for donor lungs to become available.

Selection Criteria

To be considered for lung transplantation, patients must demonstrate evidence of progression of disease by decreased exercise tolerance or deteriorating pulmonary function studies, increasing oxygen requirements, increasing complications, or a combination of these factors such that survival beyond 12 to 18 months would not be expected. Patients undergo a structured inpatient evaluation before final acceptance into the program. Approximately 10% of patients referred to our program are actually accepted for transplantation [2]. Reasons for refusal include advanced age, poor cardiac function or coronary artery disease or both, or psychosocial difficulties. Although, as in the past, patients were routinely turned down because of previous thoracic procedures, which might increase technical problems relating to the transplantation, the development of this operation has rendered these problems exceedingly rare contraindications. All of the patients demonstrated marked clinical deterioration with increasing oxygen requirements over the several months before referral. Patients with other systemic disease or with major renal or hepatic disease are not accepted. Absence of serious coronary artery disease is documented in each patient by coronary arteriography, and right ventricular function is assessed by radionuclide scan. Nutritional status has to be adequate (weight within 10% to 20% of ideal body weight), and patients are required to participate in a supervised outpatient pulmonary rehabilitation program.

Patients accepted for transplantation are required to move to St. Louis and are followed up as outpatients on a regular basis. The rehabilitation program for pretransplantation patients consists of stationary bicycling, treadmill walking, or both with continuous transcutaneous oximetry. Psychological and psychiatric evaluation of all potential candidates is performed to rule out a major pathological process or drug abuse problems. Patients are required to have a support person accompany them and must demonstrate significant motivation for transplantation and the rigors of lifelong follow-up care.

Pulmonary Function Tests

Pulmonary function studies were carried out using a Gould-FRL Medical 1000 IV pulmonary function system. The system measures both inspiratory and expiratory flow directly and electronically integrates the flow signals to obtain volume measurements. A full set of pulmonary function tests including diffusing capacity was measured at the time of evaluation and at 3, 6, 12, 24, and 52 weeks after transplantation. Perfusion lung scans in the immediate postoperative period were performed with the patient in the supine position after the intravenous administration of 1.5 mCi of technetium 99 m–labeled microaggregated albumen. A portable gamma camera was used, and blood flow to each lung was calculated and reported as a percentage of total blood flow.

Exercise Testing and Evaluation

As part of the pretransplantation evaluation, functional ability is assessed with a 6-minute walk test and treadmill-walking protocol. For the 6-minute walk, patients walk at their own pace around a level, 150 m (500-foot) circuit. They can stop as often as necessary. During testing before transplantation, patients use as much supplemental oxygen as they require to minimize desaturation as determined by transcutaneous oximetry. Total distance traveled during the 6 minutes is recorded, as are heart rate and the number of stops required.

Treadmill walking is also assessed. Initially this was done for 30 minutes while the patient walked on a treadmill set at 1.6 km (1 mile) per hour with a 4% grade. Subsequently we have used more standardized exercise-testing protocols and have chosen the low performance protocol (LoP) as being most appropriate for this group of patients [3]. This protocol, if performed in its entirety, requires 20 minutes and consists of ten progressive stages. The speed begins at 1.76 km (1.1 mile) per hour at a 1% elevation and progresses to 7.2 km (4.5 miles) per hour at a 10% elevation. Metabolic equivalents are quantitated based on the stage completed and recorded along with the total time. Patients were tested at the time of their initial evaluation and periodically up until the time of transplantation. This allows for an assessment of the progress made during the rehabilitation phase before transplantation. After transplantation, patients continue a pulmonary rehabilitation program and are reassessed periodically with the 6-minute walk and treadmill protocol.

Surgical Technique

A detailed description of the technical aspects of the operation has been previously published [1]. Briefly, with the patient supine and the arms secured overhead to an ether screen, bilateral anterolateral thoracotomies are made accompanied by a transverse sternal division. The patient is maintained on one-lung ventilation while the contralateral hemithorax is opened. We purposely do not immediately open the pleural space on the side being ventilated, as this seems to allow for better ventilation and oxygenation, especially in patients with emphysema. This does require that the thoracotomy and hilar mobilization be completed after the first lung has been replaced. Despite the fact that this prolongs the ischemic time of the second lung, the advantage that it provides in ventilating the patient seems to outweigh any potential advantage of a slightly shorter ischemic time. Occasionally we have used high-frequency jet ventilation during the initial phase of the procedure.

We continue to use an omental pedicle to wrap each bronchial anastomosis. Through an upper midline incision, the gastrocolic omentum is mobilized off the transverse colon and split into two pedicles. The pedicles are tunneled substernally into each pleural space. We try whenever possible to avoid the use of CPB, usually basing the decision on our ability to adequately oxygenate the patient and by observing changes in pulmonary artery pressure that occur with a trial period of unilateral pulmonary artery clamping. The use of intraoperative transesophageal echocardiography has greatly contributed to the operative management by allowing continuous assessment of right ventricular function, which, if noted to deteriorate with pulmonary artery clamping, is an indication for instituting CPB. If CPB is required for replacement of either lung, a single, two-stage right atrial cannula and a standard aortic cannula are placed, and partial bypass sufficient to unload the right ventricle is employed.

The first lung is removed at the time the donor lungs arrive in the operating room. The lung block is split, and the first lung is implanted in the standard fashion by first completing the bronchial anastomosis, followed by the arterial and atrial anastomoses. After the first lung is reperfused and following a short period of equilibration, the opposite hemithorax is opened, the lung collapsed, and the patient ventilated solely on the newly implanted lung. The second recipient pneumonectomy is then performed with subsequent implantation of the second lung. The transverse sternotomy is closed with several heavy-gauge wires. The anterolateral thoracotomies are closed in a standard fashion using paracostal sutures.

An indwelling epidural catheter, placed preoperatively, is used for continuous infusion of narcotic to minimize postoperative pain, hasten extubation, and maximize respiratory effort. Aggressive diuresis is begun immediately after the procedure, as patients commonly are in positive fluid balance. Immunosuppression is instituted with intravenous cyclosporine on the day of transplantation along with azathioprine and Minnesota antilymphocyte globulin. Suspected acute rejection episodes are treated with methylprednisolone by bolus for 3 consecutive days. Maintenance steroids are withheld until day 6 after transplantation. Patients usually leave the intensive care unit 1 day after extubation.

Results

Patient Outcome

Cardiopulmonary bypass was used in 8 patients. The mean bypass time was 197 minutes (range, 92 to 365 minutes). For the initial five transplant procedures, we elected to use CPB after implantation of the first lung because of our concern of "flooding" the newly implanted lung. We have subsequently found that CPB rarely is necessary and now use it only when indicated by the clinical situation. In 21 patients, the right side was replaced first. The mean ischemic time for the first lung implanted was 276 ± 43 minutes (range, 180 to 360 minutes). The mean ischemic time for the second lung was 410 ± 64 minutes (range, 300 to 565 minutes). Time between implantation of the first and second lungs averaged slightly more than 2 hours (134 minutes), much of this consumed by the performance of the second recipient pneumonectomy. Quantitative perfusion scan data obtained immediately after completion of the transplant operation failed to demonstrate any major predominance of flow to the lung with the shorter ischemic time (mean, 53% ± 11%, range, 27% to 80%). Even when a substantial dichotomy in perfusion was present on the first scan, the perfusion tended to equalize within 48 hours.

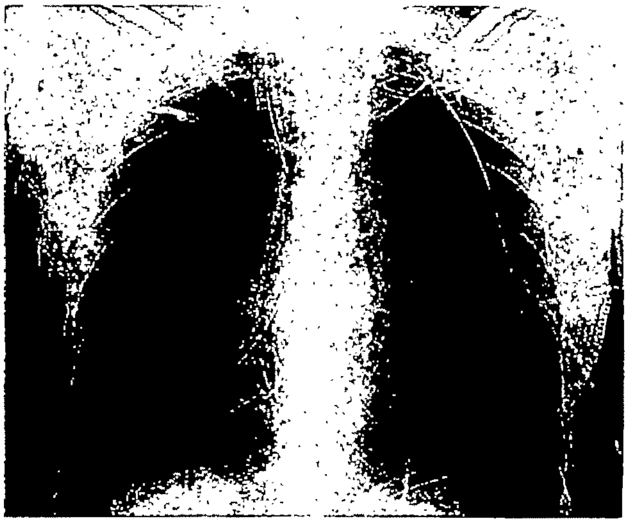


Fig 1. Early postoperative chest radiograph after bilateral sequential lung transplantation.

The early postoperative course for the majority of the recipients was remarkably uneventful. No patient required reexploration for bleeding. The typical appearance of an early postoperative chest radiograph is demonstrated in Figure 1. Patients were extubated at a mean of 3 ± 2 days (range, 1 to 7 days) and had a mean stay in the intensive care unit of 4.1 ± 3 days (range, 1 to 14 days). Discharge from the hospital occurred at a mean of 24 ± 8 days and ranged from 14 to 52 days. The number of days required to achieve a room air oxygen pressure greater than 60 mm Hg averaged 7 ± 6 with a range of 2 to 29 days.

The diagnosis of rejection depended mainly on clinical criteria, such as development of an infiltrate on chest radiograph, decreased oxygenation, or a slight temperature elevation. Transbronchial lung biopsies are not performed on a strict protocol basis in the early postoperative period but are done as indicated by the clinical situation, often when an infiltrate persists on the chest radiograph after treatment for rejection. Differentiating between infection and rejection remains difficult, but rejection occurs so commonly within the first 7 days after transplantation that we have a low threshold for administering steroids by bolus injection. If the chest radiograph does not improve within 12 to 24 hours after steroid administration, then bronchoalveolar lavage, protected brush specimens, and at times transbronchial lung biopsy are performed. In the early posttransplantation period, the diagnosis of rejection depends more on experience than any single diagnostic test. Transbronchial lung biopsy plays a major role in the diagnosis of rejection later in the course and is used frequently. Recipients in this series sustained a mean of 2.1 ± 1 rejection episodes (range, 0 to 4) during the period of hospitalization.

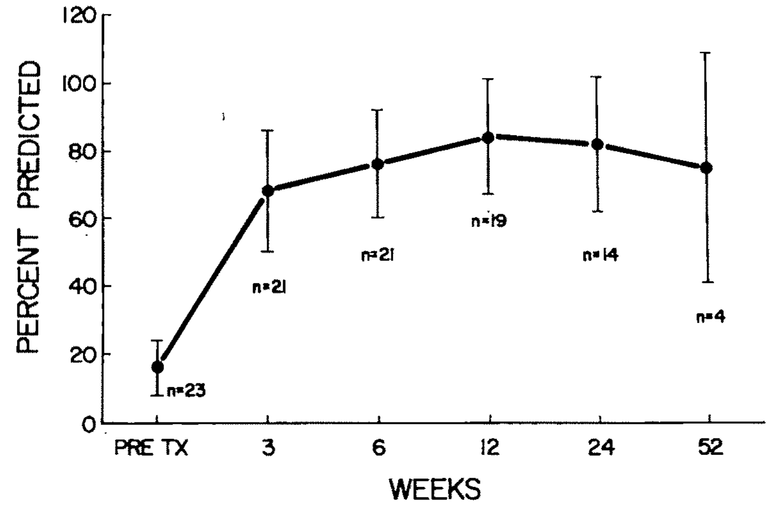


Fig 2. Change in percent of predicted forced expiratory volume in 1 second. Data are shown ± the standard deviation. (TX = transplantation.)

There have been five deaths among the 27 patients. Three occurred in the postoperative period, an 11% operative mortality. Actuarial survival at 1 year is 85%. One patient died of a presumed cardiac arrythmia 2 weeks after transplantation as he was making an otherwise uneventful recovery. Two patients died of what was presumably donor lung dysfunction. In 1, there was diffuse necrosis of both airways evident within 5 days after transplantation. He subsequently underwent retransplantation 1 week after the initial operation but ultimately died of overwhelming sepsis and multiple-organ failure.

There were two late deaths. One patient died of metastatic pancreatic carcinoma at 7 months, soon after presenting with multiple hepatic metastases. Exploratory laparotomy provided biopsy confirmation. The other died 1 year after transplantation, which was performed because of end-stage obliterative bronchiolitis, which developed after a previous en bloc double-lung transplantation 1.5 years earlier. Despite a change in her immunosuppression regimen, the patient again had changes consistent with obliterative bronchiolitis as well as lymphoma and pulmonary aspergillosis. The immediate cause of death was acute pancreatitis.

All other patients are alive and well 2 months to 15 months after transplantation though morbidity from the procedure is not insubstantial. Eight patients sustained ten anastomotic defects or dehiscences. In only 2 patients were these of clinical significance, with 1 requiring two dilations of a bronchial stricture. Cytomegalovirus (CMV) pneumonitis continues to be a problem after lung transplantation, and 8 patients in this series had CMV disease proven by transbronchial lung biopsy. We attempt at transplantation to provide CMV-negative recipients with only CMV-negative lungs, whenever possible, in an effort to keep CMV pneumonitis to a minimum. We do not, at present, employ any routine antiviral prophylaxis against CMV, even in high-risk situations, as no data are available to support this approach. Six patients had culture-proven bacterial pneumonitis, a situation most commonly seen in recipients with cystic fibrosis. Intraabdominal complications have also occurred commonly. Two patients had major intraabdominal bleeds necessitating laparotomy. Both occurred secondary to mesenteric artery aneurysms in recipients with α-antitrypsin deficiency, which may prove to be a contributing factor. One patient had clinical and radiographic evidence of angulation at the antrum and required laparotomy to divide the omental pedicle, which had caused this displacement of the stomach. Cardiac arrhythmias also were common, with supraventricular tachycardia noted in 6 patients. Other complications included pneumothorax (5), donor lung dysfunction (4), early sternal instability (3), wound infection (2), gastrointestinal bleeding (2), obliterative bronchiolitis (2), recurrent laryngeal nerve injury (1), and meningoencephalitis (1).

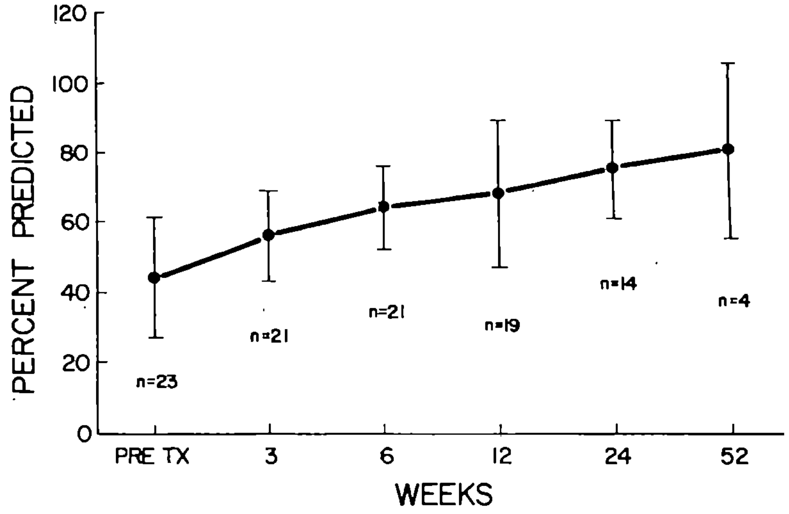
Pulmonary Function Results

Before transplantation, the forced expiratory volume in 1 second ranged from 8% of predicted to 51% (mean, 16% ± 8%). This corresponded to a value of 0.54 ± 0.21 L. By 3 weeks after transplantation, the mean percent of predicted forced expiratory volume in 1 second for the group rose to 68% ± 18% (Fig 2). This rose to a peak of 84% ± 17% of predicted at 12 weeks and then seemed to plateau with a value of 82% ± 20% of predicted at 24 weeks and 75% ± 34% of predicted at 52 weeks for the 4 patients evaluable for that period. The change in forced vital capacity after transplantation was not quite as marked (Fig 3). It rose from a pretransplantation value of 44% ± 17% of predicted to 75% ± 14% of predicted at 24 weeks and showed a continued rise at 52 weeks.

Functional Testing

At the time of evaluation, patients walked a mean of 251 ± 91 m in 6 minutes, which improved to 353 ± 122 m at the final measurement before transplantation (Fig 4). This dropped to 323 ± 110 m at 3 weeks after transplantation, but by 6 weeks, had increased to 520 ± 131 m and continued to increase at 12 weeks and 24 weeks to values

Fig 3. Change in percent of predicted forced vital capacity. Data are shown ± the standard deviation. (TX = transplantation.)



of 613 ± 128 m and 666 ± 42 m, respectively. All patients used supplemental oxygen for the pretransplantation exercise studies, whereas postoperative evaluations were performed without supplemental oxygen.

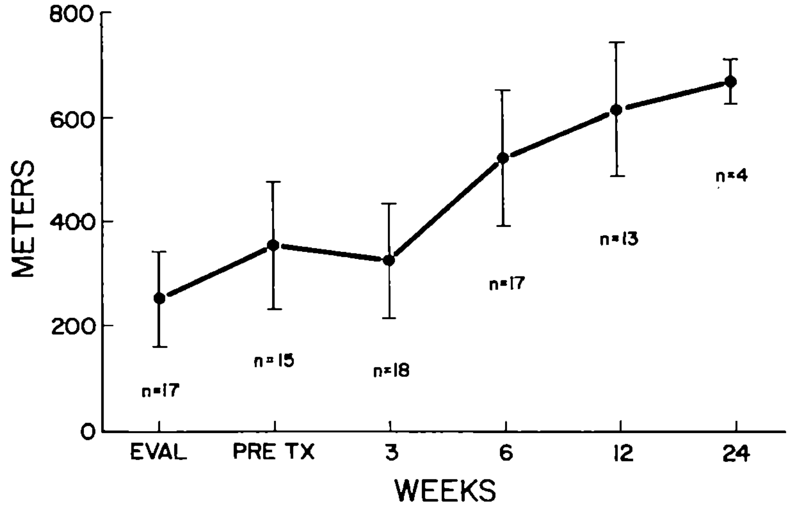
The pattern for the 30-minute treadmill-walking evaluation was similar. At the time of evaluation, patients covered a mean of 1,156 ± 464 m, which increased to 1,807 ± 417 m at the last measurement just before transplantation (Fig 5). At 3 weeks after operation, this dropped back almost to the evaluation level but increased to 2,149 ± 529 m by 6 weeks (range, 1,207 to 3,363 m). By 12 weeks, the mean had increased to 2,661 ± 230 m. Using a standardized treadmill protocol (LoP) and quantitating metabolic equivalents, Figure 6 shows the same trend with an increase between the time of evaluation and pretransplantation and a subsequent marked increase by 6 weeks and further increase at 12 weeks (8.5 ± 1.0). By 12 weeks, the mean value of metabolic equivalents translates into stage 8 of the ten-stage protocol.

Comment

The development of the bilateral sequential lung transplant procedure was prompted by the recognition of major complications occurring after the original en bloc double-lung operation. Not only were there problems with healing of the tracheal anastomosis, but complications after CPB were also noted. Several deaths early in our experience with the original operation were directly related to myocardial problems. Because of the recognition of difficulties with tracheal healing, we and others [4] initially modified the en bloc operation by performing bilateral bronchial anastomoses. This seemed to have an impact on the incidence of anastomotic problems, and it was thus a logical extension to proceed to a bilateral sequential replacement technique in an attempt to reproduce the high success rate that had already been achieved with single-lung transplantation, as the operation is essentially two single-lung transplantations performed in the same patient.

The initial theoretical objection to this approach was the mandatory increase in ischemic time that would accrue before the second lung was in place and the uncertainty regarding the maximum "safe" cold ischemic time. We believed that it was optimal to keep the time of ischemia less than 6 hours. Based on the current experience, however, we have found that ischemic times up to and

Fig 4. Results of 6-minute walk test. Data are shown ± the standard deviation. (EVAL = evaluation; TX = transplantation.)



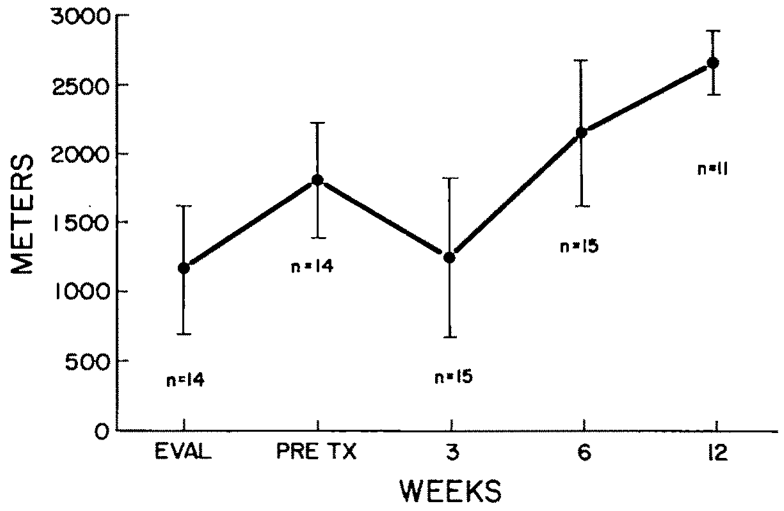


Fig 5. Total distance walked after 30 minutes on the treadmill at 1.6 km (1 mile) per hour with a 4% grade. Data are shown ± the standard deviation. (EVAL = *evaluation;* TX = *transplantation.)*

beyond 9 hours are well tolerated. In our laboratory [5], routine safe lung preservation for 24 hours has been achieved in a canine model. Ideally the ischemic times should be kept as short as possible, but the recognition that longer periods of ischemia are still compatible with excellent gas exchange in the majority of patients lends further support to the concept of sequential lung replacement. Although we observed four instances of what we characterized as donor lung dysfunction with a histological picture of diffuse alveolar damage, there was no correlation with ischemic time.

Even though we are replacing both lungs, we have not routinely used CPB subsequent to our initial 5 patients but have chosen instead to support the patient on the newly implanted lung as we sew in the second lung. We now recognize that despite an increase in permeability known to occur after ischemic injury [6], the newly implanted lung seems to tolerate the entire cardiac output and provides adequate gas exchange, thus allowing implantation of the second lung without CPB. It is only in those situations where clamping the pulmonary artery results in an unacceptable pressure rise or where decomposition of right ventricular function is noted on transesophageal echocardiography that CPB is required. The thoracosternotomy incision provides excellent exposure to the right atrium and aorta, facilitating cannulation.

The ability to perform this operation routinely without the need of CPB and the exposure afforded to both hemithoraces have allowed us to use the operation for patients who previously would have been rejected for transplantation because of technical factors related to the operation itself, such as a previous major pulmonary resection or pleurectomy. The bilateral thoracosternotomy approach not only makes a difficult dissection entirely feasible but allows it to be accomplished without great difficulty. It is the transverse sternal division that allows this excellent exposure. In this series, several patients who previously would not have been considered for transplantation were able to undergo the operation. They include 1 patient who had had a previous double-lung transplantation, a patient who had previously undergone a right middle lobectomy and total parietal pleurectomy, and another with recurrent pneumothoraces treated with multiple tube thoracostomies. On four occasions, ventila-

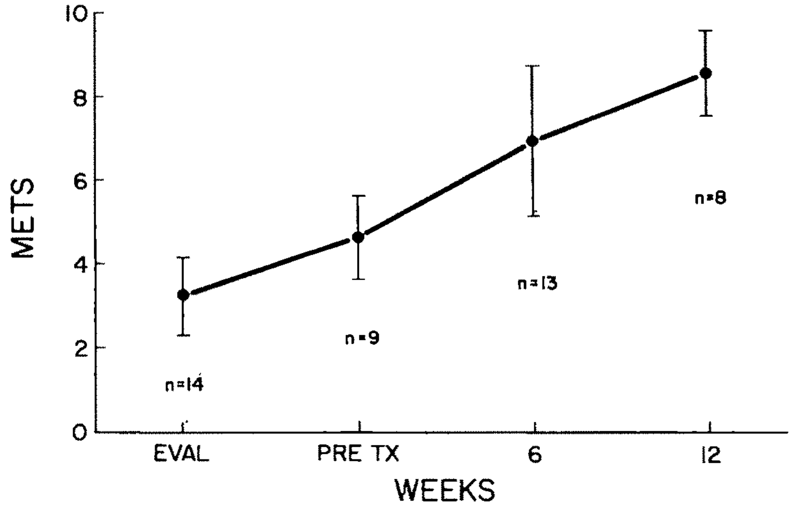


Fig 6. Metabolic equilavents (METS) generated during the lower performance (LoP) standardized treadmill protocol. Data are shown ± the standard deviation. (EVAL = *evaluation;* TX = *transplantation.)*

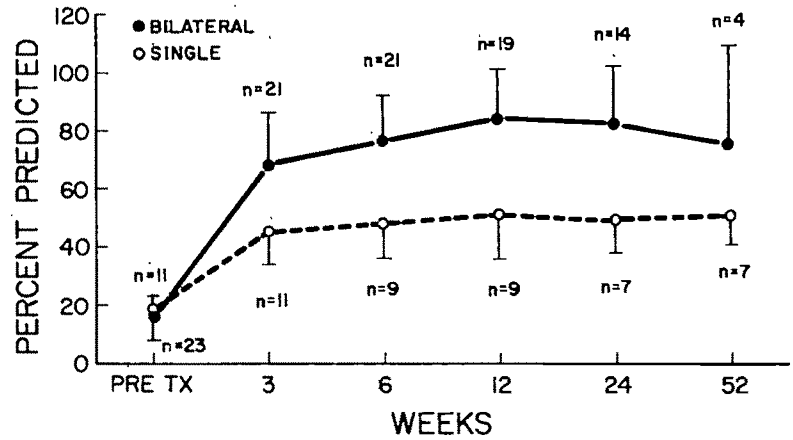


Fig 7. Comparison of change in percent predicted forced expiratory volume in 1 second for patients undergoing bilateral transplantation and those undergoing single-lung replacement for emphysema. Error bars indicate standard deviation. (TX = transplantation.)

tor-dependent patients had successful transplantation, 3 of whom were able to be weaned quickly from mechanical ventilation and survived; the fourth died after repeat transplantation necessitated by primary donor lung dysfunction accompanied by diffuse airway necrosis. One of these patients had undergone bilateral bullectomy 2 weeks previously, had massive air leaks requiring multiple chest tubes, and had had a major deterioration in condition during the period after bullectomy. In another patient, respiratory failure developed immediately after completion of her initial evaluation for transplantation. Previously we have not considered patients for transplantation who are ventilator dependent. The procedure is particularly ideal for patients with cystic fibrosis, who often have marked pleural symphysis resulting from the recurrent infections that characterize their course. The ability to perform the operation without the routine use of CPB effectively reduces the risk of major hemorrhage as a result of the difficult dissection.

With the original en bloc double-lung operation, airway healing constituted a major problem [7]. The current operation builds on the modification of bilateral bronchial anastomoses originally suggested by Noirclerc and colleagues [4], and airway problems have, for the most part, been minor and of little clinical significance. If not for the performance of routine bronchoscopies, all but two airway defects would have remained inapparent. We have not had to place a bronchial stent in any patient in this series; all airway defects have healed with conservative therapy, although 1 patient required dilation of a bronchial stricture.

The thoracosternotomy incision has not proved to be a major problem, either from the standpoint of pain in the initial perioperative course or in long-term healing. We have noted 3 patients with early sternal instability, but this has not proved to be a long-term problem. Pain in the perioperative period is well controlled with epidural narcotic analgesia.

Perhaps the biggest question that remains is whether or not bilateral lung replacement offers an advantage over single-lung replacement in patients with end-stage emphysema. Obviously in patients with infection, such as those with cystic fibrosis or bronchiectasis, this is not an issue. Figure 7 compares the change in forced expiratory volume in 1 second for patients undergoing bilateral sequential lung replacement with that for patients undergoing single-lung replacement for emphysema. Although there seems to be a slight advantage for those receiving two lungs, this advantage is amplified somewhat by the inclusion of the patients with cystic fibrosis, most of whom are considerably younger. Likewise, a review of the 6-minute walk and 30-minute treadmill data seems to suggest a trend favoring those who receive two lungs. It remains to be determined if the slightly increased morbidity associated with a bilateral sequential lung replacement is justified on the basis of improved function with two lungs instead of one. If indeed there is very little functional significance to having the second lung, especially in those patients with end-stage emphysema, then perhaps the increase in morbidity and mortality and the less efficient use of donor lungs cannot be justified. Further answers will be provided as long-term follow-up becomes available.

Our initial experience indicates that bilateral sequential lung transplantation can be performed safely, with a minimum of early mortality and acceptable morbidity, and compares favorably with single-lung transplantation in spite of being a more complex procedure with twice the number of airway anastomoses at risk. From May 1989 to May 1991, 70 patients had 37 single-lung transplantations with 1 perioperative death (97% operative survival) and 34 bilateral transplantations with 5 perioperative deaths (85% operative survival). Major problems remain especially in regard to infection from both bacterial and CMV pneumonitis, though we report no deaths directly attributable to either. We think that for patients who require double-lung replacement, the development of the bilateral sequential operation represents a major advance. The operation has special benefits for those patients with difficult technical problems and especially patients with cystic fibrosis. Despite initial concerns to the contrary, the added ischemic time for the second lung has not proved to be a problem, as we routinely have ischemic times in the 7-hour range seemingly without ill effect.

Currently, we continue to offer bilateral lung replacement to patients with end-stage lung disease with an infectious component as well as to most patients with emphysema who are less than 50 years old, to those with a large bullous component to their disease, or to those with the highest values of total lung capacity. With the successful development of this operation and its application to a variety of situations, the indications for heart-lung replacement continue to dwindle and now seem to be confined to patients with irreparable congenital heart disease accompanied by secondary pulmonary hypertension or patients with poor left ventricular function.

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DISCUSSION

DR G. ALEXANDER PATTERSON (Toronto, Ont, Canada): I congratulate Dr Kaiser on an excellent presentation and on a manuscript that will prove to be a valuable addition to the transplantation literature. The importance of this presentation is not in the detail of the postoperative functional results, as we already know that bilateral lung transplantation provides excellent results among operative survivors, but in the success of this technical modification, which the Washington University group has so nicely described.

The original double-lung transplantation technique was flawed because of its technical difficulty, its reliance on cardiopulmonary bypass, and the high incidence of airway complications in patients after transplantation. However, the concept of double-lung replacement with preservation of the recipient heart has always been a sound one. What was required was the technical innovation to resolve some of the problems that have plagued the initial procedure. Bilateral bronchial anastomoses had already been employed, and the Marseille group deserves a large amount of credit for this. This modification had reduced the incidence of airway complications. At the suggestion of Dr Grillo, my colleagues and I had employed a clamshell type of incision previously and demonstrated its outstanding exposure of the pleural space.

In September 1989, we performed an inadvertent bilateral sequential lung transplantation in a tall patient (192.5 cm or 6 feet 5 inches) with emphysema who had waited some 21 months for a donor. The planned left lung transplantation was completed; however, the graft looked ridiculously small in the pleural space. I had worked with Drs Pearson, Cooper, Ginsberg, and Todd long enough to know when trouble was about to occur, so we elected to turn the patient and perform a right lung transplantation through another thoracotomy. The postoperative result was excellent, but the technical implementation of this procedure was not correct at all. Drs Michael Pasque and Larry Kaiser and the Washington University group, under Dr Cooper's typical leadership, have evolved a procedure that is elegantly simple in its conduct.

Of our 38 bilateral lung transplantations, the most recent 20 have been performed using the sequential single-lung technique, the decision being to replace the lung with the least function first in hopes of avoiding a long cardiopulmonary bypass run. The incidence of major airway complications has diminished dramatically. In fact, since adopting a telescoping bronchial anastomotic technique with routine perioperative steroid administration and no omentopexy, we have had only one minor anastomotic stricture. However, our increase in deaths from sepsis reflects the number of patients with cystic fibrosis in our group and the tendency for these patients to experience septic complications from the resistant gram-negative organisms that continue to contaminate the airway after transplantation.

I have a couple of questions for Dr Kaiser. First, how do you determine intraoperatively which lung is to be removed first and transplanted? Second, what are the precise indications in this Washington University experience at the moment for the application of single-lung or bilateral lung replacement in patients with obstructive lung disease?

DR ERNST WOLNER (Vienna, Austria): I, too, congratulate Drs Kaiser and Cooper for their excellent results. We from Europe are grateful to Dr Cooper and Dr Patterson for teaching us how to do lung transplantation. We cannot compete with their results, but our limited experience in Vienna now comprises 7 patients who have had bilateral sequential lung transplantation. There are 5 long-term survivors.

Our ischemic time is very similar to the reported status. The only difference between our technique and the technique described is that we do not use the omentum for wrapping the bronchial anastomosis. We use an intercostal muscle flap.

I have three questions for Dr Kaiser. First, what is your policy about using steroids in patients before, during, and immediately after the operation in regard to bronchial problems? Second, can you foresee whether a candidate for double-lung transplantation will need extracorporeal circulation or not? In our series, we have used femoral–femoral partial bypass in 3 of the 7 patients. Last, we recently had 2 patients with severe obliterative bronchiolitis after heart-lung transplantation. We performed single-lung transplantation in both, and 1 is a long-term survivor. What is your policy in the case of patients with excellent heart function but obliterative bronchiolitis? Do you perform single-lung transplantation, double-lung transplantation, or heart-lung transplantation?

DR THOMAS M. EGAN (Chapel Hill, NC): This was an excellent presentation. Both the University of Toronto and Washington University groups have been instrumental in the development and application of isolated lung transplantation.

At the University of North Carolina in Chapel Hill, our experience is somewhat smaller. In the last year, we performed eight double-lung transplantations in 7 patients. Four patients had cystic fibrosis, 1 had chronic obstructive pulmonary disease, and 2 had other infectious end-stage lung disease, including a young man who had been ventilator-dependent for 4 months with end-stage adult respiratory distress syndrome. All 7 patients survived and were discharged, none require supplemental oxygen, and all are ambulatory. Cardiopulmonary bypass was necessary in 3 of the 8 transplant procedures. One patient required retransplantation for graft dysfunction on the seventh postoperative day.

The hospital stay for our patients is somewhat longer than that of the Washington University group. Our mean stay is 44 days. Three patients required a long hospitalization. One of them had a Haldol (haldoperidol)-related drug reaction, which resulted in prolonged ventilation; 1 had a prolonged period of ventilation before transplantation and was in a considerably debilitated condition at the time of transplantation; and the patient who had a redo transplantation required ventilation for a long period. The remaining 4 patients, however, had a mean hospital stay similar to that reported by the Washington University group. Fourteen airways that are at risk have been evaluated. There has been one partial dehiscence, which was in the patient having repeat transplantation. That airway is healing. At present, we have no strictures and, to date, have not needed to use stents.

I have a couple of questions for Dr Kaiser. First, what are your current contraindications to double-lung transplantation now that we recognize that this procedure can be applied to patients who have had previous thoracotomies and previous invasion of the pleural space? Second, and I guess this really echoes Dr Patterson's question, what do you do for a young patient with end-stage chronic obstructive pulmonary disease?

DR KAISER: I thank all the discussants for their comments, and I will try to answer the questions.

First, Dr Patterson, I very much appreciate your discussing the manuscript and congratulate you on the excellent results of the Toronto group, who have truly been the leaders in this field. In answer to your question as to which side we do first, if there is a major difference on the perfusion scan, we tend to do the side that has the least perfusion first, so that we can try to avoid cardiopulmonary bypass by saving the better lung and doing it second.

In terms of indications for single versus bilateral replacement—and this will address one of Dr Egan's questions as well—we had initially limited bilateral transplantation to patients less than 50 years old. I think we have extended that somewhat now. We have tended to do bilateral lung transplantation in patients with the larger total lung capacities as well as those with huge bullous disease, our concern being that perhaps single-lung transplantation in patients with emphysema with a big bullous component might lead to more air trapping. Certainly in the younger patients I think we have favored bilateral transplantation as a routine.

Dr Wolner, I congratulate you on your early results. In regard to the question about steroids, we do not use steroids perioperatively, although we recognize the excellent results that have been obtained by Dr Trinkle, Dr Patterson, and others. I guess old habits are hard to break. We have had good results by withholding steroids until postoperative day 6. We are now considering patients and do see patients who are on a regimen of preoperative steroids, so our policy may change.

With reference to cardiopulmonary bypass and how we assess the need, we use transesophageal echocardiography and a period of trial clamping of the pulmonary artery before removing the lung. Watching the right ventricular function and pulmonary artery pressures, we think that we can avoid bypass if clamping seems to be well tolerated. I think in the majority of instances we can do that.

Concerning obliterative bronchiolitis, we have now seen 3 cases in our series. One of the patients was a previous heart-lung recipient on whom we did a single-lung transplantation, 1 had pulmonary hypertension, and 1 had bilateral lung transplantation.

The main contraindications to the bilateral sequential operation are poor left ventricular function and age greater than 50 years, although, as I said, we are somewhat flexible with the age cutoff. Dr Egan, I also congratulate you on your results, which are certainly excellent.

Indications and Limitations of Aortic Valve Reconstruction

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To elucidate the value of conservative operation for aortic regurgitation, all consecutive patients operated on between July 1988 and July 1990 were reviewed. Of 251 patients with aortic regurgitation, 107 (42.6%) had nonprosthetic operation. The mean age was 23 years, and 90 patients (84.1%) were rheumatic. Two techniques were used: repair (annular and leaflet plasties, 69 cases) and cusp extension with glutaraldehyde-treated pericardium (25 bovine, 13 autologous). There were two hospital deaths (1.8%), both in the repair group, and no late deaths or embolic events. Only 5 patients (4.7%) were anticoagulated. In the repair group there were 12 reoperations, four (5.9%) due to aortic and eight to mitral dysfunction. In the cusp extension group there were two reoperations due to mitral dysfunction. Echocardiographic follow-up showed better results with cusp extension. In conclusion, conservative operation for aortic regurgitation is possible in a high percentage of young rheumatic patients and does not require anticoagulation. Cusp extension is more reliable than repair in terms of early results, although its long-term durability is not yet known.

(Ann Thorac Surg 1991;52:447–54)

Conservative operation on the aortic valve has received considerably less attention than the repair of the atrioventricular valves. With the exception of stenotic lesions in the very young and regurgitation secondary to septal defects, most surgeons treat all aortic valve lesions with a replacement. This attitude is justified by the satisfactory results of the available prostheses in the aortic area, the lack of valve tissue usually found, and the very precise geometry required to achieve competence.

Our long-standing interest in valve repair, together with an encounter with a young population in whom anticoagulation constitutes a major problem, stimulated an aggressive attitude toward aortic valve conservation. To elucidate the value of this approach, all consecutive patients operated on for aortic regurgitation during a 2-year period were reviewed. Analysis of the results should clarify the indications and limits of the different surgical techniques available.

Material and Methods

Patients

Between July 1988 and July 1990, 251 consecutive patients with aortic regurgitation were operated on at our Institution. One hundred forty-four underwent valve replacement and 107 (42.6%), a conservative procedure. This last group constitutes the basis for this report, and their preoperative characteristics are presented in Table 1. Their ages ranged from 4 to 60 years with a mean of 23.0 years. Forty-seven of these patients (43.9%) were less than 20 years of age. The cause was rheumatic in 84.1%; 81.3% were in New York Heart Association functional class III or IV and 87.8% were in sinus rhythm. Seventy-seven patients (71.9%) had pure aortic regurgitation and 30 had some degree of stenosis. Of the 107 patients, 42 (39.2%) had an isolated lesion whereas 65 (60.7%) also had a mitral and 22 (20.5%) a tricuspid lesion that also needed operation. Triple-valve operation was performed in 22 patients (20.5%).

The diagnosis was established by transthoracic two-dimensional color-flow Doppler echocardiography. The degree of aortic regurgitation was graded 1+ to 4+ according to the height of the regurgitant jet relative to the left ventricular outflow tract height, measured from a parasternal long axis just below the aortic valve [1]. The findings were confirmed intraoperatively by a transesophageal probe.

Surgical Techniques

All patients underwent operation under cardiopulmonary bypass with ascending aorta and single dual-stage right atrial cannulation. Only those with serious tricuspid disease had bicaval cannulation. An apical left ventricular vent and systemic moderate hypothermia (28°C) were used in all patients. After aortic cross-clamping, the aorta was opened transversely close to the clamp. The incision curved downward toward the noncoronary sinus and stopped a few millimeters from the base of the noncoronary cusp. Cold crystalloid cardioplegia injected into the coronary arteries and topical cooling of the heart with ice slush were used routinely in all patients for myocardial protection. An insulating pad was placed behind the

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Table 1. Preoperative Patient Characteristics[a]

Variable	Total (n = 107)	Repair (n = 69)	Cusp Extension (n = 38)	*p* Value
Age (y)[b]				
Mean	23	23.4	22.2	NS
Range	4-60	4-60	12-35	
Sex				
Male	64 (59.8)	39 (56.5)	25 (65.8)	NS
Female	43 (40.2)	30 (43.5)	13 (34.2)	NS
Functional class				
I	2 (1.8)	1 (1.4)	1 (2.6)	
II	18 (16.8)	11 (15.9)	7 (18.4)	
III	73 (67.5)	47 (68.1)	26 (68.4)	NS
IV	14 (13)	10 (14.4)	4 (10.5)	NS
Cause				
Rheumatic	90 (84.1)	54 (78.2)	36 (94.7)	<0.05
Congenital	15 (14)	14 (20.2)	1 (2.6)	
Degenerative	2 (1.8)	1 (1.4)	1 (2.6)	
Pathology				
Isolated	42 (39)	20 (28.9)	22 (57.8)	<0.05
Mitral	43 (40.1)	30 (43.4)	13 (34.2)	
Mitral + tricuspid	22 (20.5)	19 (27.5)	3 (7.8)	
ECG				
Sinus rhythm	94 (87.8)	59 (85.5)	35 (92.2)	NS
Atrial fibrillation	13 (12.1)	10 (14.4)	3 (7.8)	

[a] Numbers in parentheses are percentages. [b] Median age = 22 years, mode = 17 years.

ECG = electrocardiogram; NS = not significant.

heart, and the myocardial temperature was monitored continuously by a probe in the septum.

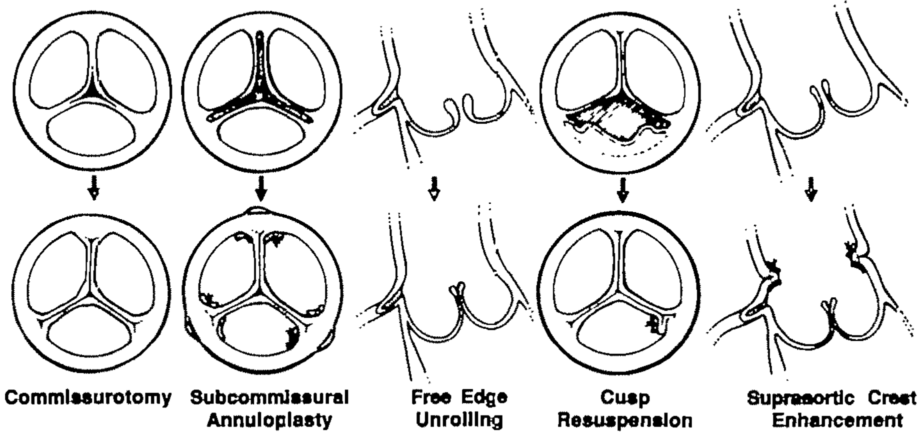
The aortic valve was inspected, but if an associated mitral valve lesion was present no decision was made at that time. A left atriotomy was performed and the mitral valve operation undertaken. A successful mitral valve repair was always a reinforcing factor toward aortic conservation. Careful exploration of the aortic valve determined the type of operation. Valves with an active infection, calcification, or bicuspid or unsuitable anatomy were excised and replaced with a prosthesis. If the valve leaflets were relatively thin and only retracted one third or less of their radial length, a repair was attempted. Various surgical maneuvers already described by us [2, 3] were used either alone or in conjunction to obtain competence. Resuspension of the prolapsed cusp, unrolling of the thickened free edge, subcommissural U annuloplasty stitches, and augmentation of the supraaortic crest were all used (Fig 1).

Valves with severe regurgitation due to thickening and gross retraction of more than one half of the leaflet area were candidates for cusp extension, also termed "reconstruction." In these valves, the thickened part of the cusp was resected leaving at least a 5-mm rim of the leaflet tissue. A rectangular strip of glutaraldehyde-treated pericardium was then cut in a tricuspid fashion and sutured to the leaflet remnant with a running 4-0 Prolene (Ethicon, Somerville, NJ) suture started at the midpoint of each leaflet. The new commissures were anchored to the aortic wall by stitches driven through the wall and tied over pledgets on the outside (Fig 2).

The quality of repair was assessed during the rewarming period by measuring the amount of vent return after unclamping the aorta. More recently the valve has been directly visualized through a "valvoscope" before unclamping while cardioplegia is injected under pressure. Once the patient was off bypass, transesophageal or epicardial color Doppler echocardiography was performed. All patients had an echocardiographic study performed before discharge and at each follow-up examination. Anticoagulation was dictated by the mitral operation.

Patients with isolated aortic operation were not anticoagulated. Those with pericardial reconstruction were given antiaggregants. All patients were followed up at 3-, 6-, and 12-month intervals by one physician. Any patient who failed to report was contacted by a social worker who had visited the patient during his or her hospital stay. All events were recorded according to the set of definitions established by the Ad Hoc Committee on Valvular Surgery [4].

Fig 1. Aortic valve repair techniques.



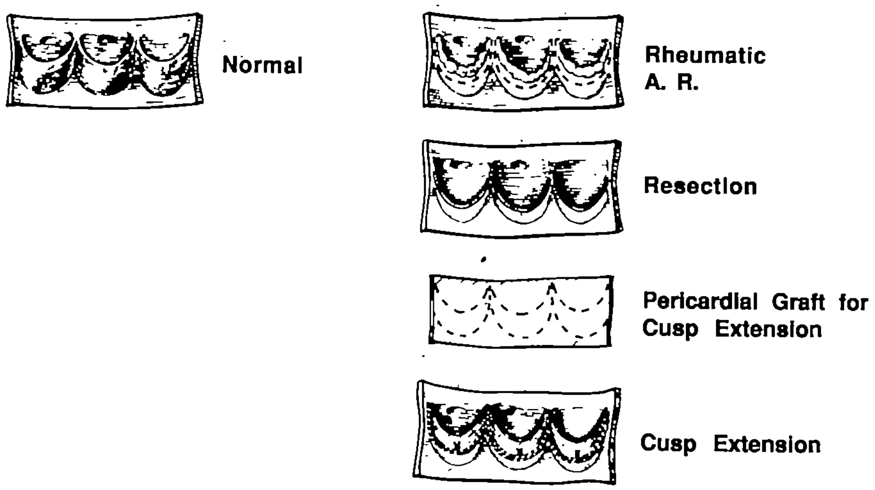


Fig 2. Aortic valve reconstruction with glutaraldehyde-treated pericardium. (A.R. = *aortic regurgitation.)*

Results

In 69 patients, repair was possible, whereas in the remaining 38 reconstruction with glutaraldehyde-treated pericardium was necessary. The first 25 of these latter patients had commercially available bovine pericardium used for their reconstruction, and the last 13 had autologous pericardium. The preoperative patient characteristics of the total group and of the repair and reconstruction groups are shown in Table 1. Although the mean ages were similar, reconstruction was only performed in young patients. More than 80% of the patients had a rheumatic lesion, but 94.7% of those with reconstruction were rheumatic versus 78.2% of those with repair. More isolated aortic lesions were treated by reconstruction (57.8%) than by repair (28.9%). Only 3 patients (7%) with triple-valve operation had reconstruction versus 19 (27.5%) in the repair group. The functional class was similar in both groups of patients (2.8 versus 2.9, reconstruction versus repair). The preoperative echocardiographic degree of aortic regurgitation was significantly different between the two groups (Table 2). The patients with reconstruction had more regurgitation (mean, 3.24 ± 0.8) than those with repair (mean, 2.29 ± 1.1). The surgical repair maneuvers performed in the repair group were 44 commissural annuloplasties; 30 commissurotomies, 18 cusp resuspensions, 16 free edge unrollings, 11 crest enhancements, and 10 other maneuvers. Most patients had more than one maneuver to attain competence (mean, 1.8). Of the 65 patients who had simultaneous mitral operation, 10 (15.4%) had their valve replaced and 55 (84.6%) underwent conservative mitral valve procedures (Table 3). The possibility of mitral repair was an incentive to also conserve the aortic valve. Twenty-one of 22 tricuspid valves were treated conservatively. It is worth noting that none of the patients who underwent aortic reconstruction with pericardium had prosthetic replacement of the other valves; their associated mitral lesion was repaired in 16 and the tricuspid valve was repaired in 3 of them. The mean cardiopulmonary bypass and ischemic time for the whole group was 128.48 ± 46.3 and 88.24 ± 32.94 minutes, respectively. The mean ischemic time for isolated aortic repair was 49.05 ± 19.6 minutes versus 99.32 ± 21.7 minutes for isolated aortic reconstruction. Reconstruction always took longer than repair.

There were two unsuccessful attempts at repair that were followed by cusp extension. However, the unsuccessful attempts at conservation that ended with an aortic replacement are not included in this series. Five patients in the repair group had an unsuccessful attempt at mitral repair followed by replacement. Transvalvular gradients were measured intraoperatively after the patient came off bypass in 16 aortic cusp extensions. The gradients varied between a few millimeters and 20 mm Hg. One single patient, with a mixed aortic lesion, showed a residual gradient of 25 mm Hg.

There were two (1.8%) hospital deaths in the whole group; both occurred after repair. One patient had a simultaneous mitral repair, and the other a mitral and tricuspid repair. The cause of death was low cardiac

Table 2. Echocardiographic Mean Degree of Aortic Regurgitation (graded 0 to 4+)

Group	Preoperative	Intraoperative	Discharge	Follow-up	p Value[a]
Repair	2.29 ± 1.1	0.75 ± 0.6	1.13 ± 0.9	1.19 ± 0.8	<0.05
Cusp extension	3.24 ± 0.8	0.59 ± 0.5	0.66 ± 0.5	0.59 ± 0.6	<0.05

[a] Preoperative versus follow-up.

Table 3. Associated Valve Operations Performed in the 107 Patients With Aortic Conservative Procedures[a]

Valve	Total (n = 107)	Repair (n = 69)	Extension (n = 38)
Mitral			
n	65 (60.7)	49 (71.0)	16 (42.1)
Repair	55 (84.6)	39 (79.5)	16 (100)
Replacement	10 (15.3)	10 (20.4)	. . .
Tricuspid			
n	22 (20.5)	19 (27.5)	3 (7.8)
Repair	21 (95.4)	18 (94.7)	3 (100)
Replacement	1 (4.5)	1 (5.2)	. . .

[a] Numbers in parentheses are percentages.

output in one and a spontaneous brain hemorrhage while in the ward in the other.

There were no hospital deaths in the reconstruction group. However in 3 patients severe but transient electrocardiographic ischemic changes were detected soon after coming off bypass. These patients, however, did not have permanent residual or recurrent ischemic changes during their hospital stay or in the follow-up period. Ninety-six patients (91.4%) left the hospital in sinus rhythm. The remaining 9 patients (8.6%) were in atrial fibrillation; all had associated mitral disease. Only 5 (4.7%) of the 105 surviving patients with aortic conservation were anticoagulated with warfarin. Fifty-six (53.3%) received antiplatelet drugs and 44 (41.9%) received no treatment.

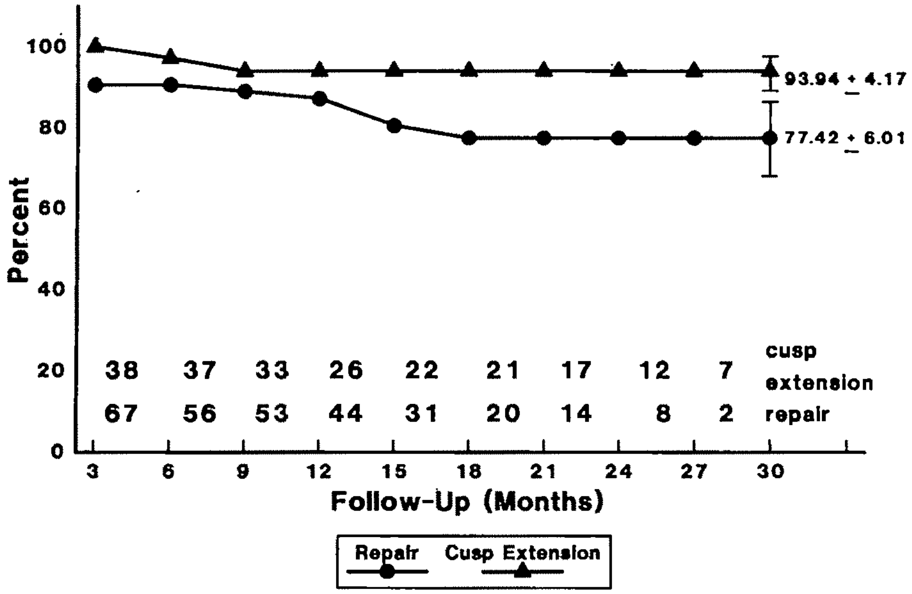
The follow-up of the patients has been complete from a minimum of 6 months to a maximum of 30 months. So far, no thromboembolic events have been detected in this group of patients and there have been no late deaths.

Fourteen patients required reoperation (14/105 or 13.3%) due to failure of the aortic or mitral repair. In the repair group, 4 patients had aortic valve dysfunction as the primary cause for reoperation (4/67 or 5.9%) and the valve was replaced, whereas in 8 patients dysfunction of the mitral valve repair was the cause of reoperation. In the 8 patients with mitral dysfunction, 4 did not have any procedure done to the repaired aortic valve at the second operation. In the other 4 with only moderate regurgitation, the valve was replaced to reduce the risk of a second reoperation. Three patients were reoperated on within 11 days of the first operation owing to an unsatisfactory surgical technique, whereas the other 9 patients were reoperated on between 2 and 9 months postoperatively. One of these patients was reoperated on at 7 months after rheumatic reactivation.

In the pericardial cusp extension group, 2 patients underwent reoperation at 4 and 8 months postoperatively. In both patients reoperation was necessitated by dysfunction of the mitral repair. In 1 of them the aortic valve was reoperated on because of the presence of 2+ regurgitation, present since the first operation. Resuspension of one of the pericardial free edges was performed. In the other patient the aorta was opened just to inspect the appearance of the pericardium. In both patients the bovine pericardium was pliable without evidence of degeneration or calcification. No thrombus formation was observed. There was no mortality in the reoperation group. One patient suffered a transient monoplegia with full recovery. The event-free actuarial curves for the repairs and for the reconstructions are presented in Figure 3. Given that no late deaths or thromboembolic events have yet occurred in this group of patients, these data correspond to freedom from reoperation.

The echocardiographic findings preoperatively and intraoperatively, at discharge and at follow-up, are pre-

Fig 3: Actuarial freedom from any reoperation on the aortic valve after repair and pericardial cusp extension.



sented in Table 2. Those patients who required reoperation due to aortic dysfunction were not included. At last follow-up, all 38 patients with pericardial reconstruction were in functional class I or II with 36 (94.7%) in functional class I. Sixty-three patients (94%) who underwent repair were also in class I or II. Four patients were in class III or IV, all of whom had had multiple-valve operation.

Comment

Conservative operation on the aortic valve has a long history parallel to the development of cardiac surgery. Before the advent of cardiopulmonary bypass the two techniques described for the treatment of regurgitation were circumclusion [5, 6] and bicuspidization [7, 8]. Lillehei and associates [9] in 1958, already using cardiopulmonary bypass, also used bicuspidization and described single-cusp enlargement with an Ivalon sponge. Mulder and colleagues [10] in 1960 described a variety of surgical maneuvers that they referred to as "valvuloplasty." The advent of the valve prostheses, with their ease of implantation and guaranteed immediate competence, soon displaced the rather unpredictable conservative maneuvers. More recently, the awareness of the long-term problems of the available prostheses and the standardization and universal acceptance of the repair techniques on the atrioventricular valves have awakened a new interest in aortic valve repair.

Our encounter with a mostly rheumatic, young population in whom anticoagulation represents a very serious problem in terms of compliance and frequent pregnancies stimulated an aggressive attitude toward valve conservation. The fact that 60% of our patients undergoing aortic operations required concomitant mitral operation, in which the mitral valve was very often successfully repaired, further stimulated avoidance of an aortic prosthesis. The mean age (23 years) and etiology (84% rheumatic) reflect both the type of population and our indications for aortic conservation. It was considered that beyond 35 years of age the durability of a bioprosthesis is reasonable and justifies its use when anticoagulation contraindicates the implantation of a mechanical prosthesis.

The conservative surgical techniques applied fell under two distinct categories. Those patients judged to have enough valvular tissue underwent a variety of techniques directed toward achieving competence without the use of any extravalvular tissue [2]. These techniques, grouped under the heading of "repair," should be taken as a whole, as usually each of them only achieves partial improvement and requires reinforcement by others. A review of the long-term results obtained in a group of 50 patients who underwent operation with these techniques between 1974 and 1986 showed a 13-year actuarial survival of 86% and only four reoperations due to severe aortic dysfunction [3]. These techniques included (1) commissurotomy, always performed in the presence of even minimal fusion to maximize cusp mobility; (2) unrolling of the free edge of each leaflet, which increases the area by a few millimeters; (3) annuloplasty by means of the placement of a pledgeted U stitch at the base of each commissure, which by plicating the aortic wall reduces its total circumference (this technique, which we described as original [2], had in fact already been described by Cabrol and associates in 1966 [11, 12]; very recently, Cosgrove and co-workers [13] reported its successful use in a group of 21 patients); (4) in those cases with prolapse, the resuspension of the cusp free edge was also performed following the technique of Trusler and colleagues [14]; and (5) finally, in some cases an enhancement of the supraaortic ridge was induced to improve the valve hemodynamics. We [15] recently showed in the experimental animal that this technique induces an earlier closure of the aortic valve probably due to an increase in the vortices within the sinuses of Valsalva. It can be postulated that this vortex increase would reduce the tendency toward the inward rolling of the leaflet free edges. In any case, this is a very fast surgical maneuver that, at worst, is innocuous. Among the 65 survivors in whom we used these techniques, 12 required reoperation but dysfunction of the concomitant mitral repair was the cause in 8 of them. Eight aortic valves (12.3%) were, however, replaced due to severe regurgitation in 4 and moderate regurgitation in the other four. It was believed that the risk of a second reoperation should be reduced in this very young group of patients in whom rheumatic reactivation is a permanent threat.

In the presence of very severe cusp retraction, these maneuvers cannot be used and extension of all three cusps was performed with a single strip of glutaraldehyde-treated pericardium. These patients were grouped as "reconstruction." Two main questions must be addressed when considering the use of this approach. The first is the need for a standard surgical technique that ensures a correct, reproducible, and safe result in terms of immediate competence. The second is the long-term durability of the selected material. The data from our series show that this surgical technique achieves immediate competence in all cases as shown by intraoperative echocardiography.

However, in 3 patients intermittent electrocardiographic ischemic changes were observed after bypass. It is interesting to note that Batista and associates [16] reported that the only four operative deaths in their series were due to severe myocardial dysfunction in 3 patients and a myocardial infarction in the fourth without any obvious coronary problem at autopsy. Since we started using the valvoscope before aortic unclamping, in 3 patients the left coronary part of the pericardium was observed as prolapsing outwardly toward the sinus of Valsalva. The aortotomy was reopened and a few millimeters of the pericardial free edge were resected. It can be postulated that too long a piece of pericardium can prolapse and induce left coronary ischemia.

The second important question is the durability of the glutaraldehyde-treated pericardium. During the 1960s a variety of techniques were used for single or multiple cusp extension, but with rather poor results. In 1964 Björk and Hultquist [17] reported the calcification of single-cusp autologous pericardium used in 2 patients. In 1967 Bailey [18] and Kay [19] described the use of aortic wall and homologous and heterologous cusps, respectively.

Bahnson and associates [20] in 1969 studied the use of pericardium, peritoneum, pleura, and fascia lata to construct single aortic cusps. In the same year Edwards [21] described a technique for the use of a single pericardial strip. The small number of cases and lack of adequate myocardial protection probably resulted in poor anatomical results, which invalidates any conclusion. The much larger negative experience of fresh autologous fascia lata described by Senning [22] emphasizes the importance of tissue pretreatment.

More recently, several authors have used glutaraldehyde-treated bovine pericardium with, so far, favorable results. Batista and co-workers [16] in 1986 described a technique for the enlargement of all three cusps with a single strip of pericardium. Batista later reported that no calcification had occurred in 206 patients with a maximum follow-up of 6 years [23]. Yacoub and associates [24, 25] reported 6 cases of early tears (mean, 3.3 months) and four degenerations among 135 patients followed up for a maximum of 7 years in whom glutaraldehyde-treated strips of calf pericardium had been used. In 1988 Al Fagih and co-workers [26] reported the use of bovine pericardium for single-cusp extension in 20 cases with a maximum follow-up of 23 months. On the other hand the rather limited durability of the Ionescu-Shiley bioprosthesis [27], particularly in the young patient [28], cast some doubt on the long-term results of glutaraldehyde-treated bovine pericardium. The absence of a rigid stent when used in cusp extension, however, not only reduces the transvalvular gradient, especially important in very young patients, but also reduces the tissue stress, hopefully increasing its durability. This hypothesis is supported by the reports of Angell and colleagues [29, 30] showing that the time to free-hand homograft failure is approximately 12 years versus 8 years when the homograft is mounted on a stent. Even in the event of failure, the excision of the calcified pericardium should be easy, given that the patient's leaflet remnants have been preserved. The recent report by Chachques and associates [31] of the biological advantages of glutaraldehyde-treated autologous pericardium and its clinical application for cusp extension in the mitral position [32] encouraged us to use it in the last 13 patients.

In this series, 2 patients with cusp extension were reoperated on because of dysfunction of the mitral valve at 4 and 8 months postoperatively. In 1 of them, the aorta was opened just to observe the aortic reconstruction. The pericardium was thin and pliable, and no operation was undertaken. In the other patient, who had 2+ aortic regurgitation present since the first operation, the pericardium was also thin and mobile, but one of the cusps was too long and therefore a plication of its free edge was performed.

The total hospital mortality of 2 patients (1.8%) reflects the young age of our population. Both patients were in the repair group, and 1 of them died due to a cerebral hemorrhage just before discharge. During the short (although complete) follow-up available, no thromboembolic events or late deaths have occurred. It is worth noticing that only 5 patients in the whole series, who were in atrial fibrillation and had a mitral bioprosthesis, were anticoagulated.

The follow-up of this group of patients is still too short to derive a meaningful long-term outcome. However, the object of this report is to show that for a certain group of patients in whom anticoagulation represents a serious problem, conservative operation on the aortic valve can be performed in a high percentage of cases. This is particularly relevant for those patients who have had a successful mitral valve repair, in whom a nonprosthetic option for the aortic valve regurgitation would be preferable. The absolute best type of patients for the repair techniques described are patients with successful mitral repair and moderate aortic regurgitation too serious to be ignored. When the regurgitation is severe these maneuvers are unlikely to achieve perfect competence and cusp extension is necessary. In these cases only the age of the patient and difficulty of anticoagulation justify the use of autologous pericardium until its long-term behavior is better known. Aortic valve conservation, even though free of valve-related morbidity and mortality, carries a risk of reoperation practically always of a technical nature. This incidence should be reduced with a greater reliance on intraoperative echocardiography and a better knowledge of the limits of this operation.

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DISCUSSION

DR DELOS M. COSGROVE III (Cleveland, OH): This report is another seminal contribution by Dr Duran to our knowledge and capability to repair insufficient aortic valves. For 20 years he has led the way in exploring new techniques and reporting his results. We have followed this work closely and borrowed heavily from it.

The increasing ability to repair aortic valves is apparent in this series, where 43% of insufficient valves were reconstructed. At the Cleveland Clinic the incidence has increased to 21% in the period 1988 through 1989. However, aortic valve repair lags substantially behind repair of atrioventricular valves. There are three reasons for this. First, the mechanism of insufficiency has not been well understood; further, there has been no reliable intraoperative method for valve function analysis; and third, there is less healthy tissue in the aortic position with which to reconstruct the valve.

Substantial progress has been made in each of these areas. Carpentier has classified the causes of insufficiency according to the range of motion of the leaflets. This has provided an intellectual framework upon which we can build our understanding of the mechanism of insufficiency. Cusp extension, as reported here, corrects the restricted leaflet motion. Intraoperative echocardiography has been an advance in our ability to visualize the aortic cusp function in the physiological state. It has become an integral part of valve reconstruction, providing the quality control essential for consistent results. A valvoscope, as described in the report, would be a further advance, allowing direct visualization. The third major impediment to aortic reconstruction, lack of sufficient healthy tissue, has in large part been solved by the technique of cusp extension. We have used this technique in single cusp reconstruction and found our results to be inconsistent. Assuming that pericardium is a stable valve substitute as reported by Chauvaud at the meeting of American Association for Thoracic Surgery, standardization of this technique is the remaining hurdle to widespread application.

Dr Duran, could you share with us your current thinking on the technique of cusp extension and the status of your valvoscope?

DR DURAN: Thank you very much, Dr Cosgrove, for your kind and very pertinent comments. You pinpoint the three main problems that face aortic valve repair. The first one, as in mitral repair, is identifying the exact lesions responsible for the regurgitation to apply the appropriate solution. We have advanced considerably in this field. The second one is the need for a reliable intraoperative method for testing valve competence. We now use a modified cystoscope that is introduced through the partially closed aortotomy; while the aorta remains clamped, cardioplegia is run under pressure so we can observe the closed repaired aortic valve. We found this method very useful, allowing us to reopen the aortotomy and apply further reconstructive maneuvers such as further trimming of the pericardial free edge after cusp extension. Once the patient is off bypass, intraoperative echocardiography, either transesophageal or epicardial, is essential. We found epicardial echocardiography particularly useful in aortic repair.

The third point is the surgical technique of pericardial cusp extension. Our initial experience with individual cusp extension was also rather poor, and we therefore moved toward a single pericardial three-cusp extension as described by Batista. We, however, modified it substantially based on the echocardiographic aortic annulus diameter. The autologous pericardial strip is molded into three curved cusps of the appropriate size by immersion in glutaraldehyde for 10 minutes. This strip is cut down according to the amount of patient's tissue left. This technique is simple and has been shown to achieve immediate competence.

Warm Heart Surgery and Results of Operation for Recent Myocardial Infarction

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Revascularization procedures after recent myocardial infarction are associated with higher mortality and morbidity compared with elective coronary artery bypass grafting. Traditional methods of myocardial protection impose a further ischemic insult on already compromised myocardium. Continuous cold blood cardioplegia may eliminate ischemia but may still leave the heart anaerobic. Theoretically, warm aerobic arrest addresses both of these issues and may become an attractive alternative to standard hypothermic ischemic arrest in this setting. In 115 nonrandomized patients undergoing coronary artery bypass grafting within 6 hours to 7 days of an acute myocardial infarction, myocardial protection was provided with continuous cold (4°C) or continuous warm (37°C) blood cardioplegia. Fifty-one patients (after 1988) protected with warm blood cardioplegia were compared with a historical cohort of 64 patients (before 1988) protected with cold blood cardioplegia. Results indicate that the warm cardioplegia group had no mortality versus 10.9% for the cold group ($p < 0.05$), a myocardial infarction rate of 2.0% in the warm versus 9.3% in the cold group, and use of intraaortic balloon pump of 0% versus 12.5%, respectively ($p < 0.05$). It is concluded that continuous warm aerobic arrest may minimize ischemia and anaerobic metabolism during the operative procedure, and may be of benefit to patients who have a limited tolerance to ischemic insult.

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The initial, yet on-going, goal of myocardial protective techniques during cardiac operations has been to prolong safe operating time by minimizing ischemic damage to the heart. This goal ultimately becomes an attempt at maintaining the myocardial energy supply at a level greater than its demand during the time of operation. Cardioplegic preservation therefore strives to optimize the ratio of energy supply to consumption and the capacity of the heart to utilize oxygen and substrate, whether aerobic or anaerobic. This capacity will be determined by the temperature and composition of the perfusate, and the distribution, adequacy, and duration of the infusion.

During routine coronary artery bypass grafting procedures, the aorta is cross-clamped and intermittent cold cardioplegia (crystalloid or blood) is administered—cold anaerobic arrest. This allows for a dry and quiet operative field but leaves the myocardium potentially anaerobic for the duration of the cross-clamp period. The ability of blood cardioplegia to deliver and the myocardium to utilize oxygen and substrate is limited at low temperatures resulting in essentially cold, ischemic, anaerobic arrest [1]. It is known that patients who have sustained a recent myocardial infarction have higher morbidity and mortality rates associated with the revascularization procedure, which is directly related to the extent of the preoperative ischemic injury [2]. This is likely due to the fact that the already ischemic (infarcted or infarcting) myocardium is subjected to a second ischemic insult during the operative procedure.

Continuous warm blood cardioplegia—warm aerobic arrest—has recently been introduced [3, 4] as an alternative method of myocardial protection, which may be of benefit in these high-risk patients. We herein report our initial clinical experience in historical sequential cohorts of patients operated on for angina after a recent myocardial infarction in whom the methods of myocardial protection and systemic perfusion differed only with respect to temperature.

Material and Methods

Patients

Over a 4-year period, 115 patients underwent coronary artery bypass grafting for angina within 6 hours to 1 week of an acute myocardial infarction. Patients with mechanical defects, such as ischemic mitral insufficency and ventricular septal defect, were excluded from this series. Fifty-one of these patients were operated on after 1988 using continuous warm blood cardioplegia. These patients were compared with a historical cohort of 64 patients operated on before 1988 using continuous cold blood cardioplegia.

The diagnosis of preoperative myocardial infarction was made by the appearance of a new Q wave on two or more contiguous electrocardiographic leads or elevation in the level of the MB isoenzyme of creatine kinase greater than 50 U/L when associated with prolonged ST elevation, left bundle-branch block, or poor R wave progression. In 1 patient in the cold group and 2 patients in the warm

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group, the operation was performed early due to an episode of prolonged chest pain at rest with electrocardiographic changes despite maximal medical therapy, and it was not appreciated until later that a preoperative enzyme spike had occurred.

Cardiac catheterization was performed in all patients to assess left ventricular function and the extent of coronary artery disease. The left ventricular ejection fraction was estimated from a single-plane contrast left ventriculogram on a scale of 1 to 4 (1, >0.60; 2, 0.40 to 0.60; 3, 0.20 to 0.39; and 4, <0.20). The degree of coronary artery narrowing was assessed from at least two views of each vessel and deemed serious if equal to or greater than 50% stenosis of the luminal diameter. Sixty-one percent of the patients in the cold group and 79% of the patients in the warm group underwent urgent revascularization. The urgent group consisted of patients with postinfarction unstable angina or unstable angina with unrecognized non–Q-wave infarct (3 patients) who were revascularized within 72 hours of cardiac catheterization. Unstable angina was defined as prolonged (>15 minutes) rest angina with transient changes in the electrocardiogram. These patients normally came to the operating room from the coronary care unit receiving intravenous nitroglycerin, on intraaortic balloon pump counterpulsation, or both.

Surgical Technique

The technical aspects have been discussed in detail [4, 5]. Briefly, standard cardiopulmonary bypass techniques were used through right atrial and aortic cannulation. After institution of cardiopulmonary bypass, systemic core temperature was lowered to 28°C in the cold group and maintained at 37°C in the warm group. The aorta was cross-clamped with the heart empty and beating, and 1.5 L of blood cardioplegia (hematocrit, 0.22 to 0.25; KCl, 27 to 30 mEq/L) was infused into the ascending aorta at 4°C in the cold group and 37°C in the warm group, which invariably resulted in cardiac arrest within 30 seconds. In the cold group an iced saline pericardial bath was used to keep myocardial temperature at 15°C. Distal anastomoses were performed in order of angiographic importance, and the vein grafts were attached to a perfusion manifold from the cardioplegia line to permit independent control of aortic and graft flow [5]. Low-potassium blood cardioplegia (7 to 10 mEq KCl/L) at 37°C or 4°C was infused into the ascending aorta and the bypass grafts when constructed at 50 to 100 mL/min. If blood obscured the operative field when the coronary artery was incised, aortic root cardioplegia was discontinued and the root vented. Similarly, cardioplegia flow in grafts to neighboring territories was interrupted if necessary and local control achieved with saline irrigation. On occasion cardioplegia flow was totally discontinued for up to 15 minutes. When the heart was returned to the pericardial cradle after completion of a distal anastomosis and while the graft was sized, cardioplegia was initially delivered into the aortic root at an accelerated rate, not unlike the standard use of a cardioplegia bolus. The internal mammary artery anastomoses were constructed last and left clamped until completion of all proximal anastomoses and removal of the aortic cross-clamp. Both the distal and proximal anastomoses were constructed during a single aortic cross-clamp period. The aorta was vented during construction of the proximal anastomosis while cardioplegia perfusion continued down the vein grafts, leaving the region at highest risk perfused to the end.

Data and Analysis

The data were collected in a prospective manner for the coronary surgery database and included information on operative deaths (death from any cause occurring within 30 days of the operation or during the hospital stay), perioperative myocardial infarction (defined as appearance of a new Q wave, left bundle-branch block, or poor R wave progression in association with elevation in level of creatine kinase MB isoenzyme greater than 50 units or exceeding 7% of the total creatine kinase concentration), and low output syndrome (ie, systolic blood pressure <90 mm Hg and cardiac index $<2.0\ L \cdot min^{-1} \cdot m^{-2}$ despite adequate preload and appropriate afterload reduction that required inotropic support or intraaortic balloon pump support).

Data are presented as mean ± standard deviation of the mean. Chi-square analysis without the continuity correction was used to compare the differences in number of deaths, perioperative myocardial infarction, spontaneous return to sinus rhythm, low-output syndrome, and the use of intraaortic balloon pump. Nonpaired t test was used to compare cross-clamp times and reperfusion times, defined as the time from removal of the aortic cross-clamp to attempted discontinuation of bypass.

Results

The demographics of both groups were similar with respect to age, sex distribution, and left ventricular grade (Table 1). There was, however, the tendency to operate on more New York Heart Association class IV patients in the warm group, which was also reflected in the number of urgent operations in this group (79% versus 61%) (Table 2). This is likely due to a more aggressive approach to unstable angina adopted recently, rather than preferring medical stabilization, as was done before 1988.

There was no mortality in the warm group compared with 7 patients (10.9%) in the cold group ($p < 0.05$). The cause of death in 4 patients was related to perioperative myocardial infarction, systemic hypoperfusion, and arrhythmia. Two patients died intraoperatively and 1 patient died of sepsis due to intraabdominal pathology. The operative mortality associated with a preoperative Q-wave myocardial infarction was 14% as compared with 9% in preoperative non–Q-wave infarction. The perioperative myocardial infarction rate was 9.3% in the cold group and 2% in the warm group. One non–Q-wave myocardial infarction occurred perioperatively in the warm group. Low-output syndrome occurred in 9 patients (14%) in the cold group and only 4 patients (6.3%) in the warm group. However, the difference in perioperative myocardial infarction and low-output syndrome between the two groups did not achieve statistical signif-

Table 1. Demographic and Angiographic Data

Variable	Cold Group	Warm Group
Demographics		
No. of patients	64	51
Male/female	46/18	39/12
Age (years ± SD)	63 ± 12	67 ± 10
Q-wave MI	21 (33%)	21 (41%)
NYHA class		
I	0	0
II	0	0
III	22	9
IV	42	42
Angiographic data		
LV grade		
1	21	10
2	19	13
3	17	17
4	7	11
Vessels involved		
Left main	6	5
1	0	2
2	14	7
3	44	37

LV = left ventricular; MI = myocardial infarction; NYHA = New York Heart Association; SD = standard deviation.

icance due to the small number of patients. Postoperative intraaortic balloon pump support was needed in 8 patients (12.5%) in the cold group and in no patient in the warm group ($p < 0.05$). For those patients coming to the operating room with intraaortic balloon pump support, the need for postoperative support was evaluated by turning the pump off and evaluating the degree of hemodynamic stability. Return to spontaneous normal sinus rhythm was significantly increased in the warm group, and the reperfusion time necessary after removal of the aortic clamp was significantly decreased in the warm group despite similar cross-clamp times in both groups (see Table 2). Although there was an increase in use of the left internal mammary artery as a bypass conduit in the warm group ($p < 0.05$), this likely reflects a change in surgical attitude toward this conduit in recent years.

Table 2. Operative Data and Results

Variable	Cold Group	Warm Group
Operative data		
Urgent	39 (61%)	40 (78%)
Reoperation	8 (13%)	10 (20%)
No. of grafts	3.3 ± 0.6	3.4 ± 0.7
LIMA	7 (11%)	15 (29%)[a]
Cross-clamp (minutes)	58 ± 13.7	64 ± 15.2
Reperfusion (minutes)	37 ± 8.2	13 ± 2.3[a]
Results		
Mortality	7 (10.9%)	0 (0%)[a]
Periop MI	6 (9.3%)	1 (2%)
Low output syndrome	9 (14%)	4 (6.3%)
IABP	8 (12.5%)	0 (0%)[a]
Spontaneous NSR	9 (14%)	50 (98%)[b]

[a] $p < 0.05$; [b] $p < 0.005$

IABP = intraaortic balloon pump; LIMA = left internal mammary artery; MI = myocardial infarction; NSR = normal sinus rhythm.

Comment

Preoperative myocardial infarction has been shown to be an independent predictor of operative mortality in coronary artery bypass grafting after adjustment for other risk variables in several studies [6–8]. In a multiinstitutional review, Kirklin and associates [9] demonstrated that a recent myocardial infarction increased the risk for early death after operation. Nevertheless, early operation has been proposed for this high-risk subgroup in the belief that infarct extension, reinfarction, and subsequent death are more common in the medically treated patients [10, 11].

Although initial surgical experience was disappointing with this patient population [7, 12], recently published results are more encouraging, with operative mortality varying between 0% and 10.5% but certainly exceeding that of elective operation [12, 13]. This may be in part due to improvements in myocardial protection and provides an impetus for exploring alternative methods of myocardial protection in this high-risk group of patients.

Cold hypothermic arrest, as practiced with intermittent blood or crystalloid cardioplegia, requires a mandatory period of ischemic arrest between infusions of the cardioplegia and also invokes the detrimental effects of hypothermia. Theoretical considerations and potential advantages of continuous warm blood cardioplegia have been discussed in detail elsewhere [4, 14]. Briefly, electromechanical arrest alone [15] accounts for nearly all of the oxygen consumption of the heart, with little further benefit gained even with profound hypothermia [16]. Continuous aerobic perfusion of the arrested heart is, therefore, theoretically an ideal protected state of the heart that maximizes the oxygen supply and minimizes demand. Yau and colleagues [17] have shown that with average flow rates greater than 80 mL/min, warm blood cardioplegia keeps the heart relatively aerobic, inhibits lactate production, and spares energy stores.

The technical constraints of coronary artery operation may, however, on occasion necessitate local vessel control or the interruption of global perfusion, resulting in a transient period of normothermic regional or global ischemia, and potential reperfusion injury. Intuitively this would seem particularly detrimental. However, as already discussed, there is only a slight further decrease in myocardial oxygen consumption in cold arrested hearts compared with those arrested warm [16]. Therefore, as originally demonstrated by Melrose and co-workers [15], an equal period of ischemia (<15 minutes) should be tolerated as safely with warm electromechanical arrest as with intermittent hypothermic cardioplegia.

The mortality reported in the continuous cold cardioplegia group in this study is comparable with that in the literature [6, 7]. However, the reduced mortality in the warm cardioplegia group, most likely due to reduced and limited perioperative myocardial infarction (non–Q-wave), suggests that there may be advantages to this method of myocardial protection that are not as yet clearly understood. It is interesting to speculate that maintenance of the integrity of basal cardiac metabolism with normothermia may enhance the tolerance to ischemia, as well as the ability to shift from aerobic to anaerobic metabolism, and the capacity to deal with reperfusion after short periods of ischemia. Given that continuous perfusion cannot be achieved in all types of cardiac procedures, there may be a benefit conferred during ischemia at 37°C that offsets the slightly higher oxygen consumption. Opie [18] has suggested that the rate of anaerobic metabolism during ischemia ultimately determines cellular viability. We would postulate that perhaps intermittent normothermic ischemia permits the highest rate of anaerobic glycolysis and is the best adaptive state of the myocardium. Shaper and associates [19] have calculated that in the absence of electromechanical activity, anaerobic energy-producing pathways and cellular energy stores should be able to maintain the viability of myocardial tissues for many hours. Alternatively, intermittent warm ischemia and reperfusion may precondition the myocardium, improving the tolerance to ischemia [20, 21]. The mechanisms responsible for this tolerance are likely temperature dependent.

Many improvements in preoperative and postoperative care, anesthetic management, and the conduct of cardiopulmonary bypass have taken place over the time frame of this study. Therefore, the proof of a definitive clinical advantage of continuous warm blood cardioplegia awaits the results of randomized trials. It is suggested, however, that continuous warm aerobic arrest may .confer an advantage on patients with limited tolerance to ischemia and anaerobic metabolism.

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DISCUSSION

DR RICHARD M. ENGELMAN (Springfield, MA): Thanks to the authors, I have had the opportunity to read the manuscript, which was highly beneficial in formulating a rational discussion. What they have shown in a retrospective analysis is that normothermic perfusion and normothermic blood cardioplegic preservation provided significantly improved mortality and morbidity compared with hypothermic perfusion and preservation. During this 4-year time frame they used only antegrade, not retrograde cardioplegia, thus missing the presumed benefits of immediate perfusion of ischemic, stunned, or infarcted myocardium. Regardless of this, they had zero mortality and one perioperative infarct in the 51 warm patients, 10 of whom were undergoing reoperations, and all of whom were having postinfarction angina from 6 hours to 1 week after infarction. We must therefore consider what is so special about normothermia versus hypothermia.

With hypothermia, there is a marked shift to the left of the oxyhemoglobin dissociation curve. This means that cold blood will have to come to a very low oxygen tension to release its oxygen into the myocardium. This is not true at normothermic levels, where the oxygen content of arterial blood in the pump is greater than 15 mL of oxygen per 100 mL of blood, most of which oxygen is available for aerobic metabolism.

In our own prospective analysis of three cardioplegic approaches using antegrade for arrest and retrograde for maintenance cardioplegia, my colleagues and I were unable to distinguish cold blood from warm blood preservation but found that there was increased malonaldehyde generation during reperfusion only in the cold crystalloid group. Further, we found that there was increased creatine kinase-MB generation during the first 24 hours after operation only in the cold crystalloid group, whereas cold blood and warm blood preservation were essentially identical.

Dr Lichtenstein, first, could you please elaborate on your thoughts, which were briefly mentioned in the manuscript, regarding warm preservation and myocardial preconditioning? If indeed the process of warm preservation leads to preconditioning, a feature of this reported by Dr Shaper's laboratory from Germany could be increased messenger RNA, a reflection of altered gene expression, leading to improved myocardial energy levels and sarcolemmal membrane integrity, clearly a beneficial effect of warm preservation. Second, could you possibly explain how you and Dr Salerno avoided infarction in the warm group when only antegrade cardioplegia was administered and the left internal mammary artery, which was used in 15 patients, was allowed to remain clamped until all proximal and distal vein grafts were completed? It is remarkable to me that you were able to avoid infarction in this group of patients, particularly in patients who were so critically ill.

DR GEORGE E. CIMOCHOWSKI (Wilkes-Barre, PA): I rise to congratulate Lichtenstein and associates on their innovative thinking. Actually, I got involved in this procedure about 3 months ago in a patient with a very high level of cold agglutinins, with my associate, John Anderson. We commenced a small, prospective, consecutive series of patients at Wilkes-Barre General Hospital over the past couple of months. We have only been there about 6 months, so we compared our first 93 patients receiving antegrade cold cardioplegia with 52 patients receiving retrograde warm cardioplegia.

They were a group similar to that of Dr Lichtenstein: 68 years of age, 29% female, 26% diabetic, 26% with a recent myocardial infarction, and 37% with left ventricular hypertrophy. We used 400 mL of antegrade warm cardioplegia to stop the heart and then used continuous retrograde in each case, occasionally turning it off for a distal, maintained a pressure of 18 to 22 mm Hg, which is about half of what you would see with cold. We had flow rates easily of 100 to 150 mL/min, and there were no venous injuries using the retrograde cardioplegia. Now, we used blood cardioplegia for both. In the retrograde group we used 22 mEq of potassium for the high dose of 400 mL antegrade, and then continuous at 7 mEq retrograde.

One thing we did notice, when we took the cross-clamp off, the potassium level was 5.7 mEq/L, and that was substantially higher than what we are used to. And, if fact, when you take the clamp off, there are very bizarre electrocardiographic complexes (Some of our perfusionists call them tombstone electrocardiograms). These are due to the hyperkalemia, but within a few minutes this washes out. In only 1 patient out of 52 did we have to treat that, as within a few minutes you see a normal-looking electrocardiogram. The mean time to stop the heart is 38 seconds, just like that of Lichtenstein and associates. Finally, we vented the aortic root, used no topical saline solution, and maintained systemic perfusion at 34°C in case there was a perfusion accident. The warm cardioplegia was going in at 37°C.

We prospectively evaluated 52 patients. Forty-six were undergoing isolated coronary artery bypass grafting, 6 valve operations, double valve, and valve coronaries. It is interesting that with this technique we were able to use internal mammary arteries in all of the coronary cases. In fact, we are averaging 1.4 arterial grafts per patient.

We compared the two groups. The mortality was low in both, 1.1% and 0%. Perioperative myocardial infarction rate is about the same in each group. There was significantly less phrenic nerve paralysis in the warm group, as one would guess because we were not cooling. Inotropic usage dropped from 52% to 11%. We counted using inotropes as little as 5 minutes coming off the pump. So 89% of the warm group came off with only a nitroglycerin drip. Atrial fibrillation was markedly reduced from 21% to 5.7%. If you like to do arterial vascularization, this facilitates it. We had a patient with a free gastroepiploic artery graft to the obtuse marginal, a left internal mammary artery graft to the left anterior descending, a free right internal mammary artery graft to the right coronary artery, and one vein graft.

Now, we found it particularly useful on a difficult case such as was described this morning with proximal dissections. We had a patient with a large left hypertrophic ventricle from aortic stenosis. I created a serious iatrogenic complication. Early in the series, I gave warm antegrade cardioplegia into the aortic root. I perforated the left main, dissected it, and dehisced the aorta off the annulus. This was in a 30-year-old man whose father worked in our hospital. We took down the left internal mammary artery graft to the left anterior descending on the pump, placed a vein graft to the circumflex artery, and fixed the aorta. After 5½ hours on the pump and a cross-clamp time of 4½ hours with continuous warm retrograde cardioplegia, he came off bypass with only a nitroglycerin drip.

I have a few questions for Dr Lichtenstein. Is warm retrograde cardioplegia better distributed because we do not have a cold vasospastic phenomenon? We think there are no contraindications. Do you think there are any? Atrial activity occurs; we have ignored it. What is your experience? How do you minimize hyperkalemia after removal of the aortic cross-clamp? Finally, would you comment on the thought of this being a superior way to protect the donor heart for heart transplants?

DR LICHTENSTEIN: I thank Dr Engelman and Dr Cimochowski for their kind comments. Dr Engelman asked me to comment on the issue of potential preconditioning. It is certainly of interest to us. As I mentioned, if one can run warm blood cardioplegia continuously, it is hard to argue with that as representing a near-ideal state of protection. This is usually easily achieved in valvular operations; however, in coronary bypass operations technical constraints necessitate repeatedly interrupting the flow. There is evidence in the literature, as Dr Engelman alluded to, that suggests that short periods of intermittent normothermic ischemia actually precondition the heart and seem to protect against a greater insult at a later period of time. In other words, in animal experiments short periods of ischemia at normothermia before ligation of the left anterior descending coronary artery results in a smaller infarct than acute ligation of the left anterior descending coronary artery. This phenomenon is being looked at in Dr Slutsky's laboratory in Toronto, and preliminary results suggest that normothermic ischemia gives rise to potentially protective heat shock proteins, a nonspecific stress-induced protein. I really cannot comment any further on that other than it is an interesting and likely important finding.

There was no significant difference in the perioperative infarct rate in the warm group over the cold continuous group, although the trend for reduction was certainly there and with larger numbers it may very well prove to be significant. Those of us who use a semicontinuous method of cardioplegia, whether it be down vein grafts or retrograde coronary sinus perfusion, have long ago come to realize that blood is ubiquitous and distributes everywhere in the vasculature. The question in terms of protecting against perioperative infarct really becomes one of adequate amount of perfusion for the electromechanically arrested state. Whenever possible we like to graft a diagonal or a posterior descending artery feeding the left anterior descending coronary artery territory if an internal mammary artery is being used with the antegrade approach. Although there are theoretical advantages of distribution beyond stenosis to retrograde cardioplegia, many of these are clinically unimportant and are counterbalanced by the drawbacks related to retrograde perfusion itself.

Dr Cimochowski asked a number of pertinent questions. Since we started using continuous warm cardioplegia both antegrade and retrograde we noted a reduced coronary vascular resistance, which may possibly be due to the absence of cold vasospasm. In fact, with warm retrograde cardioplegia our coronary sinus pressures rarely get above 20 mm Hg even at flow rates in excess of 150 mL/min, which initially caused us great concern regarding the position and location of the cannula. The only contraindication to retrograde warm cardioplegia would be a congenital anomaly such as an unroofed coronary sinus, in which case very little of the cardioplegia would be delivered into the coronary vasculature. It was actually Dr Engelman who called and made us aware of this potential problem with retrograde warm perfusion, which he unfortunately experienced in one of his patients. One would of course not use retrograde cold cardioplegia in that situation either. When atrial activity occasionally occurs we too have ignored it. We have not seen any clinical sequelae attributable to intraoperative atrial activity, with most patients spontaneously reverting to normal sinus rhythm after removal of the cross-clamp. Hyperkalemia has not been a problem for us thanks to the interest and involvement of our anesthetists, who pretreat with glucose and insulin or diuretics if the systemic potassium level is elevated before the removal of the cross-clamp.

In terms of donor heart protection, Dr Feindel in Toronto and Dr Murphy in Halifax frequently perfuse the donor heart with warm blood cardioplegia as soon as it comes into the operating room. Drs Feindel and Fremes in Toronto are looking at a normothermic perfusion system in arrested hearts for long-term preservation. Unfortunately, at the present time it still remains cumbersome and not clinically practical.

I think a major advantage of warm blood cardioplegia, particularly if it can be run nearly continuously, is in the problematic cases with prolonged cross-clamp times and in complex high risk procedures, as in the case presented by Dr Cimochowski. As his experience and ours have shown, ischemic time is relatively independent of cross-clamp time. The cross-clamp can be on for 4½ hours and the true ischemic time can in fact be only minutes. I think that is a major technical advantage of this conceptual framework.

Current Treatment for Wolff-Parkinson-White Syndrome: Results and Surgical Implications

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From July 1986 to January 1991, 123 patients with Wolff-Parkinson-White syndrome underwent operation for ablation of aberrant conduction pathways. There were 85 male and 38 female patients ranging in age from 11 months to 68 years. Associated anomalies included Ebstein's anomaly, sudden death syndrome, coronary artery disease, cardiomyopathy, abdominal aortic aneurysm, neurofibromatosis, other arrhythmias, or other complex congenital heart disease. Forty-one patients had multiple accessory pathways. Operative results showed a 7% initial failure rate, which dropped to 3% after reoperation. One patient had undergone previous operation for Wolff-Parkinson-White syndrome at another institution. Procedures performed concomitantly included mitral or tricuspid valve repair or replacement (6), right ventricular conduit replacement, subaortic resection, Fontan repair, corrected transposition repair, coronary artery bypass, and placement of an automatic internal cardioverter defibrillator. There was no operative mortality. Late follow-up is 27 ± 16 months, and complications included mitral regurgitation and myocardial infarction. By comparison, in the last 12 months 124 patients with the Wolff-Parkinson-White syndrome underwent catheter ablation using radiofrequency current. There were 9 patients with multiple pathways. One hundred twelve patients (90%) had all accessory atrioventricular connections ablated and have remained free of symptomatic tachycardia. There have been 12 failures (10%), of which 5 have had operation and 7 are being treated medically. Mean follow-up is 7 ± 5 months, and complications included circumflex coronary artery occlusion, excessive bleeding, valve perforation, and cerebral vascular accident. Both procedures offer excellent success with low morbidity. Although continued late follow-up is mandatory to assess the results of radiofrequency ablation, the early results suggest that operation for Wolff-Parkinson-White syndrome may become indicated for radiofrequency ablation failure, multiple pathways, or when additional procedures are required.

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The Wolff-Parkinson-White (WPW) syndrome, originally described in 1930 [1], may account for up to 20% of all cases of paroxysmal supraventricular tachycardia, and may have a prevalence as high as 1 in 500 patients in the general population [2]. Although patients with WPW syndrome may be asymptomatic, many suffer serious or fatal consequences. The first successful surgical division of an accessory atrioventricular (AV) pathway for the treatment of WPW syndrome was performed in 1968 [3]. By 1981, the operative procedure for the treatment of WPW syndrome was fairly standardized, and the results in the first 200 patients demonstrated an overall success rate of 86%, including a reoperation rate of approximately 15% [4]. The vast majority of these patients became totally asymptomatic and enjoyed a normal life.

Since that initial surgical series for the correction of WPW syndrome was reported, this procedure has been applied to an increasing number of patients, with hundreds of operations for WPW syndrome being performed each year. Operation has become the primary therapeutic option for many of these otherwise young and healthy patients, most of whom fail medical therapy or tolerate antiarrhythmic medications poorly. However, recent preliminary reports have suggested that catheter ablation of accessory AV connections using radiofrequency current can also be accomplished with a high success rate in patients with WPW syndrome [5–7]. Therefore, this study was undertaken to assess the results of surgical treatment for WPW syndrome and to compare and contrast these results with the initial group of patients undergoing radiofrequency catheter ablation of accessory AV connections in our institution.

Methods

Patient Data

This study retrospectively examined the results in 247 consecutive, nonrandomized patients treated for the WPW syndrome at the University of Michigan Medical Center from July 1986 to January 1991. There were 124 patients who underwent surgical correction of the WPW syndrome or paroxysmal supraventricular tachycardia secondary to a concealed accessory AV connection (Table

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Table 1. Surgical Versus Radiofrequency Catheter Ablation in the Treatment of Wolff-Parkinson-White Syndrome

Variable	Operation	Radiofrequency Ablation
No. of patients	123	124
Age (y)	26 ± 13	36 ± 14^a
Male (%)	69	59
Duration of symptoms (y)	11 ± 10	14 ± 13
Failed drugs (No.)	2.7 ± 1.8	2.1 ± 1.6
Follow-up (mo)	26 ± 7	7 ± 5^a
Other abnormality (patients)	13 (11%)	5 (4%)
Accessory pathways (total)	166	133
Multiple accessory pathways	41	9^a
Anterior septum	10	3
Left free wall	87	85
Right free wall	21	17
Posterior septum	43	22^a
Intermediate septum	5	6

^{a} $p < 0.05$ versus operation.

1). Twelve of these operations were undertaken in the last 12 months. Ninety-nine of these patients demonstrated overt ventricular preexcitation. The mean age of the surgical patients was 26 ± 13 years (mean ± standard deviation; range, 1 to 68 years). There were 38 female and 85 male patients. Associated abnormalities were present in 13 patients and included Ebstein's anomaly, sudden death syndrome, coronary artery disease, cardiomyopathy, atrial fibrillation, pulmonary atresia, subaortic stenosis, abdominal aortic aneurysm, neurofibromatosis, and mitral and tricuspid regurgitation. All patients had a history of symptomatic paroxysmal supraventricular tachycardia or atrial fibrillation. Seven patients had a history of syncope, and 3 had survived a cardiac arrest. The mean duration of symptoms in these surgical patients was 11 ± 10 years. These patients had previously failed or been intolerant to pharmacologic therapy with a mean of 2.7 ± 1.8 antiarrhythmic agents or had previously failed direct-current catheter ablation techniques.

In contrast, there have been 124 patients who underwent radiofrequency ablation treatment for WPW syndrome between March 1990 and January 1991 (see Table 1). Seventy patients demonstrated overt ventricular preexcitation. Two thirds of the patients had never had an electrophysiological test before referral. The mean age of these patients was 36 ± 14 years (range, 13 to 74 years). There were 51 female and 73 male patients. Associated abnormalities were present in 5 patients and included Ebstein's anomaly, dilated cardiomyopathy, hypertrophic cardiomyopathy, and coronary artery disease. All patients had a history of symptomatic paroxysmal supraventricular tachycardia or atrial fibrillation. Eighteen patients had a history of syncope and 2 had survived a cardiac arrest. The mean duration of symptoms was 14 ± 13 years. Eighty-two patients had previously failed or been intolerant to a mean of 2.1 ± 1.6 antiarrhythmic agents. The first 100 patients were included in an earlier report [8].

Electrophysiological Testing

Electrophysiological tests were performed in the fasting state. All antiarrhythmic medications were discontinued at least 24 hours before the procedure. Three catheters with 1 cm interelectrode spacing were inserted into a femoral vein and positioned in the high right atrium, His bundle position, and right ventricle. Another catheter was positioned in the coronary sinus through the right internal jugular vein. Chest leads and the intracardiac electrograms were recorded as pacing was performed with a programmable stimulator (Bloom Associates, Ltd) using stimuli that were twice the diastolic threshold and 2 milliseconds in duration.

The effective refractory periods of the AV node and accessory AV connection as well as the anterograde and retrograde block cycle lengths of the accessory AV connection were determined, the tachycardia was defined, and the accessory AV connection was localized to a general region by standard methods, as previously described [8]. The accessory AV connections were localized to one of five general regions: right free wall, posteroseptal, anteroseptal, left lateral free wall, or intermediate septal, which was defined as a pathway that was believed to pass from the anteroseptal to the posteroseptal space.

Surgical Protocol

The surgical techniques for WPW syndrome have been well described previously [9–11]. A median sternotomy was performed in all but 2 patients, in whom a right thoracotomy was used. The intraoperative electrophysiological mapping techniques employed fixed right atrial and right ventricular epicardial surface leads. Complete epicardial mapping was carried out by hand on the normothermic heart. Atrial pacing was initiated, after which either induced reciprocating tachycardia or ventricular pacing was used to effect retrograde conduction across the accessory pathway to construct a retrograde atrial map. Accessory AV connections that were capable of antegrade conduction were mapped primarily during sinus rhythm or atrial pacing. Concealed accessory AV connections were mapped during orthodromic tachycardia or ventricular pacing. Endocardial mapping was used in those patients requiring atriotomy to locate the His bundle and further augment the epicardial map.

Systemic cooling and cold potassium cardioplegic arrest were used in all patients. These measures facilitated identification of surgical landmarks and allowed for precise division of the accessory pathways, which was accomplished with blunt dissection using a nerve hook and electrocautery. In patients with either a right free wall, anterior septal, intermediate septal, or posterior septal accessory pathway, the approach was through a right atriotomy. In patients with a left free wall pathway the approach was through a left atriotomy. Our standard surgical technique involved an endocardial approach and extended the dissection circumferentially along the valve annulus well beyond the identified location of the earliest activation from the accessory pathway to include the entire anatomical space containing the pathway, as has been previously described [11]. Repeat mapping or pacing was carried out after separation from cardiopulmonary bypass to confirm the absence of preexcitation. A cardiac electrophysiologist was present in the operating room for all prebypass and postbypass mappings.

Ablation Protocol

After preliminary localization of the accessory AV connection, precise mapping within the targeted region was performed with the catheter used for delivering radiofrequency energy (Mansfield-Webster) by positioning the ablation catheter against the mitral or tricuspid annulus. Left-sided accessory AV connections were approached from the ventricular aspect of the mitral annulus; with the ablation catheter passed retrograde across the aortic valve into the left ventricle. Right-sided and posteroseptal accessory AV connections were approached from the atrial aspect of the tricuspid valve, and for ablation of posteroseptal accessory AV connections the catheter was positioned in the vicinity of the ostium of the coronary sinus.

Ablation sites were identified by the presence of discrete atrial and ventricular electrograms, early ventricular activation relative to the delta wave, or a discrete potential preceding the local ventricular electrogram suggestive of an accessory AV connection potential. Catheter ablation was performed using radiofrequency energy delivered as a continuous sine wave at 350 kHz (Radionics RF-3B, Burlington, VT). Twenty-five to 36 W was delivered for 10 to 20 seconds or until a sudden rise in impedance, indicative of coagulum formation. Whenever an application of radiofrequency energy at a given site was successful, a second application was delivered at that site before removal of the catheter. The duration of the ablation procedures was limited to 4 hours.

Follow-up

In all patients, 12-lead electrocardiograms were obtained and evaluated for the presence of preexcitation after either surgical or radiofrequency ablation. Surgical ablation patients underwent a follow-up electrophysiological test, using the temporary atrial and ventricular epicardial wires placed during operation, on the third to seventh postoperative day to rule out the return of conduction through the accessory AV connection. Radiofrequency ablation patients in whom the accessory AV connection had been concealed underwent a follow-up electrophysiological test on the second day after ablation to rule out the return of conduction through the accessory AV connection.

Late follow-up in all of these patients was undertaken by a combination of repeat electrophysiological testing, patient and physician contact, and office file and chart review. Successful outcome was determined by electrophysiological testing or the absence of symptomatic tachycardia during follow-up. Patient data are expressed as mean ± one standard deviation. Comparisons were made using Student's t test. A p value less than 0.05 was considered significant.

Results

Operation

Eighty-two patients were found to have a single accessory AV connection, 40 patients had two accessory AV connections, and 1 patient had three accessory AV connections. One hundred forty-two accessory AV connections were manifest and 24 were concealed. Table 1 shows the locations of the accessory AV connections. Eighty-seven were located in the free wall of the left ventricle, 43 were posteroseptal, 21 were located in the free wall of the right ventricle, 5 were intermediate septal, and 10 were anteroseptal, for a total of 166 accessory pathways in 124 patients.

Other procedures performed at the time of WPW syndrome correction included mitral and tricuspid valve repair or replacement (6), right ventricular conduit replacement, subaortic resection, Fontan repair, corrected transposition repair, single coronary artery bypass, and placement of an automatic internal cardioverter defibrillator (1 each). Mean aortic cross-clamp time was 49 ± 24 minutes and mean total cardiopulmonary bypass time, including electrophysiologic mapping, was 124 ± 28 minutes. One hundred nineteen patients (97%) had a successful outcome of surgical ablation during a mean follow-up of 26 ± 7 months. One hundred fourteen patients (93%) underwent a single operative procedure with a successful outcome. Five patients required a second operation for successful ablation of their pathway, ranging from hours after the initial procedure to 1 patient whose preexcitation reoccurred 24 months after his first operation.

Four patients had an unsuccessful outcome and continued to demonstrate preexcitation after operation. This included 1 patient who underwent four operative procedures on a left free wall pathway over a 4-year period without a successful outcome. Of these surgical failures, 1 patient underwent successful radiofrequency catheter ablation after operation. The remaining 3 patients who had an unsuccessful outcome have been treated with antiarrhythmic medications.

Complications after operation included postoperative bleeding necessitating a return to the operating room in 4 patients (3%). Additional complications occurred in 1 patient who had a prior attempt at surgical ablation of left free wall and posteroseptal pathways at another institution and had repeat surgical ablation procedures performed at our institution. This redo procedure resulted in ablation of his preexcitation but also in intermittent third-degree heart block requiring placement of a permanent pacemaker. Furthermore, 1 patient, an 11-year-old girl with Ebstein's anomaly and a large atrial septal defect, was noted to have had a cerebral vascular accident after the surgical procedure; however, she recovered completely from this insult. Another complication occurred in a 34-year-old woman who was shown to have mitral prolapse before operation. She was noted to have trivial to 1+ mitral regurgitation on follow-up two-dimensional echocardiogram. Another patient was thought to have electrocardiographic changes consistent with a small lateral wall myocardial infarction 4 months after operation.

Cardiac catheterization in this patient revealed an occlusion of a small posterolateral branch of the circumflex coronary artery, but it is unknown whether this was related to the original surgical procedure. A 32-year-old woman who had two failed episodes of direct-current catheter ablation underwent surgical ablation of her left lateral free wall and posterior septal aberrant atrial ventricular connections. In the postoperative period, her preexcitation disappeared from her surface electrocardiogram. However, 3 months after this procedure she again was symptomatic from her supraventricular tachycardias, with return of her delta waves. After this she underwent radiofrequency ablation in these areas, which failed. She continues to be followed up on medical therapy.

Interestingly, 1 patient, whose WPW syndrome was found in the preoperative evaluation of a large abdominal aortic aneurysm, had had multiple episodes of supraventricular tachycardias through his concealed pathway for more than 30 years. This patient had received an antitachycardia pacemaker 4 years before the diagnosis of his WPW syndrome. This pacemaker did not improve his clinical symptoms and was removed at the time of WPW syndrome operation. He has now undergone successful repair of his abdominal aortic aneurysm.

There was one death, in a 9-year-old patient who was believed to have concealed anteroseptal and intermediate septal pathways. This patient also had corrected transposition and multiple other congenital anomalies. After operation for his cardiac anomalies, as well as correction of his WPW syndrome, he most probably suffered a hypoxic brain injury during a cardiac arrest. Although the patient recovered somewhat from this neurological event, he died slightly more than 2 months after operation of a combination of his impaired neurological status and his other congenital anomalies. He had continued to have intermittent preexcitation in the postoperative period. At autopsy, his heart was inspected but no specific area of AV accessory pathways could be identified.

Radiofrequency Ablation

One hundred fifteen patients had a single accessory AV connection and 9 patients had two accessory AV connections. Forty accessory AV connections were concealed. Table 1 shows the locations of the accessory AV connections. Eighty-five were located in the free wall of the left ventricle, 22 were posteroseptal, 17 were located in the free wall of the right ventricle, 6 were intermediate septal, and 3 were anteroseptal for a total of 133 accessory AV pathways in these 124 patients. One hundred twelve patients (90%) had a successful outcome after radiofrequency ablation during a mean follow-up of 7 ± 5 months. One hundred patients (81%) underwent a single ablation procedure with a successful outcome. Successful outcome after radiofrequency ablation was achieved in 11 patients who required a second ablation session and in 1 patient who required a third ablation, either because the first attempt was unsuccessful or because of a recurrence of conduction through the accessory AV connection. The mean number of applications of radiofrequency energy during successful ablation sessions was 7 ± 5.

Twelve patients have had an unsuccessful outcome. Nine of these patients undergoing unsuccessful ablations had pathways located in the left free wall, 1 had a posteroseptal pathway, and 2 had intermediate septal pathways. The mean number of applications of radiofrequency energy during unsuccessful ablation sessions was 16 ± 12. One patient underwent four unsuccessful ablation procedures of a left free wall pathway, but the pathway was successfully ablated at operation. Five of these patients have undergone surgical ablation and were found to have areas of ecchymosis and edema on both the atrial and ventricular side of the mitral annulus, near the site of the accessory AV connection (Fig 1). One patient underwent successful catheter ablation at another institution. The remaining patients who had unsuccessful outcome after attempted radiofrequency ablation have been treated with antiarrhythmic medications.

Complications occurred in 4 patients. Chest pain and lateral ST segment elevation developed in 1 patient with a concealed left lateral accessory AV connection during an application of radiofrequency energy [8]. Coronary angiography demonstrated occlusion of the circumflex coronary artery and indicated that the ablation catheter had been inadvertently positioned within the circumflex itself. Patency of the vessel was restored by angioplasty. Left ventricular radionuclide studies 1 week later demonstrated an ejection fraction of 0.55. This patient has had no recurrences of paroxysmal supraventricular tachycardia during 8 months of follow-up. A second patient was noted to have a new diastolic murmur after successful ablation of a left lateral accessory AV connection. Doppler echocardiogram demonstrated mild aortic insufficiency and a small perforation of a deformed aortic valve leaflet, which has persisted with follow-up. A retroperitoneal hematoma and an 8% decrease in hematocrit developed in a third patient. A transient neurological deficit occurred in 1 patient 5 days after she underwent successful ablation of a concealed left lateral accessory AV connection. Transient right-sided weakness and aphasia developed and lasted 5 minutes. A computed tomographic scan of the brain was normal. No patient demonstrated a pericardial effusion or a new wall motion abnormality on the echocardiogram performed immediately after the ablation procedure.

Comment

This comparison study demonstrates that, as opposed to long-term pharmacological therapy, both surgical ablation (97%) and radiofrequency ablation (90%) offer excellent success with low morbidity for the treatment of WPW syndrome.

Although long-term pharmacological therapy may eliminate or ameliorate tachycardia-related symptoms, it is associated with the risk of adverse effects and is also inconvenient and expensive. The cost of surgical treatment versus long-term pharmacological treatment of WPW syndrome has been compared recently [12]. In that report, 77 patients with WPW syndrome treated with antiarrhythmic drugs were compared with 50 patients

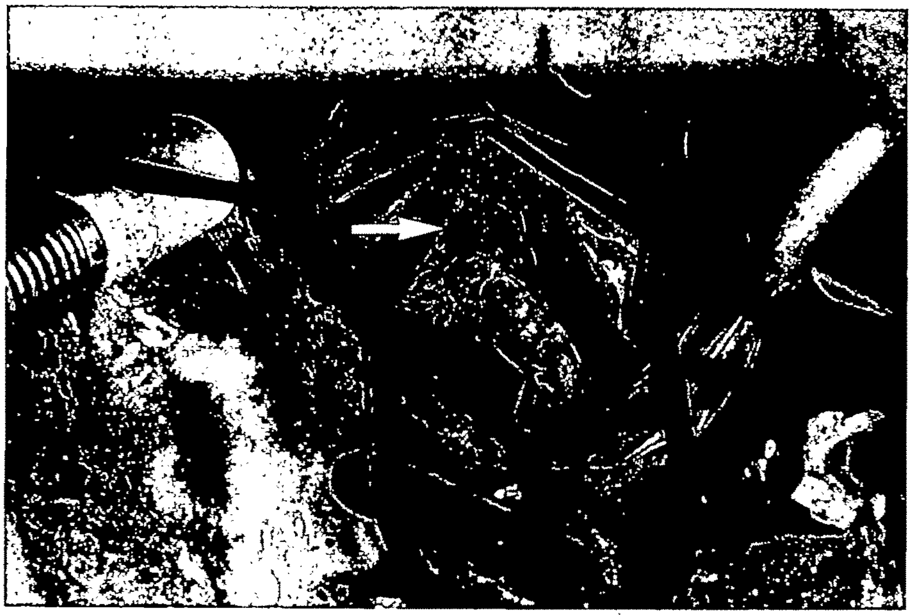

Fig 1. Shown is the atrial side of the mitral annulus in a patient who underwent four separate unsuccessful sessions of radiofrequency energy application. The arrow *illustrates the central hemorrhage and necrosis, with surrounding edema typically seen with radiofrequency lesions. Intraoperative mapping demonstrated that this was the area of accessory conduction. This patient was successfully treated with operation.*

with WPW syndrome treated surgically. Costs including drugs, operative procedures, pacemakers, electrophysiological testing, clinic visits, and readmissions because of tachycardia were considered. The total cost at the end of 4.5 years of follow-up for the pharmacological-medical group was $4,200 as opposed to $10,800 in the surgically treated group. However, when a projection was made based on these figures, it was demonstrated that the cost of pharmacological-medical treatment became higher than the cost of surgical treatment after 12 years of treatment. As the mean age of presentation in the current series is young (26 and 36 years for surgical and radiofrequency patients, respectively), long-term pharmacological therapy is often not a desirable therapeutic option for these patients. Additionally, it was concluded that medically treated patients remained symptomatic and at risk for syncope and sudden death [13]. This was believed to warrant primary surgical treatment for all patients. With a success rate for surgical ablation of accessory AV connections as high as 97% to 100% [14, 15], surgical correction for WPW syndrome has become the gold standard with which any other therapy must be compared.

The excellent success rate and low morbidity for the surgical treatment of WPW syndrome have been consistent regardless of the institution, the age of the patients, or the surgical technique used. In a recent report, the results of surgical treatment of WPW syndrome in adult patients, divided equally into the classic endocardial operative approach versus the epicardial technique, supplemented with cryoablation was reviewed [16]. Each group had aberrant pathways successfully ablated in 22 of the 25 patients (88%), and fewer than 20% of the patients had even minor postoperative complications.

The surgical management of WPW syndrome has also been applied to infants and children. In another recent study, results after operation for WPW syndrome were reported in a series of children aged 5 years or younger [17]. With a mean follow-up of 12 months, 18 of 19 children remained cured of their supraventricular tachycardias. Additionally, in a report from this institution [18], the surgical management of 30 children and young adults with WPW syndrome was reviewed. In that group, WPW syndrome operation was curative in 27 of 30 patients (90%), and symptoms were eliminated in the other 3, despite intermittent electrocardiographic demonstration of delta waves. These experiences suggest that the surgical treatment of WPW syndrome is safe, effective, and frequently the preferred treatment for this disorder in even young patients.

That the procedure of the surgical treatment of WPW syndrome has become widespread is evidenced from a 1989 article from Beijing, China [19], which reported 100 patients with WPW syndrome. In this series, 90% of the right-sided pathways were cured, 82% of the left-sided pathways were cured, and 65% of the patients who had multiple pathways were cured.

Recently, however, radiofrequency catheter ablation of accessory AV connections, using the local application of radiofrequency microwave energy to cauterize or "boil" tissue, has been described in an increasing number of reports [5–8], including one with a success rate of 100% in 44 patients who underwent radiofrequency catheter ablation of left-sided accessory AV connections [6]. The success rate of 90% for radiofrequency catheter ablation at our institution compares favorably with these results and also with previous reports of catheter ablation using direct-current shocks. Several investigators have reported success rates of only 50% to 70% for direct-current shock ablation of posteroseptal accessory AV connections [20, 21], whereas others have noted successful ablation of 32 of 35 accessory AV connections in all locations with direct-current shocks [22]. At our institution, direct-current catheter ablation met with only partial relief of symptoms in 32 of 48 patients [21].

The width of accessory pathways as they cross from the atrium to the ventricle has important implications for the treatment of WPW syndrome with radiofrequency catheter ablation and may affect the long-term follow-up for radiofrequency ablation patients. In a recent report [23] the atrial and ventricular activation times along the AV groove were measured in 23 patients with WPW syndrome, and the width of the earliest atrial and ventricular activations were assessed. When the AV ring was divided into the standard 15 predefined anatomical landmarks, the extension of the accessory pathway was noted to be 1.9 and 1.6 anatomical landmarks on both the atrial and ventricular insertions, respectively. An overlap of the atrial and ventricular insertions was noted, and these observations suggested that wide surgical dissection along the AV groove was more likely to divide the aberrant conduction pathways, even if the epicardial mapping disclosed only a discrete area of preexcitation. In another anatomical study, accessory AV connections were demonstrated to slant over a distance of 1 to 2 cm from their atrial to ventricular insertions [7]. These findings have also been confirmed in a recent report of 73 patients with WPW syndrome, comparing preoperative mapping and intraoperative mapping [24]. In this study, aberrant conduction pathways often were seen as multiple closely spaced or arborized accessory pathways, and intraoperative mapping led to widening of the margins of the surgical dissection. These findings are consistent with the findings from the present study, which demonstrated a significant increase in the number of surgical patients with multiple pathways. Although multiple site ablations are possible with radiofrequency catheter ablation, operation may be more effective.

Many of the surgical patients were believed to have increased conduction times in the posterior septal area during intraoperative mapping, accounting for the increased number of pathways in this area. Although this may represent the vagaries of inexact intraoperative mapping, it may also be related to the overlap and wide areas of increased conduction. All of these areas were believed to be important and were vigorously and widely dissected. These anatomical observations have been confirmed by others [11] and are at odds with the findings that a relatively small (<1 mm) radiofrequency lesion can permanently destroy the aberrant AV connection.

There are anatomical constraints that may preclude radiofrequency therapy for some patients. Although accessory AV connection activation can be recorded throughout a wide area, the precise point at which accessory AV connections are susceptible to radiofrequency energy ablation lesions may vary. Furthermore, it is possible that some accessory AV connections are located quite epicardially in some patients and that the radiofrequency lesion, given from the endocardium, is not "deep" enough for successful ablation. In our experience, the lesions produced by radiofrequency ablation, when identified at the time of operation after a failed radiofrequency catheter ablation, have been very small, often less than 2 mm (see Fig 1). Long-term follow-up will be needed to ensure that the injury at the ablation sites of radiofrequency energy was adequate and that these pathways do not recover and recur, particularly in light of the surgical patient who had recurrence 24 months after "successful" ablation.

Other factors must be considered when comparing and contrasting the two modes of ablation therapy. The first factor is cost. In a study of 22 patients with WPW syndrome from our institution [25], the cost of radiofrequency catheter ablation was compared with that of surgical ablation. Radiofrequency catheter ablation was successful in 8 of 11 patients (73%), whereas, in the surgical group, the accessory pathway was successfully ablated in all patients. The mean duration of hospitalization was 6 ± 2 days in the catheter group versus 8 ± 4 days in the surgical group. However, the mean cost per patient expressed in 1988 dollar values was approximately $14,000 ± 4,500 in the radiofrequency catheter group, as compared with $34,000 ± 5,400 in the surgical ablation group ($p < 0.001$). The mean time lost from work or school was 10 days in the radiofrequency catheter ablation group as compared with 60 days in the surgical group. Even assuming eventual switch over to surgical ablation in the failed radiofrequency catheter ablation patients, the mean cost of therapy in the radiofrequency catheter ablation group was $24,000 ± 4,700, still significantly less than surgical cost.

The risks and benefits of each procedure are an additional important consideration when comparing the two modes of ablation therapy. The risks of surgical ablation for WPW syndrome, as for any cardiac surgical procedure, are well known. These include postoperative hemorrhage, pulmonary compromise, and wound healing problems. However, the rate of surgical complications in this series was quite low, and radiofrequency ablation also involves risk. Perforation of cardiac structures, bleeding, pulse deficits, myocardial infarction, and transient neurological deficits are well known to occur with any catheter-based procedure. It is likely that acute coronary artery thrombosis, as reported after radiofrequency ablation [8], is an uncommon complication of catheter ablation and can be prevented by careful catheter positioning. However, the long-term effects of radiofrequency energy delivery on coronary arteries close to ablation sites is unknown. The mean duration of exposure to fluoroscopy during the radiofrequency ablation procedures was 48 ± 30 minutes. This duration of fluoroscopy exposes patients to approximately 0.52 Gy (52 rads) of radiation, and is similar to that of coronary angioplasty [26]. This amount of radiation has been estimated to result in approximately 56 to 90 fatal malignancies per one million patients [27]. Years of follow-up will be needed to rule out adverse effects of radiofrequency ablation.

Although the benefit of avoiding a sternotomy when radiofrequency ablation is the sole procedure for WPW syndrome is obvious, one benefit of surgical ablation is that other cardiac reparative procedures may safely be performed simultaneously when they are required. In our series, procedures performed concomitantly included mi-

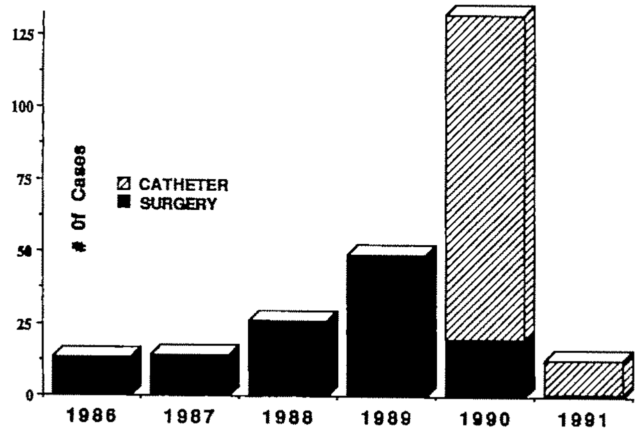

Fig 2. Shown is the rate of both surgical and radiofrequency catheter ablation procedures for the treatment of Wolff-Parkinson-White syndrome at the University of Michigan Medical Center from July 1986 to January 1991.

tral and tricuspid valve repair and replacement, right ventricular conduit replacement, subaortic resection, Fontan repair, corrected transposition repair, coronary artery bypass, and placement of an automatic internal cardioverter defibrillator. Finally, because radiofrequency ablation sessions have been reported to last up to 16 hours [6], the treatment of multiple pathways may be more efficacious with a surgical approach.

The results of the present study demonstrate that surgical and radiofrequency ablation procedures offer an excellent success rate with low morbidity in the treatment of WPW syndrome. Although continued late follow-up is mandatory to assess the results of radiofrequency ablation, the early results and the trends in the number of procedures performed (Fig 2) suggest that operation for WPW syndrome may become indicated for failure of radiofrequency ablation, multiple pathways, or when additional procedures are required.

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DISCUSSION

DR JAMES E. LOWE (Durham, NC): I would like to commend Bolling and colleagues for presenting to us a very honest and straightforward comparison of operation versus radiofrequency catheter ablation for the treatment of the WPW syndrome. Just as percutaneous transluminal coronary angioplasty has changed our coronary practice, radiofrequency catheter ablation will definitely affect operation for the WPW syndrome. It appears obvious that patients referred for operation in the future will be more complex and have more associated anomalies. Also, for the first time in the field of WPW syndrome operation, we will be asked to intervene emergently or at least urgently for the multiple complications resulting from catheter therapy.

In the present study, 93% of patients underwent successful surgical ablation on the first attempt compared with 81% of those subjected to radiofrequency catheter therapy. It is important to point out that the surgical group had significantly more patients with multiple pathways and other cardiac pathology. It is also apparent from this study that radiofrequency catheter therapy is associated with substantial morbidity, including circumflex coronary occlusion, aortic valve injury resulting in aortic insufficiency, a transient neurological event, and an eight-unit retroperitoneal bleed.

I have had the opportunity at Duke Hospital to operate on nearly 300 patients with the WPW syndrome. The success rate after the initial operation has been 97%, and complications have been minor, including one reexploration for bleeding and one wound infection. There have been no coronary artery, atrioventricular valve, or neurological injuries.

There are several critical unknowns regarding radiofrequency catheter therapy. First, what are the long-term effects of prolonged radiation exposure, especially in young patients? I think Bolling and colleagues should be congratulated because they limited fluoroscopy time in this study. At other centers, patients have been subjected to 16 hours of general anesthesia in the electrophysiology suite to accomplish radiofrequency catheter ablation. Second, what are the long-term effects of radiofrequency ablation on coronary arteries? And finally, what is the natural history of relatively minor catheter-induced valvular injury? I think to answer these three important questions careful, sequential, long-term follow-up will be required.

Dr Bolling, what are the details of a planned follow-up protocol of patients subjected to radiofrequency catheter ablation at the University of Michigan? Second, in your opinion as a surgeon, what are the current contraindications for radiofrequency therapy? Finally, is general anesthesia required for radiofrequency procedures at your center?

DR TAKASHI IWA (Kanazawa, Japan): I congratulate Bolling and associates on their success in devising a new strategy for the treatment of WPW syndrome. While cardiac surgeons are arguing about the respective merits of endocardial versus epicardial approaches in the surgical treatment of WPW syndrome, it seems that the times are changing to catheter ablation versus operation. Some groups, including Dr Bolling's, have reported extremely good results with catheter ablation using direct-current shock or radiofrequency waves.

Dr Evans in San Francisco, however, reported at the Annual Meeting of the American Heart Association last November that in an international multicenter study, 3 of 65 patients died after catheter ablation and the success rate of the procedure was only 57%. This report told us that catheter ablation is still in the process of developing and that it is difficult to perform in average cardiac centers.

I have two questions, Dr Bolling. First, do you have definite, respective indications for operation and catheter ablation for WPW syndrome? I believe that if cardiologists decide on nonpharmacological treatment instead of drug treatment for certain patients with WPW syndrome and catheter ablation fails to cure the WPW syndrome, then all the patients in whom catheter ablation fail should be operated on to be cured. You mentioned that there have been 12 failures, of whom 5 had operation and 7 are being treated medically. Why are they being treated medically? Were they indicated for nonpharmacological therapy once, not medical therapy? I would like to ask, therefore, what are the real indications for nonpharmacological treatment for WPW syndrome in your group?

Second, I know that radiofrequency is less traumatic than direct-current shock. But Dr Jackson of Oklahoma, one of the pioneers of this field, needed 10 to 14 hours for one session of radiofrequency catheter ablation. For how many hours are your patients exposed to x-rays during the catheter ablation?

DR BOLLING: I thank both of the discussants. The patients who are subjected to radiofrequency ablation at our institution are part of a long-term follow-up, and obviously 7 months of follow-up is not adequate. One of the questions in terms of follow-up is the width of these pathways, which is believed to be perhaps 2 to 3 cm and slanting laterally between the atrial and ventricular connections. Obviously the lesion that is placed at the time of radiofrequency catheter ablation is very small, and perhaps these pathways will reoccur.

In terms of contraindications to radiofrequency ablation, there are very few. Our patients do not undergo these procedures under general anesthesia. The radiofrequency ablations are limited to 4 hours. Dr Morady and the cardiologists believe that this is a good limit and will not subject the patients to any more. At a limit of 4 hours per catheter ablation session, the radiation exposure is equivalent to angioplasty and equals approximately 56 malignancies per 1 million patients. It is fairly low and acceptable in most of the cardiology literature.

Regarding medical treatment for some of the 7 patients with failed radiofrequency ablations and why we saw a sudden surge in our cases of WPW syndrome, I think that there is a vast population of these patients with WPW syndrome who have not come to operation yet and have been attracted to institutions that have started radiofrequency ablation programs. Because of that, these patients do not want operation, and despite the fact that they have had failed radiofrequency ablation, they stay on medications afterwards.

DNA Ploidy Patterns of Tumors Diagnosed as Metachronous or Recurrent Lung Cancers

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The relationship between the first tumor and the second tumor resected in 8 patients with non–small cell lung cancer was analyzed using deoxyribonucleic acid (DNA) flow cytometry. Of the 8 patients, 6 were clinically diagnosed as having metachronous lung cancers and 2, local recurrent tumors. The mean interval between operations in patients with metachronous lung cancers was 62 months (range, 15 to 128 months). Both tumors showed the same histology in 4 patients and a different histology in 2. In the 2 patients with local recurrent tumors, the interval between operations was 9 months and 39 months. In the analysis of DNA flow cytometry of the first and second tumors in the same patient, the tumors were defined as independent of each other when one tumor showed diploidy and the other, aneuploidy, or when each DNA index of abnormal clones between two aneuploid tumors was different. When both tumors showed diploidy or when at least one DNA index of abnormal clones between two aneuploid tumors was identical, the tumors were defined to be related to each other. According to these criteria, in 5 (83%) of the 6 patients clinically diagnosed as having metachronous lung cancers, the second tumor was classified as independent of the first tumor. On the other hand, in the 2 patients clinically diagnosed as having recurrent tumors, the second tumor was judged to be related to the first tumor. These data suggest that DNA flow cytometric analysis of tumors may be of value in the diagnosis of metachronous lung cancers.

(Ann Thorac Surg 1991;52:469–73)

Long-term survivors of curative resection of lung cancer are known to have a high risk of developing metachronous lung cancers. The incidence of metachronous lung cancers in these patients has been reported to be 10% to 25% [1–3]. However, the overall incidence of metachronous lung cancers varies from report to report [1–6]. This can be ascribed to the lack of standard criteria for defining such cancers. It is, in fact, difficult to differentiate between metachronous lung cancers and recurrent or slow-growing metastatic tumors, especially when the first tumor and the second tumor show the same histology. The percentage of first and second (metachronous) tumors with identical histological types is reported to be about 70% to 80% [2–4]. This fact indicates that new diagnostic tools, in addition to clinical and pathological examinations, should be developed to diagnose metachronous lung cancers. In the present study, we used deoxyribonucleic acid (DNA) flow cytometry to determine the characteristics of first tumors and second tumors that were strongly suspected to be metachronous or recurrent lung cancers.

Material and Methods

Eight patients and 3 patients with non–small cell lung cancer who were suspected to have metachronous lung cancers and local recurrent tumors, respectively, after surgical treatment of primary lung cancer were operated on at National Kyushu Cancer Center from 1977 to 1989. The present study concerns 8 patients in whom we were able to analyze the DNA ploidy pattern in the first and second tumors by DNA flow cytometry. Pathological staging was done according to the new international staging system proposed by Mountain [7]. Preoperative examinations at the time of the second operation in all patients included bronchoscopy, bone scintigraphy, and computed tomography of the thorax and abdomen. There was no evidence of occult distant metastases in any of the patients.

In 6 patients, the first tumor and the second tumor, which was strongly suspected to be a metachronous lung cancer, were analyzed by DNA flow cytometry. Patient data are shown in Table 1. Patient 3 has had no evidence of recurrence on radiological examinations but again demonstrated an increase in serum carcinoembryonic antigen level after the second operation. He is thus classified as alive with recurrence. The first tumor in patients 2 and 3 was removed by lobectomy combined with resection of the chest wall.

In 2 patients, the primary and local recurrent tumors

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Table 1. Patients With Metachronous Lung Cancers

Patient No.	Sex	Age (y)	Histology and Location	TN Stage	Operation	Interval Between Operations (mo)	Survival After Second Operation (mo)
1	M	65	Squamous cell carcinoma (W); RLL	T2 N0	Lobectomy	15	NED, 60
			Squamous cell carcinoma (M); RUL	T1 Nx	Wedge resection		
2	M	53	Squamous cell carcinoma (P); RUL	T3 N0	Lobectomy	32	NED, 18
			Squamous cell carcinoma (P); LUL	T1 Nx	Wedge resection		
3	M	63	Large cell carcinoma; LUL	T3 N0	Lobectomy	64	Alive with recurrence, 20
			Adenocarcinoma (M); RUL	T1 Nx	Wedge resection		
4	M	59	Adenocarcinoma (M); LUL	T2 N0	Segmentectomy	65	Alive with recurrence, 17
			Adenocarcinoma (M); RUL	T1 Nx	Wedge resection		
5	M	65	Squamous cell carcinoma (P); RUL	T3 N0	Lobectomy + wedge resection	70	Alive with recurrence, 6
			Adenocarcinoma (M); LUL	T2 N1	Lobectomy		
6	M	62	Adenocarcinoma (M); RML	T1 N0	Lobectomy	128	Alive with recurrence, 18
			Adenocarcinoma (M); LUL	T4 Nx	Wedge resection		

LUL = left upper lobe; M = moderately differentiated; NED = no evidence of disease; P = poorly differentiated; RLL = right lower lobe; RML = right middle lobe; RUL = right upper lobe; W = well differentiated.

were also analyzed by DNA flow cytometry (Table 2). In each patient, the primary tumor invaded the adjacent lobe. Although the tumor seemed to have been completely removed by lobectomy and wedge resection at the time of the first operation, a recurrent tumor developed near the margin of the previous wedge resection.

Flow cytometric analysis of cellular DNA content was performed using paraffin-embedded blocks of surgically resected specimens. Well-preserved areas of the tumor were chosen on slides, and the corresponding areas on paraffin blocks were identified. A single cell suspension was made, and the cells were stained with propidium iodide according to the method described by Tosi [8], Vindeløv [9], and their associates. The stained cells were analyzed on an FACS-CAN (Becton Dickinson, Mountain View, CA). At least 20,000 cells were examined in each specimen, and the DNA content per cell, measured by fluorescence intensity, was recorded and displayed in a histogram. The resulting DNA histograms were interpreted without knowledge of patient data.

The DNA index was calculated as the ratio of the aneuploid G_0G_1 peak to the diploid G_0G_1 peak. When only one G_0G_1 peak (DNA index = 1) was observed, the tumor was considered to be diploid. Tumors that had evidence of at least one distinct G_0G_1 population (DNA index > 1.0) in addition to a diploid stemline were classified as aneuploid. The lowest peak was assumed to represent the diploid G_0G_1 peak (DNA index = 1). Samples whose coefficient of variation of the diploid G_0G_1 peak exceeded 10% were not used to determine DNA ploidy of the tumor.

The tumors were defined to be independent of each other when they showed completely different patterns of DNA ploidy, that is, (1) one tumor showed diploidy and the other, aneuploidy, or (2) when two tumors showed aneuploidy, each DNA index of abnormal clones between the two tumors was different. The tumors were defined to be related to each other when they showed diploidy or when at least one DNA index of abnormal clones between two aneuploid tumors was the same or almost identical. The criterion that defined an identical abnormal clone between two aneuploid tumors was the following: mean

Table 2. Patients With Local Recurrent Tumor

Patient No.	Sex	Age (y)	Histology and Location	TN Stage	Operation	Interval Between Operations (mo)	Survival After Second Operation (mo)
1	F	63	Squamous cell carcinoma (W); RUL	T2 N0	Lobectomy + wedge resection	9	NED, 83
			Squamous cell carcinoma (P); RLL	T2 N0	Completion pneumonectomy		
2	F	41	Adenocarcinoma (W); LUL	T2 N0	Lobectomy + wedge resection	39	NED, 30
			Adenocarcinoma (M); LLL	T1 N2	Complete pneumonectomy		

LLL = left lower lobe; other abbreviations are the same as in Table 1.

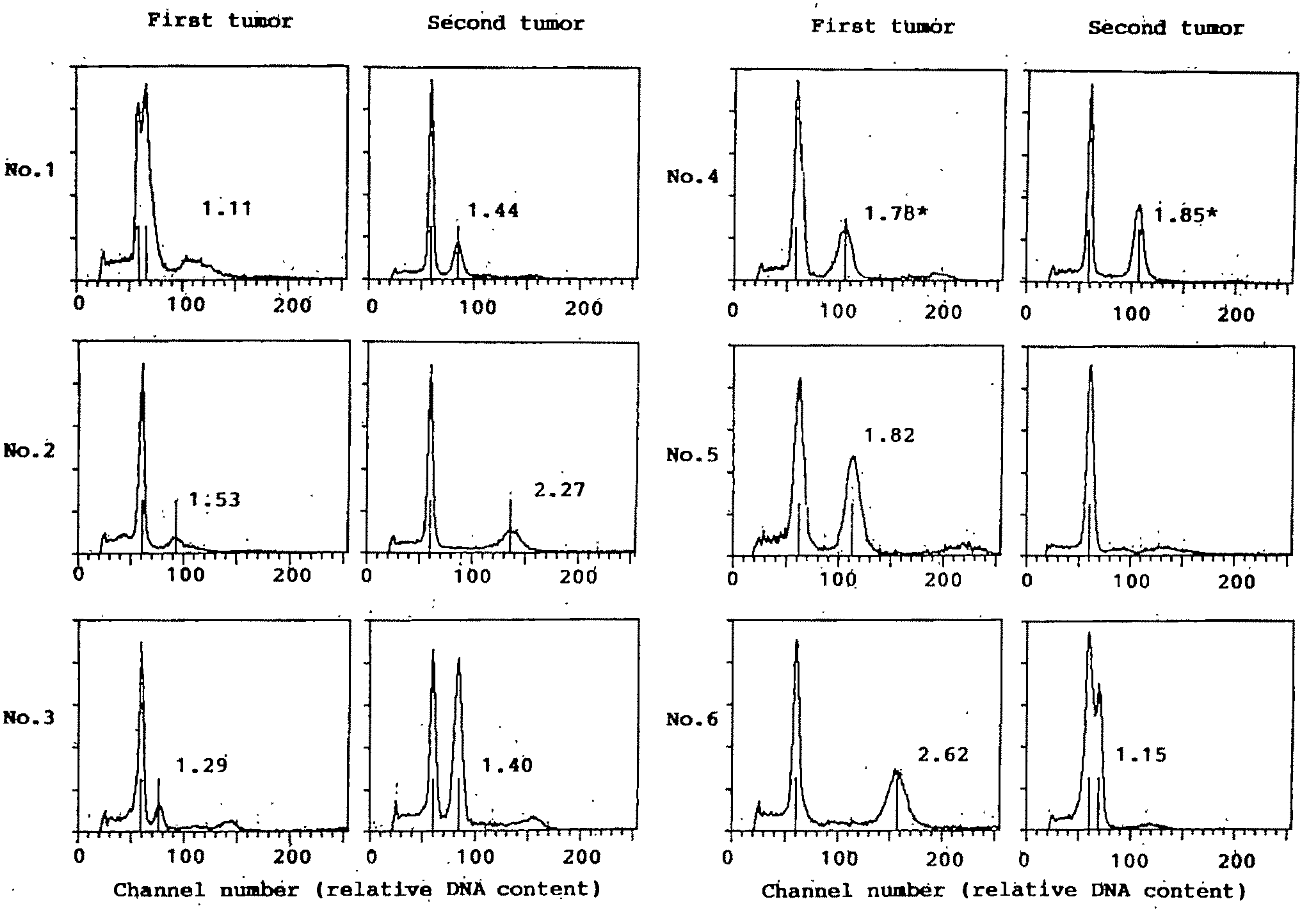

Fig 1. Deoxyribonucleic acid (DNA) ploidy patterns of the first and the second (metachronous) tumor. The DNA index of aneuploid peak of each tumor is shown. The second tumor in patient 5 shows diploidy. (= identical abnormal DNA clone between the first and second tumors.)*

DNA index of the subject clones × 0.975 < DNA indices of each subject clone < mean DNA index of the subject clones × 1.025.

Results

DNA Ploidy Patterns of Metachronous Lung Cancers

Figure 1 shows DNA histograms of the first and second tumors in the 6 patients. All tumors except for the second tumor of patient 5 showed aneuploidy. By our criteria, in 5 (83%) of the 6 patients, the second tumor was judged to be independent of the first tumor. In the tumors of patients 1, 2, 3, and 6, each DNA index of abnormal clones between the two aneuploid tumors was different. The first and second tumors in patient 5 showed aneuploidy and diploidy, respectively. As the DNA indexes of abnormal clones between the two aneuploid tumors in patient 4 were almost identical, the two tumors were defined to be related to each other.

DNA Ploidy Patterns of Recurrent Lung Cancers

To compare the relationship of DNA ploidy patterns between the primary and recurrent tumors with that between the first and second (metachronous) lung tu-

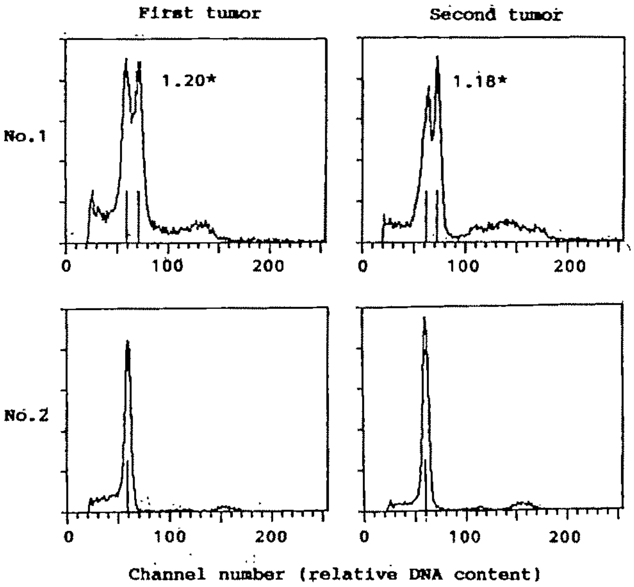

Fig 2. Deoxyribonucleic acid (DNA) ploidy patterns of the first and the second (recurrent) tumor. The DNA index of aneuploid peak of each tumor for patient 1 is shown. The tumors in patient 2 demonstrate diploidy. (= identical abnormal DNA clone between the first and second tumors.)*

mors, both tumors of the 2 patients with recurrent lung cancers were analyzed by DNA flow cytometry (Fig 2). In patient 1, the DNA indexes of abnormal clones between the aneuploid tumors were almost the same. In the other patient, both tumors showed diploidy. Therefore, in terms of DNA ploidy analysis, the second tumor in the patients clinically diagnosed as having recurrent tumor was judged to be related to the first tumor.

Comment

The patients in the present study were those in whom the second tumor was strongly suspected to be a metachronous or recurrent lung cancer. All patients underwent bone scintigraphy and computed tomography of the thorax and abdomen to rule out the existence of occult distant metastases at the time of the second operation. When the first and second tumors show identical histological types, the disease-free interval between the tumors, one of the criteria for defining metachronous lung cancers, is 2 or 3 years in most reports [3, 4]. In our study, the interval between operations in patients 1 and 2 was 15 months and 32 months, respectively (see Table 1). However, these patients are alive without disease 60 months and 18 months, respectively, after the second operation. Therefore, the second tumor in these patients can be considered a metachronous lung cancer. The interval between operations in the other 4 patients was more than 60 months.

We used DNA flow cytometry to determine the relationship between the first tumor and the second tumor in each patient. The criteria used are based on the following two hypotheses: (1) In the case of both being diploid tumors, there are no abnormal clones in terms of DNA ploidy, and thus their biological characteristics can be thought to be similar. (2) When at least one DNA index of abnormal clones between two aneuploid tumors is the same or almost identical, both tumors can be thought to be related, in part, to each other. When the two tumors did not meet these conditions, they were considered to be independent of each other. However, this independence is not absolute because normal (diploid) G_0G_1 peaks, which were made by tumor cells and normal epithelial or stromal cells or both within tumors and which were observed in all specimens, were ignored.

In this study, the second tumor in 5 (83%) of the 6 patients clinically diagnosed as having metachronous lung cancers was classified as being independent of the first according to the criteria. On the other hand, recurrent tumors in 2 patients were related to the primary tumors in terms of DNA ploidy pattern.

Although we could not analyze the metastatic tumor in the lung after resection of the primary lung cancer because of the lack of suitable specimens, analysis of DNA flow cytometry for primary lung cancers and lymph node metastases has been reported. Volm and colleagues [10] reported the results of analysis of DNA flow cytometry of 18 primary lung cancers and their lymph node metastases. There was one identical abnormal index between the primary and metastatic tumors in 12 patients, two identical abnormal DNA indices between both tumors in 1 patient, and both tumors with diploidy in 1. When our criteria were applied in the analysis of these data, the metastatic lesions were related to the primary lesions in 14 (77.8%) of 18 patients. We found similar observations in representative DNA histograms of primary and metastatic tumors reported by Salvati and associates [11]. These findings may lend support to our criteria for defining the relationship between the first tumor and the second tumor.

In most cases (70% to 80%) of tumors reported as metachronous lung cancers, the first and second tumors showed identical histological types [2–4]. In 1987, the Lung Cancer Study Group [12] reported that the first recurrent site of T1 N0 non–small cell lung cancer is the lung in 41% of patients. Therefore, it is difficult to differentiate between recurrent and metachronous tumors in patients whose two tumors show the same histology. In general, when the histologies of the two tumors are different, it can be accepted that they are metachronous lung cancers, regardless of the stage of the second tumor [1–6].

However, sometimes a histological difference exists between one portion of a tumor and another. In the case of poorly differentiated lung cancer, it is especially difficult to distinguish between poorly differentiated adenocarcinoma, squamous cell carcinoma, and large cell carcinoma by conventional pathological studies. In fact, it has been reported [13] that the most poorly differentiated adenocarcinoma contained squamous components when analyzed by electron microscopy. Furthermore, it is known that even some small cell lung cancers have components of non–small cell lung cancer [14].

Therefore, we think that the diagnosis of metachronous lung cancers should be given careful consideration. We believe that our hypothesis and method may shed light on the issue, but it should be noted that the analysis of cancer cells collected from paraffin-embedded blocks cannot be expected to clarify the character of the whole tumor. To confirm our hypothesis, a study using samples from multiple sites in a fresh tumor is now underway.

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Acute Postinfarction Septal Rupture: Long-Term Results

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From 1973 to 1989, 66 patients received early surgical repair for acute postinfarction ventricular septal rupture. Mean age was 64 ± 7 years (range, 45 to 80 years). Ventricular septal rupture occurred soon after acute myocardial infarction (3.4 ± 4 days), and the first medical treatment occurred 6.7 ± 7 days after onset of acute myocardial infarction. Three patients had a previous myocardial infarction. The site of the rupture was anterior in 38 patients (57%) and posterior in 28 (43%). Forty-four patients (67%) were in shock at the time of admission. Intraaortic balloon pumping was used preoperatively in 28. Operation was performed at the time of maximal efficacy of medical treatment. The same technique was used in all cases. Associated procedures included coronary bypass grafting in 5 patients and valvar operation in 5. The patients have been carefully followed up for up to 16 years. Hospital mortality was 45% (30 patients) and was cardiac related or due to acute renal failure in 25 patients (83%). No correlation could be revealed between early death and age, sex, preoperative intraaortic balloon pumping, or year of operation. Location of the ventricular septal rupture (early mortality of 57% for posterior versus 37% for anterior ventricular septal rupture) and shock at the time of admission (52% versus 32%) showed a trend toward significance ($0.08 \le p < 0.10$). Response to initial active therapy has a strong predictive value (mortality of 70% in unresponsive patients versus 14% in responders; $p < 0.001$). The actuarial survival rate was 56% ± 6% at 1 year, 44% ± 6% at 5 years (n = 23), and 26% ± 10% at 10 years (n = 10). The cause of late death was cardiac related in 38% of patients. Early surgical repair of early septal rupture after acute myocardial infarction has a high perioperative mortality rate but provides a good quality of life and long-term survival. Improvement should be possible by optimized techniques of myocardial reperfusion and transplantation. The age of the patients and the presence of initial cardiogenic shock make transplantation an infrequent option.

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Early rupture of the ventricular septum (VSR) after acute myocardial infarction (AMI) has for many years been regarded as a disastrous complication. A clearer understanding of the mechanism of VSR has permitted improvements in surgical techniques [1–6]. The term *rupture* is now preferred instead of *defect*, and the anatomical characteristics of the damaged septum have been described, such as septal dilaceration and friable necrotic tissues. At the same time, a greater understanding of the pathophysiological consequences of acute shunt and myocardial dysfunction has led to progress in perioperative medical management [7]. These two factors account for the overall improvement in results after early surgical repair. A retrospective analysis of a series of 66 patients who underwent operation between 1973 and 1989 enables evaluation of long-term results. These patients underwent identical operations [5] and have been carefully followed up on an annual basis. The results allow some assessment of the role of the most recent techniques such as myocardial reperfusion, cardiac transplantation, and aggressive methods of temporary mechanical cardiac support.

Material and Methods

Clinical Experience

Between January 1973 and December 1989, 66 patients were operated on for an early rupture of the interventricular septum that complicated a recent AMI (which had occurred within the previous 3 weeks) (Fig 1). There were 51 men and 15 women. The mean age was 64 ± 7 years (range, 45 to 80 years). Only 2 patients were less than 50 years of age; 32 were in their sixties, and 19 were older than 70 years (Fig 2).

The condition at the time of admission was critical in every case. Forty-four patients (67%) were in cardiogenic shock, defined by an aortic systolic pressure less than 80 mm Hg with clinical evidence of peripheral hypoperfusion and renal insufficiency (urine output < 30 mL/h). Twenty-two patients were in severe left ventricular insufficiency with pulmonary edema. In most cases the acute episode was the first clinical manifestation of coronary disease. Only 3 patients had a history of AMI. The time period between the recent AMI and first sign of VSR ranged from 0 to 21 days, with an average of 3.4 ± 4 days. The AMI was anterior in 41 patients (62%) and posterior in 25 (38%).

In all patients, treatment after Swan-Ganz catheterization included inotropic support intravenously and vaso-

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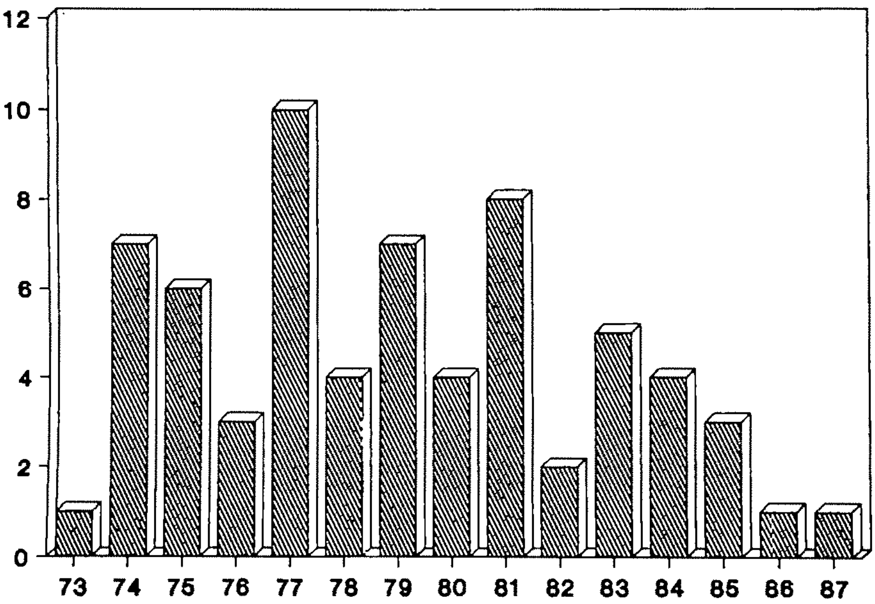

Fig 1. Annual operations for closure of ventricular septal rupture after acute myocardial infarction (none in 1988).

dilator therapy. Twenty-eight patients also had intraaortic balloon pumping in the early experience. In 1 patient, venoarterial extracorporeal circulation with renal dialysis was added to intraaortic balloon pumping in an attempt to reverse cardiogenic shock. Intraaortic balloon pumping was not used in the other patients, especially since 1980. Fluid balance and oxygen therapy were optimized in each patient. In 14 patients (21%) coronary and ventricular angiographic evaluation was performed before operation. This was done to assess the need for coronary bypass grafting and to evaluate the extent of the ventricular dysfunction and the exact location of the septal rupture. This evaluation was carried out in patients whose condition could be stabilized, and was discontinued in 1981.

During the period 1973 to 1989, the timing of operation was altered. Before 1976, operation had been postponed to the third week. Since 1976, operations have been performed earlier, within 3 days of admission in every patient. Overall, operation was performed an average of 7 ± 9 days (range, 0 to 21 days) after the diagnosis of VSR; 52 patients were operated on within 10 days and 33 (50%) within 3 days.

Operative Technique

All patients except 3 were operated on according to a previously reported original technique [5], in most cases by the same surgeon. Extracorporeal circulation was established between ascending aorta and caval cannulas. In all patients except the 8 operated on before 1975, the circuit was routinely equipped with a membrane oxygenator, either plaques or hollow fibers. Until 1978, myocardial protection was achieved by core cooling down to 18°

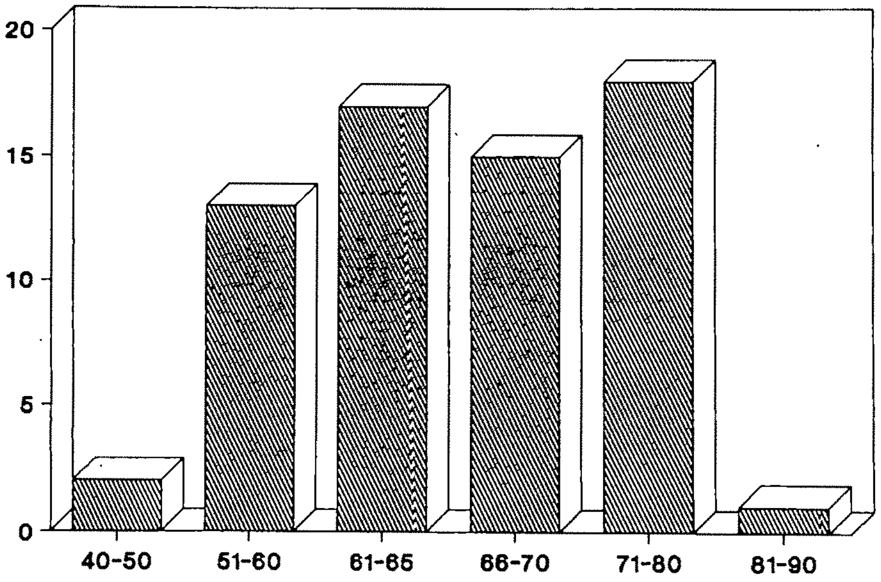

Fig 2. Age distribution (years) in 66 patients.

and pericardial crushed ice; since 1978 aortic perfusion of a cardioplegic solution has been added.

The surgical technique was as follows: (1) recognition of the anterior or posterior location of the VSR, (2) extensive infarctectomy to allow a wide exposure of the VSR through the infarcted area, (3) excision of the most friable border of the ruptured septum, (4) septal reinforcement with two Teflon felt strips, (5) closure of the VSR by insertion of a single or double Teflon patch on the reinforced septum, and (6) closure of the ventriculotomy by separate U stitches and Teflon reinforcement of the fragile walls. Treatment of associated lesions included coronary bypass grafting when indicated by the preoperative angiography and in cases where there was no technical difficulty; valve repair or tricuspid or mitral replacement was carried out if required.

Ventricular septal rupture was located anteriorly in 36 patients, and exposure was made through an anterior approach. In 33 patients, the left ventricle was involved, with the right ventricle being involved in only 3 patients. A posterior approach was used in 24 patients, depending on the location of the necrotic myocardial area. This was through the right ventricle in 15 patients and the left ventricle in 9 patients. A biventricular exposure was necessary in 6 patients because of the complexity of the septal defects. Coronary bypass grafting was indicated in 8 patients, but was actually performed in only 5 patients. Tricuspid valvoplasty and mitral valve replacement through a posterior transventricular exposure was performed in 5 patients, as previously described [5].

Statistics

Data are presented as mean ± standard error of the mean. Statistical comparisons among preoperative and postoperative factors were made by χ^2 test for discrete variables or by Student's t test for continuous variables. The significance level chosen was p less than 0.05. Actuarial survival of the 66 patients was calculated by the Kaplan-Meier method.

Results

Early Postoperative Period (<1 month)

Thirty patients (45%) died within 1 month. The cause of death was most often cardiac related (21 patients, 70%), and was due to extension of the recent AMI (in 3 patients), untreatable left ventricular arrhythmia (5), and left ventricular failure (13). In these last 13 patients, cardiac insufficiency and progressive multiple organ dysfunction necessitated a prolonged period of intensive care with tracheal intubation and subsequent renal dialysis. Persistent anuria was the cause of death in 4 patients. In 5 patients, although cardiac and circulatory function was adequate, death occurred for unrelated reasons (septic complications with mediastinitis in 3 patients, gastric ulcer bleeding in 2).

Five patients had to be reoperated on within the first postoperative month. In 3 patients, this was because of an early rupture of the septal repair. It should be emphasized that in these 3 patients, the Teflon felt strips were not used for septal reinforcement. In the other 2 patients, a second rupture of the septum occurred in a different part of the septum. In 3 of these 5 patients, death due to multiple organ failure occurred within 1 month of the original operation.

Long-Term Survival

The condition of patients was evaluated each year by either the referring cardiologist or ourselves. On January 1, 1990, follow-up was 95% complete; 2 patients did not attend but were known to be alive. The duration of survival is 2 to 16 years. During this follow-up period death has occurred in 16 patients. It was cardiac related in 6, sudden in 5, and due to intractable left ventricular dysrhythmia in 1. Ten patients died of causes unrelated to their cardiac conditions: cancer in 3, acquired immunodeficiency syndrome contracted preoperatively in 1, neurological stroke in 4, chronic pulmonary insufficiency in 1, and senility in 1. The 16-year actuarial survival curve (Fig 3) shows a 44.2% ± 6% survival rate at 5 years ($n = 23$), a 26% ± 10% survival rate at 10 years ($n = 10$), and a 17.5% ± 12% survival rate at 15 years ($n = 2$).

During this follow-up period, 1 patient was reoperated on for progressive left ventricular insufficiency related to a massive left ventricular aneurysm that had developed on the left ventricular parietal suture. A wide aneurysmectomy was performed. The patient is still surviving 5 years after reoperation.

The 20 survivors are in either class I or class II and receiving minimal therapy. All 20 receive vasodilators, and 12 patients also receive digitalis. None of them has incapacitating angina pectoris or an acute complication of coronary disease such as recurrence of myocardial infarction.

Prognostic Indices

Retrospective univariate analysis comparing patients who died within 1 month postoperatively ($n = 30$) and survivors ($n = 36$) showed that sex and age of patients, preoperative intraaortic balloon pumping, and delay between AMI and the first clinical evidence of VSR had no significant influence (Table 1). The presence of shock at the time of admission and the location of the VSR showed a trend toward significance ($p = 0.08$). Finally, the delay between VSR and time of operation and the response to intensive preoperative medical treatment were highly predictive ($p < 0.001$). These last four factors (shock at time of admission, topography of infarction, duration of preoperative treatment, and response to treatment) are known to be greatly affected by the extent of the myocardial necrotic mass.

We retrospectively evaluated the risk observed in various groups of patients. The first group was made up of patients who could have benefited from cardiac transplantation. They were patients with an age of less than 65 years and no contraindication such as gastric ulcer or cancer. There were 32 potential candidates. The actual early death rate in this group was 41%, and the late death rate during the 16 years of follow-up was 22%.

Because 67% of patients were in cardiogenic shock at

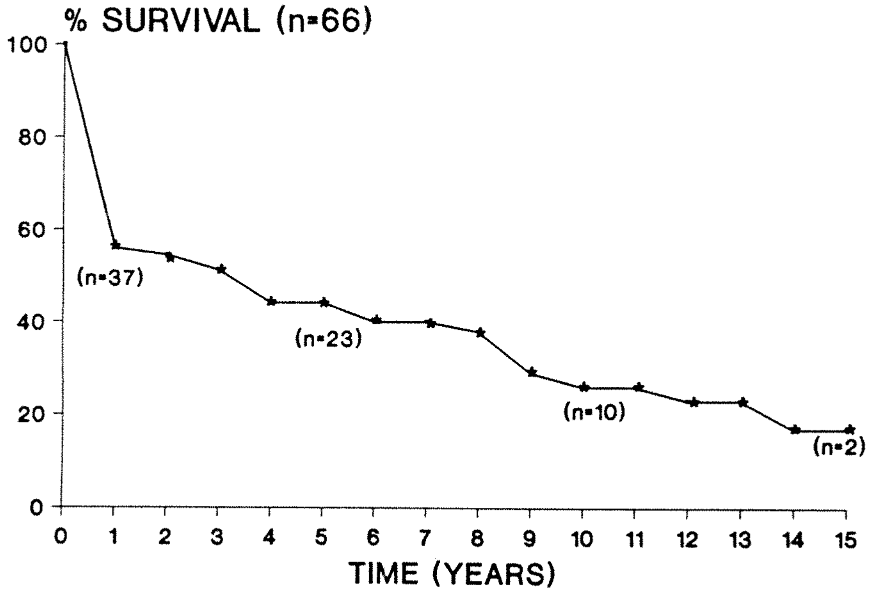

Fig 3. Actuarial survival curve of the 66 patients operated on early for ventricular septal rupture complicating an acute myocardial infarction.

the time of admission, we evaluated the number of patients who could have required temporary implantation of pneumatic ventricles as a bridge to transplantation. The age limit was considered to be 62 years, and a lack of improvement under intensive medical management and intraaortic balloon pumping was the major criterion for selection. There were only 9 patients. Seven died within 1 month and 2 within 1 year. The 1-year mortality rate was therefore 100%. These two figures, a 63% death rate in patients selected according to age only and 100% in patients in extremely serious condition, have to be compared with the present results observed in cardiac transplantation.

Comment

For many years, VSR complicating an AMI has been a severe medical and surgical challenge. In the 1960s mortality with medical treatment alone was as high as 87% within 2 months [8]. The first attempts at surgical repair showed an extremely high rate of postoperative death [9]. Surgical technique became progressively adapted to the original anatomical features of post–AMI septal defects [2, 4]. A major advance was the introduction of Teflon felt septal reinforcement. At the same time, the introduction of mechanical support by balloon pumping [7] seemed to improve the perioperative medical management of these extremely sick patients. These changes led to a more aggressive strategy, which finally should have permitted an improvement in postoperative results [5, 10, 11].

Table 1. Predictive Indices of Survival by Univariate Analysis

Variable	Death < 1 mo (n = 30)	Survivors > 1 mo (n = 36)	*p* Value
F/M ratio (%)	20	38	NS
Age (y)	65 ± 8	64 ± 6	NS
Delay (AMI–VSR) (days)	3.4 ± 4	3.5 ± 4	NS
Delay (VSR-operation) (days)	4.2 ± 4	10.7 ± 12	<0.05
Shock on admission (%)	77	58	0.08
Improved by preop Tt (%)	30	86	<0.001
AMI posterior (%)	47	30	<0.10
VSR posterior (%)	53	34	0.08

AMI = acute myocardial infarction; F/M = female/male; NS = not significant; Tt = treatment; VSR = ventricular septal rupture.

One major criticism of the data presented here is the large period of time, 1973 to 1989, required to accumulate this fairly high number of patients compared with other series and to get an adequate length for the follow-up period. In this time period, many major characteristics of cardiac surgery have been improved, including procedures of extracorporeal circulation, myocardial preservation, and postoperative pharmacological support. The indications for intraaortic balloon pumping have also been subjected to drastic changes. In the 1970s improvement in the patient's condition by prolonged preoperative support was one of the main objectives, leading eventually to the postponement of surgical repair itself by few days. Observation of secondary deterioration after an initial improvement led to prompt operation, as soon as possible [12]. In the 1980s, intraaortic balloon pumping has been used mainly to facilitate postoperative care. Nevertheless, the lack of obvious evidence of a real ventricular unloading effect of diastolic augmentation, together with practical reasons related to aging of the equipment, led us to minimize the use of this technique. Policy toward complete cardiac catheterization preoperatively has also been changed in the early 1980s. This was the consequence of the rather limited number of patients with an indication for coronary bypass grafting. This may account for the substantial number of patients with an extension of AMI or arrhythmia in the postoperative period. Finally, the progressive reduction in the annual number of cases since the early 1980s is obvious. This is not related to more restrictive indications for operation, but to a decrease in the number of patients referred to the hospital.

Despite this progress, the overall risk associated with operation remains high. The year when operation took place does not appear to be a predictive factor. Nevertheless, in more recent years, postoperative death has no longer been due to some aspect of surgical failure, because death has been seldom related to early recurrence of the septal rupture, but is more likely to be related to the extent of myocardial necrosis. In our study, the most significant predictor of postoperative failure appeared to be the response to preoperative intensive care. This confirms the conclusions of other authors [6, 10, 12, 13], who have shown the prognostic value of cardiogenic shock at the time of admission. These two variables are directly related to the amount of necrotic myocardium and also to the extent of ischemic tissue. This observation suggests that further improvement in surgical techniques for closure of the defect will not by themselves improve survival after repair of the septal rupture. Improvement is more likely to result either from more suitable techniques enabling myocardial recovery [14] or more drastic approaches, such as cardiac transplantation.

The introduction of techniques of blood cardioplegia and warm reperfusion specifically designed to cope with myocardial ischemia [14] should lead to improved postoperative left ventricular function, even if there is a rather small number of patients with coronary lesions that can be bypassed. The second alternative, emergency cardiac transplantation, remains a fairly theoretical approach for most patients. The age of the patients, which is often well above 65 years, tends to contraindicate transplantation. A careful retrospective analysis, such as in the present work, demonstrates the scarcity of suitable candidates. Nevertheless, the actuarial survival rate at 1 year of patients undergoing transplantation on an emergency basis is slightly better (70%) than survival of the 32 potential candidates of this series (60%). The analysis made about the potential number of candidates for heroic procedures such as a mechanical bridge to transplantation, who had a 100% death rate at 1 year in this series, would lead more likely to this alternative than to urgent transplantation only.

Little information is available about the long-term outcome after the repair of acute septal rupture in suitable candidates [12, 15]. The present data clearly show that patients who survive the early postoperative period will survive symptom-free for a long period of time. This observation, which on first consideration is somewhat unexpected because of the severity of the initial condition, confirms the role of the myocardial reserve in both the immediate and long-term course of events.

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Cerebral Perfusion During Canine Hypothermic Cardiopulmonary Bypass: Effect of Arterial Carbon Dioxide Tension

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Cerebral blood flow (radioactive microspheres), intracranial pressure (subdural bolt), and retinal histopathology were examined in 20 dogs undergoing 150 minutes of hypothermic (28°C) cardiopulmonary bypass to compare alpha-stat (arterial carbon dioxide tension, 40 ± 1 mm Hg; n = 10) and pH-stat (arterial carbon dioxide tension, 61 ± 1 mm Hg; n = 10) techniques of arterial carbon dioxide tension management. Pump flow (80 mL · kg^{-1} · min^{-1}), mean aortic pressure (78 ± 2 mm Hg), and hemoglobin level (87 ± 3 g/L [8.7 ± 0.3 g/dL]) were maintained constant. During bypass, intracranial pressure progressively increased in the alpha-stat group from 6.0 ± 1.0 to 13.9 ± 1.8 mm Hg ($p < 0.05$) and in the pH-stat group from 7.7 ± 1.1 to 14.7 ± 1.4 mm Hg ($p < 0.05$), although there was no evidence of loss of intracranial compliance or intracranial edema formation as assessed by brain water content. With cooling, cerebral blood flow decreased by 56% to 62% in the alpha-stat group ($p < 0.05$) and by 48% to 56% in the pH-stat group ($p < 0.05$). However, 30 minutes after rewarming to 37°C, cerebral blood flow in both groups failed to increase and remained significantly depressed compared with baseline values. Both groups showed similar amounts of ischemic retinal damage, with degeneration of bipolar cells found in the inner nuclear layer in 67% of animals. We conclude that, independent of the arterial carbon dioxide tension management technique, (1) cerebral perfusion decreases comparably during prolonged hypothermic bypass, (2) intracranial pressure increases progressively, (3) ischemic damage to retinal cells occurs despite maintenance of aortic pressure and flow, and (4) a significant reduction in cerebral perfusion persists after rewarming.

(Ann Thorac Surg 1991;52:479–89)

The incidence of neurological deficits after an otherwise uncomplicated cardiac operation is alarmingly high, with neuroophthalmological abnormalities occurring in 17% to 25% of patients and cognitive dysfunction in 30% to 79% [1–3]. In addition to increasing postoperative morbidity, neurological damage after a cardiac operation entails a threefold higher patient mortality [4]. The etiology of this neurological injury appears multifactorial and may include microembolism, macroembolism, and insufficient cerebral perfusion related to embolic phenomena or to hypotension during cardiopulmonary bypass (CPB) [3, 5].

For editorial comment, see page 417.

Of the multiple factors known to affect the level of cerebral blood flow during CPB, the arterial carbon dioxide tension (PaCO$_2$) may play an important regulatory role [6–8]. The optimal strategy of PaCO$_2$ management during hypothermic bypass has been repeatedly questioned [6–8]. The two currently used techniques to regulate PaCO$_2$ during hypothermic CPB are the alpha-stat method and the pH-stat method. Because cooling increases the solubility of CO$_2$ in blood, its partial pressure declines with hypothermia, resulting in lower PaCO$_2$ values measured by the blood gas analyzer. Using the alpha-stat technique, PaCO$_2$ is maintained at 40 mm Hg when measured by the blood gas analyzer at 37°C. In contrast, using the pH-stat technique, a 3% to 5% mixture of CO$_2$ is bubbled through the oxygenator circuit to maintain the PaCO$_2$ at 40 mm Hg when corrected to the patient's hypothermic core temperature. Measured by the blood gas analyzer at 37°C, the pH-stat value would reflect relative hypercarbia, with a PaCO$_2$ of 60 to 62 mm Hg. Because cerebral blood flow remains coupled to PaCO$_2$ during CPB, the type of PaCO$_2$ management can profoundly affect cerebral perfusion [9]. Whether the measured value of PaCO$_2$ should be corrected for the patient's core temperature (pH-stat) or remain uncorrected (alpha-stat) is controversial, and studies to date have not determined a superior method in terms of altering neurological outcome [9].

If cerebral hypoperfusion is a factor causing neurological sequelae after CPB, then an argument could be made

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for using the pH-stat approach. If, on the other hand, intracranial pressure progressively increases during bypass as reported by Janicki [10], Lundar [11], and their associates, then the hypercarbia induced by the pH-stat method could cause further intracranial hypertension and possibly reduce cerebral perfusion pressure. In addition, any luxury perfusion secondary to the increment in $PaCO_2$ using the pH-stat technique could increase the potential for cerebral embolization [6]. Both of these factors could decrease cerebral perfusion during prolonged CPB. Cerebral microembolization, in particular, has frequently been reported during clinical and experimental CPB studies [12, 13] and has caused ischemic degeneration of retinal cells in 77% of animals after CPB [13]. Because the retinal artery originates from the internal carotid artery, the retina offers a unique opportunity to monitor microcirculatory events occurring in the brain during bypass.

The purpose of the present study was to compare the alpha-stat and pH-stat techniques of $PaCO_2$ management during 150 minutes of hypothermic CPB in dogs with emphasis on intracranial pressure and cerebral blood flow. In addition, we examined retinal histopathology as a monitor of microcirculatory events and microembolism occurring in the brain during CPB. Specifically, we tested whether the pH-stat management of $PaCO_2$ during prolonged hypothermic CPB (1) caused greater intracranial hypertension than the alpha-stat technique and (2) adversely affected cerebral blood flow and retinal histopathology after rewarming.

Material and Methods

Preparation

Animals were handled in the laboratory according to the guidelines approved by the American Physiological Society and the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). The institutional animal care and use committee approved the study.

General anesthesia was induced in 23 microfilaria-free mongrel dogs of either sex weighing 22.1 ± 0.7 kg (range, 17.8 to 26.4 kg) with thiamylal sodium, 20 mg/kg intravenously. After endotracheal intubation, each animal was ventilated with a tidal volume of 18 mL/kg using a volume-cycled ventilator (model 607; Harvard Apparatus, Natick, MA). The respiratory rate was adjusted to maintain normocarbia, and the inspired oxygen concentration was set for an arterial oxygen tension greater than 100 mm Hg. Anesthesia was continued with diazepam, 0.5 to 1.0 mg/kg intravenously, and fentanyl, 100 μg/kg intravenously, administered in incremental doses over a 2-hour period. Additional doses of fentanyl (2 to 3 μg/kg) were given as necessary if an insufficient depth of anesthesia occurred as assessed by transient increases in heart rate or blood pressure and by eye or limb movement. Temporary muscle paralysis was provided by vecuronium bromide, 0.15 mg/kg intravenously, immediately before sternotomy to abolish muscular fasciculations during electrocautery and was discontinued thereafter.

Catheters (inner diameter, 1.67 mm) were inserted in the femoral artery for microsphere sampling and in the femoral vein for fluid administration. Core temperatures were recorded by thermistors (Yellow Springs Instruments, Yellow Springs, OH) placed in the rectum and esophagus. Thermistors were simultaneously calibrated in a water bath before each experiment. Both temporalis muscles were dissected free from the underlying cranium and reflected laterally. A hole 5 mm in diameter was drilled through the right parietal bone, and the underlying dura was incised. A subdural bolt was inserted to measure intracranial pressure and was secured with dental acrylic glue. Each dog received heparin sodium, 4 mg/kg intravenously, with supplements of 1 to 1.5 mg/kg every hour to maintain the activated clotting time greater than 400 seconds.

Through a median sternotomy, the hemiazygos vein was ligated and the pericardium incised and tented. Temperature-compensating solid-state pressure transducers (MPC 500; Millar Instruments, Houston, TX) were placed into the aorta through the right internal mammary artery and into the axillary vein through the right brachial vein. Cannulas (32F) were inserted through the right atrium into the inferior and superior venae cavae to harvest venous return. Depending on the size of the animal, a 24F or 28F straight arterial cannula was inserted through the left subclavian artery and carefully guided to the aorta.

A bubble oxygenator (Model A100S; Shiley, Inc, Irvine, CA) was primed with 1 unit of canine packed red blood cells, 500 mL of 6% hetastarch, and 500 mL of 0.9% saline solution. After the initial hemodilution associated with institution of CPB, additional single-donor units were transfused as necessary to maintain hemoglobin levels constant during CPB, usually between 80 and 90 g/L (8 to 9 g/dL) (Table 1). A 25-μm arterial blood filter and bubble trap (Bentley model AF-1025C; American Hospital Supply Corp, Irvine, CA) was inserted in the arterial perfusion line. Nonpulsatile perfusion was initiated using a roller pump (model 7000; Sarns Inc, Ann Arbor, MI) at a constant pump flow rate of 80 $mL \cdot kg^{-1} \cdot min^{-1}$ throughout the experiment. Total CPB was established after the tightening of the caval snares around the venous cannulas to direct all venous return to the bubble oxygenator. Peripheral venous pressure was measured in the right axillary vein to ensure adequate drainage of the cerebral circulation by the superior vena caval cannula.

During bypass, mean aortic blood pressure was maintained at 78 ± 2 mm Hg by infusing phenylephrine hydrochloride (60 μg/mL mixed in 500 mL of saline solution) as necessary. Canine studies [14] demonstrated that vasopressor support with phenylephrine does not affect cerebral blood flow. In humans, α-adrenergic vasopressors have minimal effects on cerebral vascular tone or on CO_2 reactivity [15]. The arterial oxygen tension was adjusted using an air-oxygen mixture in the oxygenator. The $PaCO_2$ was maintained at a predetermined level according to the group protocol by adjusting the gas flow rate in the oxygenator with the addition of 5% CO_2 as necessary during the period of hypothermic CPB.

Table 1. Oxygenation Variables During Hypothermic Cardiopulmonary Bypass[a]

Variable	Group	Baseline	Hypothermic CPB (min)			After Rewarming
			30	90	150	
pH	Alpha-stat	7.39 ± 0.02	7.39 ± 0.01	7.40 ± 0.01	7.40 ± 0.01	7.38 ± 0.02
	pH-stat	7.39 ± 0.01	7.28 ± 0.02[b,c]	7.30 ± 0.03[c]	7.30 ± 0.04[c]	7.41 ± 0.03[b]
$PaCO_2$ (mm Hg)	Alpha-stat	40.7 ± 0.04	40.3 ± 0.5	39.6 ± 0.5	39.6 ± 0.5	39.4 ± 0.6
	pH-stat	39.5 ± 0.6	61.7 ± 0.6[b,c]	61.2 ± 0.5[c]	61.8 ± 0.5[c]	39.8 ± 0.4[b]
PaO_2 (mm Hg)	Alpha-stat	351 ± 34	341 ± 37	312 ± 25	327 ± 37	316 ± 36
	pH-stat	348 ± 22	344 ± 36	358 ± 53	334 ± 60	307 ± 38
Hemoglobin (g/L)[d]	Alpha-stat	92 ± 5	90 ± 4	91 ± 5	89 ± 4	88 ± 3
		(9.2 ± 0.5)	(9.0 ± 0.4)	(9.1 ± 0.5)	(8.9 ± 0.4)	(8.8 ± 0.3)
	pH-stat	86 ± 4	88 ± 3	87 ± 3	88 ± 3	83 ± 3
		(8.6 ± 0.4)	(8.8 ± 0.3)	(8.7 ± 0.3)	(8.8 ± 0.3)	(8.3 ± 0.3)
Glucose (mmol/L)[e]	Alpha-stat	8.7 ± 0.7	6.8 ± 1.4	8.0 ± 1.8	8.4 ± 1.4	6.5 ± 1.5
		(157 ± 13)	(123 ± 25)	(144 ± 33)	(151 ± 26)	(117 ± 27)
	pH-stat	9.4 ± 1.6	8.4 ± 1.3	7.9 ± 1.6	7.9 ± 1.5	7.7 ± 1.6
		(170 ± 28)	(152 ± 23)	(142 ± 28)	(142 ± 27)	(139 ± 28)
Serum osmolality (mosm/L)	Alpha-stat	306 ± 7	313 ± 7	320 ± 7	311 ± 7	323 ± 9
	pH-stat	314 ± 7	304 ± 4	317 ± 7	314 ± 8	324 ± 9
Rectal temperature (°C)	Alpha-stat	38.3 ± 0.2	28.4 ± 0.2[b]	28.1 ± 0.1	28.1 ± 0.1	37.4 ± 0.2[b]
	pH-stat	37.6 ± 0.4	28.5 ± 0.3[b]	27.9 ± 0.2	27.7 ± 0.2	37.1 ± 0.3[b]

[a] Data are shown as the mean ± the standard error of the mean. [b] Significance: $p < 0.05$ compared with previous value. [c] Significance: $p < 0.05$ compared with alpha-stat value. [d] Values in parentheses are in grams per deciliter. [e] Values in parentheses are in milligrams per deciliter.

CPB = cardiopulmonary bypass; $PaCO_2$ = arterial carbon dioxide tension; PaO_2 = arterial oxygen tension.

Measurements

Cerebral blood flow (mL · min^{-1} · 100 g^{-1}) was quantitated by injecting 1 million to 2 million microspheres (15 ± 3 μm in diameter) labeled with gadolinium 153, strontium 85, scandium 46, chromium 51, and tin 113. The microspheres were sonicated for 30 minutes, mixed with 3 mL of saline solution, vortexed, and injected over 15 seconds through the subclavian cannula into the aorta as described by Rudy and co-workers [16]. Reference sampling from the femoral artery was begun 10 seconds before microsphere injection; samples were collected in separate tared vials at 6.5 mL/min over a 5-minute collection period. Absence of radioactivity in the last arterial sample ensured complete microsphere entrapment without recirculation. Tissue blood flow (mL · min^{-1} · 100 g^{-1}) was calculated as $Q_t = [(Q_r \cdot C_t)/C_r] \cdot 100$, where Q_t = tissue blood flow, Q_r = reference sample blood flow, and C_t and C_r = radioactivity in tissue and reference samples, respectively. Total cerebral blood flow was determined from both entire hemispheres, and each measurement contained greater than 400 microspheres.

Arterial blood was sampled from the aortic cannula for blood gas analysis (model 1304 blood gas analyzer; Instrumentation Laboratory, Lexington, MA) and oxygen content (Instrumentation Laboratory co-oximeter model 282). Serum osmolality was measured using a vapor pressure osmometer (Westcore, Inc, Logan, VT); serum glucose was quantitated using a glucometer (Mills Laboratories, Elkhart, IN). Cerebral perfusion pressure was calculated as the difference between mean aortic blood pressure and intracranial pressure. Cerebral vascular resistance (resistance units [ru]) was calculated by dividing cerebral perfusion pressure by cerebral blood flow. Cerebral oxygen delivery was calculated as the product of arterial oxygen content and cerebral blood flow.

Intracranial compliance was determined by injecting 1 mL of saline solution into the subdural space over 2 to 3 seconds and measuring the maximal pressure change. After each injection, intracranial pressure returned to the preinjection value over 90 to 120 seconds, which is similar to the time required using a cisternal injection technique as reported by others [17]. The sequence was then repeated using 2 mL of saline solution. Intracranial compliance was defined by the slope and intercept of the log pressure–volume response to 0-, 1-, and 2-mL injection volumes using a modification of the technique described by Marmarou and colleagues [17].

Protocol

A schematic diagram of the experimental protocol is shown in Figure 1. Baseline normothermic measurements were taken after the animal's condition had stabilized on CPB for 15 minutes. Next, hypothermia to 28°C (rectal temperature) was induced by systemic cooling using a thermostatically controlled water bath (Sarns Inc). During this period, each animal was assigned by simple randomization to a $PaCO_2$ management group using the alpha-stat ($PaCO_2$, 40 ± 1 mm Hg) or pH-stat ($PaCO_2$, 61 ± 1 mm Hg) technique. Repeat measurements were taken 30, 90, and 150 minutes after stable hypothermic condi-

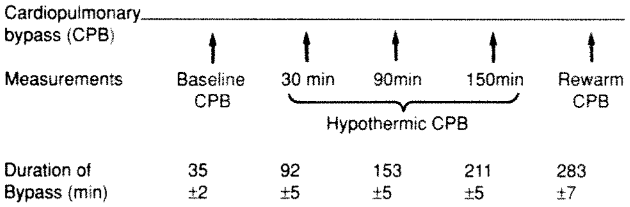

Fig 1. The experimental protocol during cardiopulmonary bypass illustrating the measurement intervals.

tions had been reached. Then, both groups of animals were rewarmed to 37°C, and repeat measurements were obtained after stabilization at normothermia for 30 minutes. During both cooling and warming procedures, the gradient between water bath and core (rectal) temperatures did not exceed 10°C [18]. Throughout the study, other variables such as pump flow, mean aortic pressure, hematocrit, glucose, and anesthesia management were maintained constant and comparable between groups, so that the effect of $PaCO_2$ during hypothermic CPB could be studied exclusively.

Tissue Analysis

After the discontinuation of CPB, the brain was quickly removed and immediately covered with a moist towel to prevent drying. Biopsy samples from each cerebral hemisphere were placed in tared vials, weighed, and dried in a convection oven at 85°C for 5 days until a stable dry weight was obtained. Percent water content was calculated as [1 − (dry weight/wet weight)] · 100. Cerebral water contents from alpha-stat and pH-stat groups were compared with those from a separate group of dogs that had not undergone CPB (n = 10). Regional cerebral blood flow was determined in the entire left and right cerebral hemispheres, the cerebellum, and the retina from one eye. A 1-cm^3 biopsy sample from the right cerebral hemisphere was taken immediately below the subdural bolt and counted separately to assess possible cerebral injury arising from bolt placement per se.

Retinal Histopathology

The other eye was enucleated and fixed in Zenker's acetate fluid for 1 hour and rinsed with distilled water. The specimen was dehydrated in ethanol and trimmed laterally to remove the lens, and the eye was embedded in paraplast. Parasagittal sections (10 μm thick) were made through the retina and optic nerve. At approximately 200-μm intervals, sections were prepared on slides and stained with hematoxylin and eosin. Specimens were examined by one of us (W.K.O'S.), who was blinded to the randomization sequence, and were categorized according to the lateral extent of nuclear pyknosis of the inner nuclear layer of bipolar cells to assess ischemic injury [19, 20]. Retinas were morphometrically analyzed according to lateral extent of retinal damage using a scoring system developed in our laboratory: none, 0 μm; mild, 252 ± 51 μm; moderate, 1,028 ± 217 μm; and severe, 19,256 ± 1,459 μm, involving the entire retinal extent. All histopathological data from the retinal cells were collected and analyzed without prior knowledge of the animal's treatment group.

Data Analysis

Data, reported as the mean ± the standard error of the mean, were analyzed by repeated-measures analysis of variance to test the effects of time and $PaCO_2$ during bypass. When the time effect was significant, paired t tests were used to assess individual time contrasts, and Holm's sequentially rejective multiple test procedure controlled the type 1 error at an α level of 0.05 [21]. Right and left cerebral blood flows were compared using linear regression analysis. The slopes and intercepts of the log pressure–volume responses taken at the baseline CPB and rewarm intervals were compared by time and group using a multivariate analysis of variance.

Control Studies

Two series of control experiments were performed to validate the measurement of cerebral blood flow using radioactive microspheres during hypothermic CPB. First, adequate mixing is achieved after injecting the radioactive microspheres through the aortic cannula. Second, complete first-pass uptake of microspheres occurs in the cerebral tissue, and this process is not affected by prolonged hypothermic CPB. Failure of microspheres to initially lodge in the cerebral capillaries could cause recirculation and a falsely low estimate of cerebral blood flow.

ADEQUATE MIXING OF RADIOACTIVE MICROSPHERES. Eight animals anesthetized and prepared as already described underwent 150 minutes of hypothermic (28° ± 1°C) CPB followed by rewarming to 37° ± 1°C. Two million to 3 million microspheres (15 ± 3 μm) labeled with gadolinium 153, strontium 85, scandium 46, chromium 51, and tin 113 were injected at the measurement intervals indicated in Figure 1. Microspheres were sonicated for 30 minutes, mixed with 3 mL of saline solution, vortexed, and injected over 15 seconds through the subclavian cannula into the aorta. Reference sampling from the femoral artery was begun 10 seconds before microsphere injection; the samples were collected in tared vials at 6.5 mL/min for 5 minutes. The adequacy of microsphere mixing was determined by comparing right and left renal blood flows. At the conclusion of each experiment, tissue samples from each renal cortex were weighed and counted separately for radioactivity. Right and left renal cortical blood flows were compared using linear regression analysis. As seen in Figure 2, the slope of this relationship approached unity with $r = 0.99$.

In the 8 dogs, right and left renal cortical blood flows were similar, indicating adequate microsphere mixing after injection through the aortic cannula on CPB. These data further support the findings of Rudy and co-workers [16] that microspheres injected through the perfusion cannula on CPB are properly mixed.

FIRST-PASS UPTAKE OF MICROSPHERES BY CEREBRAL TISSUE. Five animals were anesthetized and prepared as previously described except that the dorsal sagittal sinus was directly cannulated with a 22-gauge catheter. At the measurement intervals indicated in Figure 1 during 150 minutes of hypothermic (28°C) CPB followed by rewarming, venous blood was aspirated simultaneously with the arterial reference samples after microsphere injection. Using two precalibrated pumps that aspirated blood at 6.5 mL/min, samplings from the dorsal sagittal sinus and femoral artery were started 10 seconds before microsphere injection through the aortic cannula and continued for 5 minutes. The radioactivity in the dorsal sagittal sinus samples was compared with that in the femoral artery to determine the degree of arteriovenous shunting across the brain during CPB. Using the method described by Heistad and co-workers [22], cerebral arteriovenous shunting was calculated as (venous radioactivity/arterial radioactivity) · 100. Similar to the finding of Heistad and associates [22], the degree of arteriovenous shunting across the brain was less than 1% (Fig 3). Moreover, intracerebral shunting did not appear to change with hypothermia, rewarming, or the duration of CPB (see Fig 3).

In the 5 dogs, intracerebral shunting causing recirculation of microspheres did not occur during hypothermic CPB and did not adversely affect the accurate determination of cerebral blood flow.

Results

The study was successfully completed in 20 of the 23 animals (alpha-stat, n = 10; pH-stat, n = 10). Three animals were eliminated from the study, 1 with severe hypotension occurring immediately after blood transfusion during CPB and 2 with intracerebral hemorrhage as a direct complication of bolt placement. The total time for bypass was similar in both groups (283 ± 7 minutes for the alpha-stat group and 282 ± 7 minutes for the pH-stat group; p = not significant). As seen in Tables 1 and 2, values of mean aortic pressure, arterial oxygen tension, hemoglobin, glucose, osmolality, and temperature were comparable between groups throughout the study periods. By design, $PaCO_2$ and pH values differed significantly between groups during hypothermia. Axillary venous pressure measured cephalad to the tip of the cannula in the superior vena cava averaged 0.2 ± 0.1 mm Hg (range, −2 to 3 mm Hg) during bypass, indicating adequate venous drainage of the cerebral circulation without obstruction.

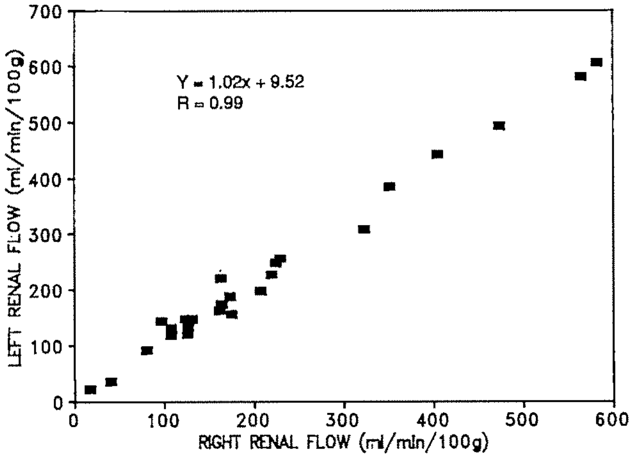

Fig 2. Correlation between left and right renal cortical blood flows in 8 animals. The slope and correlation coefficient of this relationship indicate adequate mixing of radioactive microspheres injected through the aortic cannula.

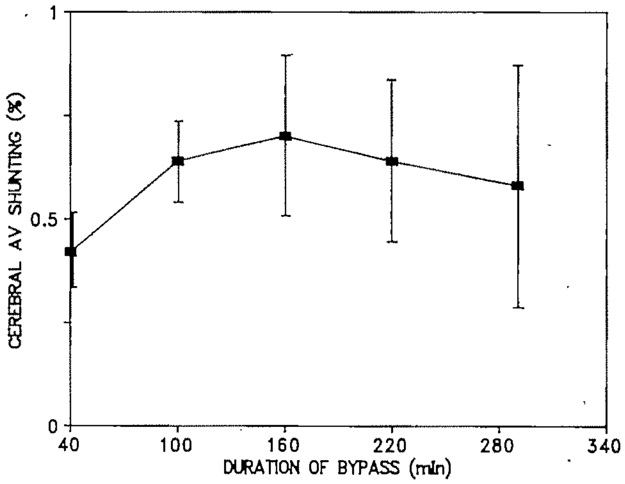

Fig 3. In 5 animals, the amount of arteriovenous (AV) shunting through the cerebral hemispheres was less than 1%. Moreover, this relationship did not change with hypothermia and rewarming. Thus, the changes in cerebral blood flow cannot be attributed to inadequate microsphere trapping in the cerebral capillary bed.

Intracranial pressure increased during CPB from 6.0 ± 1.0 mm Hg to 13.9 ± 1.8 mm Hg with alpha-stat management ($p < 0.05$) and from 7.7 ± 1.1 mm Hg to 14.7 ± 1.4 mm Hg with pH-stat management ($p < 0.05$) (see Table 2). In both groups, the increase in intracranial pressure progressed with the duration of bypass so that each subsequent measurement was greater than the preceding measurement. However, intracranial compliance did not differ between groups when baseline and rewarm values were compared (Table 3). As seen in Figure 4, the slopes of the log pressure–volume relations for the alpha-stat (Fig 4A) and pH-stat (Fig 4B) groups appeared similar and unchanged by bypass. The significant increase in intercept values with CPB after rewarming may be secondary to the increase in intracranial pressure per se (see Table 2). Cerebral water content was 76.7% ± 0.5% (alpha-stat) and 75.8% ± 0.6% (pH-stat) (p = not significant between groups). These values were similar to those found in the 10 animals not involved in this study that were not subjected to CPB (76.1% ± 0.4%). There were no differences in cerebral water content among the groups, indicating that neither CPB per se nor the strategy of $PaCO_2$ management during CPB promoted intracranial edema formation.

Subdural bolt placement in the right cerebral hemisphere did not adversely affect cerebral blood flow, as perfusion to the cerebral tissue immediately below the bolt correlated closely with right cerebral blood flow (slope = 1.08; $r = 0.92$; SEE = 8.1 mL · min^{-1} · 100 g^{-1}).

Table 2. Hemodynamic Variables During Hypothermic Cardiopulmonary Bypass[a]

			Hypothermic CPB (min)			
Variable	Group	Baseline	30	90	150	After Rewarming
Mean aortic pressure (mm Hg)	Alpha-stat	80 ± 1	82 ± 1	76 ± 1	78 ± 1	78 ± 1
	pH-stat	80 ± 1	80 ± 1	78 ± 1	77 ± 1	81 ± 2
Intracranial pressure (mm Hg)	Alpha-stat	6.0 ± 1.0	7.2 ± 0.7[b]	9.1 ± 1.2[b]	10.6 ± 1.2[b]	13.9 ± 1.8[b]
	pH-stat	7.7 ± 1.1	9.6 ± 1.3[b]	11.0 ± 1.3[b]	12.3 ± 1.2[b]	14.7 ± 1.4[b]
Cerebral perfusion pressure (mm Hg)	Alpha-stat	73.5 ± 1.4	74.5 ± 1.2	67.4 ± 1.9	67.8 ± 1.6	63.6 ± 2.4
	pH-stat	72.9 ± 1.9	70.0 ± 2.1	67.1 ± 2.1	64.9 ± 2.2	66.4 ± 1.6
Cerebral vascular resistance (ru)	Alpha-stat	1.5 ± 0.1	3.8 ± 0.3[b]	3.2 ± 0.3	3.2 ± 0.2	5.0 ± 1.4
	pH-stat	1.4 ± 0.1	2.7 ± 0.3[b]	2.8 ± 0.2	2.8 ± 0.3	5.8 ± 1.9

[a] Data are shown as the mean ± the standard error of the mean. [b] Significance: $p < 0.05$ compared with previous value.

CPB = cardiopulmonary bypass.

Similarly, cerebral blood flows in the left and right cerebral hemispheres were similar and therefore combined for total cerebral perfusion. Overall, total cerebral blood flow was similar in both groups after the initiation of CPB (Table 4). Cerebral blood flow remained stable and comparable in both groups during hypothermic bypass (Fig 5A). However, after rewarming to 37°C, both groups showed a persistent reduction in cerebral blood flow to values that were reduced by 59% and 65% of baseline values in the alpha-stat group and the pH-stat group, respectively ($p < 0.05$ between baseline and rewarm values for each group) (see Fig 5A). Cerebral oxygen delivery decreased similarly with hypothermia in both groups and remained significantly lower than baseline values after rewarming (see Table 4). Cerebellar perfusion decreased during hypothermic CPB but, unlike cerebral blood flow, increased significantly in both groups after rewarming (Fig 5B; see Table 4).

As mean aortic blood pressure was maintained constant, cerebral perfusion pressure decreased significantly after rewarming compared with baseline in both groups because of the significant increases in intracranial pressure (see Table 2). Cerebral vascular resistance was similar in both groups at baseline and increased significantly during hypothermia in both (Fig 6; see Table 2). It remained relatively constant during hypothermic bypass. However, after rewarming, cerebral vascular resistance tended to increase further in both groups, so that the values after rewarming were significantly greater than the baseline values despite comparable normothermic temperatures.

Retinas from 18 animals (alpha-stat, n = 10; pH-stat, n = 8) were morphometrically analyzed and grouped according to the lateral extent of retinal damage in bipolar cells. Retinas from 2 animals were not suitable for pathological examination because of technical errors during processing. Examples of normal and abnormal retinal histopathology are shown in Figure 7. There was no difference in the distribution of injury between groups ($p = 0.584$) (Table 5). Ischemic degeneration of the bipolar cells in the inner nuclear layer occurred in nearly 67% of animals irrespective of $PaCO_2$ management. Retinal microemboli were visualized in 2 animals, 1 having a platelet embolus (alpha-stat) and the other, a leukocyte embolus (pH-stat). Retinal thicknesses (measured 250 μm from the optic nerve) were similar in both groups, which further indicates that the different techniques of $PaCO_2$ management did not adversely affect edema formation in the retina. The changes in cerebral and retinal blood flows appeared similar in that both failed to increase after rewarming (see Table 4).

Comment

We found that the strategy of $PaCO_2$ management during prolonged hypothermic CPB had no effect on cerebral blood flow, intracranial pressure or compliance, or retinal histopathology. However, cerebral blood flow after rewarming from hypothermic CPB was not restored to baseline levels in either group despite similar normothermic temperatures, $PaCO_2$, pump flow, and mean aortic blood pressure. This amount of cerebral blood flow reduc-

Table 3. Intracranial Compliance Data During Cardiopulmonary Bypass[a]

	Alpha-stat		pH-stat	
Variable	Baseline	After Rewarming	Baseline	After Rewarming
Slope (mm Hg/mL)	0.29 ± 0.04	0.23 ± 0.02	0.26 ± 0.04	0.21 ± 0.02
Intercept (mm Hg)	0.81 ± 0.06	1.17 ± 0.07[b]	0.89 ± 0.05	1.13 ± 0.05[b]
Correlation coefficients (range)	0.975–0.998	0.898–0.999	0.937–0.999	0.929–0.997

[a] Where applicable, data are shown as the mean ± the standard error of the mean. [b] Significance: $p < 0.05$ compared with baseline value.

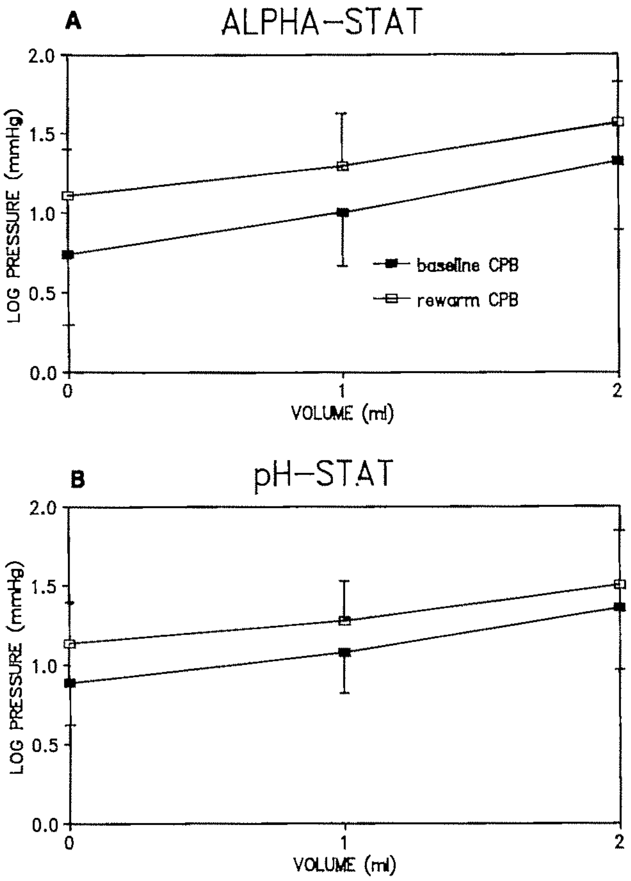

Fig 4. Cerebral compliance curves for (A) alpha-stat and (B) pH-stat groups comparing the baseline and rewarm measurement periods. The log pressure–volume relations in each group appeared similar, indicating that hypothermic cardiopulmonary bypass (CPB) did not alter cerebral compliance. The significant upward shift after rewarming may be secondary to the increase in intracranial pressure per se.

tion exceeded that expected from the mild but significant increase in intracranial pressure per se, which caused cerebral perfusion pressure to decline. The reduction in cerebral blood flow reflected a persistent increase in cerebral vascular resistance by 260% to 380% over baseline values. The etiology of this deficit is unknown, but its temporal appearance after rewarming indicates a potentially vulnerable period for oxygen supply-and-demand mismatch in the brain, particularly as cerebral oxygen delivery remained significantly depressed. Resolution of this perfusion deficit after the termination of CPB may be

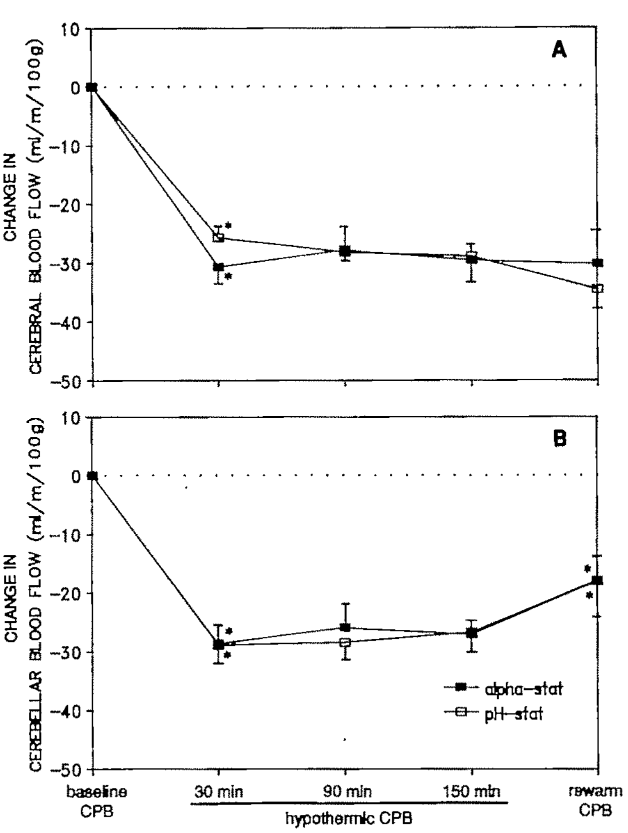

*Fig 5. Differences in blood flow relative to baseline values in the (A) cerebral hemispheres and (B) cerebellum for alpha-stat and pH-stat groups. Perfusion to both regions decreased significantly with hypothermia to comparable levels between groups. After rewarming, cerebral blood flow remained depressed whereas cerebellar blood flow increased, albeit not to baseline values. (CPB = cardiopulmonary bypass; * = p < 0.05 compared with previous value.)*

Table 4. Blood Flows and Cerebral Oxygen Delivery During Cardiopulmonary Bypass[a]

Tissue	Group	Baseline	Hypothermic CPB (min) 30	90	150	After Rewarming
Cerebral cortex ($mL \cdot min^{-1} \cdot 100\ g^{-1}$)	Alpha-stat	52.9 ± 4.3	20.3 ± 1.7[b]	23.8 ± 2.6	23.1 ± 1.9	21.5 ± 4.2
	pH-stat	55.7 ± 2.6	28.7 ± 4.1[b]	25.6 ± 2.8	24.7 ± 2.7	19.7 ± 3.5
Cerebellum ($mL \cdot min^{-1} \cdot 100\ g^{-1}$)	Alpha-stat	58.1 ± 4.5	26.7 ± 3.3[b]	30.8 ± 3.3	30.6 ± 2.6	38.6 ± 5.7[b]
	pH-stat	57.1 ± 3.3	25.8 ± 2.6[b]	26.0 ± 2.7	27.3 ± 2.3	37.1 ± 4.5[b]
Retina ($mL \cdot min^{-1} \cdot 100\ g^{-1}$)	Alpha-stat	42.3 ± 7.3	20.6 ± 4.5[b]	30.0 ± 2.3[b]	31.3 ± 2.6	18.9 ± 5.1[b]
	pH-stat	47.7 ± 7.0	25.7 ± 6.5[b]	31.5 ± 9.2[b]	31.4 ± 6.5	13.3 ± 1.8[b]
Cerebral oxygen delivery ($mL\ O_2 \cdot mL^{-1} \cdot min^{-1} \cdot 100\ g^{-1}$)	Alpha-stat	6.23 ± 0.34	2.55 ± 0.19[b]	2.76 ± 0.15	2.66 ± 0.18	2.58 ± 0.51
	pH-stat	6.29 ± 0.39	3.53 ± 0.52[b]	3.03 ± 0.33	2.92 ± 0.26	2.25 ± 0.42

[a] Where applicable, data are shown as the mean ± the standard error of the mean.
[b] Significance: $p < 0.05$ compared with previous value.
CPB = cardiopulmonary bypass.

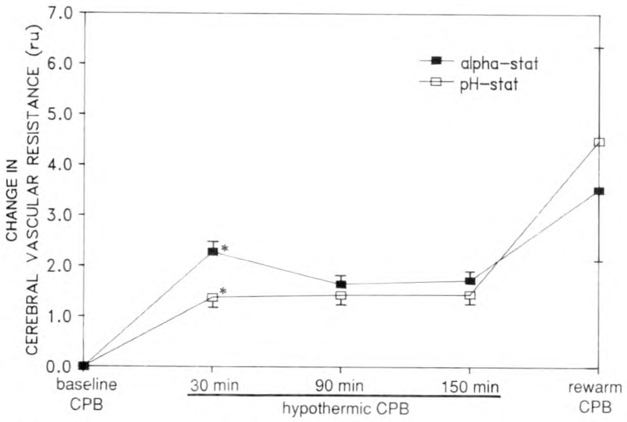

Fig 6. Differences in cerebral vascular resistance relative to baseline values in alpha-stat and pH-stat groups. Cerebral vascular resistance increased in both groups during hypothermic cardiopulmonary bypass (CPB) and remained elevated despite rewarming. (= $p < 0.05$ compared with previous value.)*

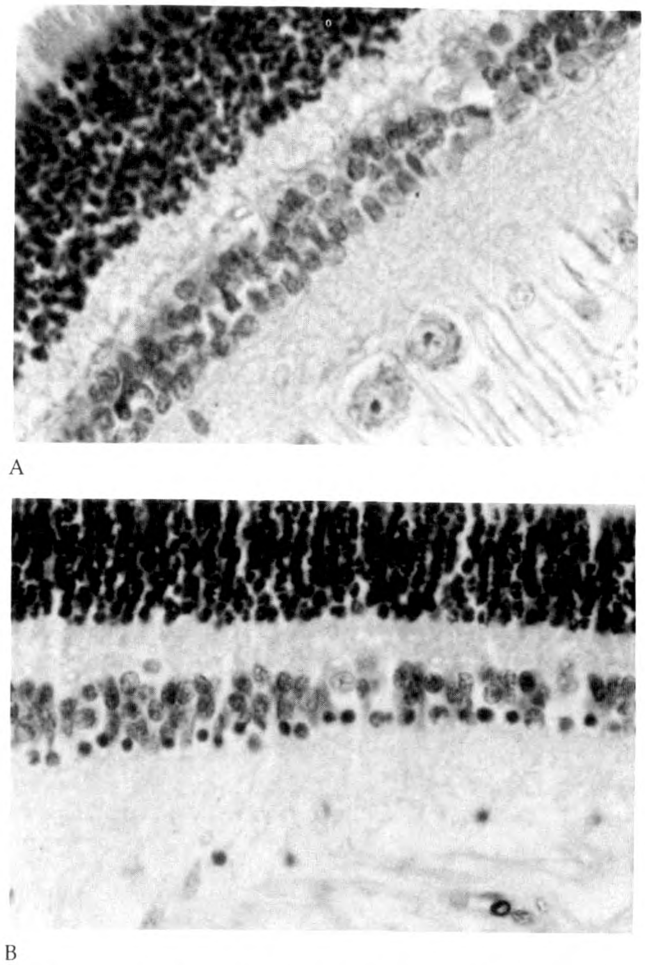

Fig 7. Examples of retinal histopathology of the bipolar cells of the inner nuclear layer showing (A) normal histology and (B) severe nuclear pyknosis. With normal histology, the nuclei of bipolar cells have a diffuse, granular staining pattern of chromatin. The cells appear globular with normally staining cytoplasm. In contrast, with severe retinal damage, the bipolar cells shrink, and markedly pyknotic nuclei, cytoplasmic clearing, and mild intercellular edema are present. The lateral extent of nuclear pyknosis from the optic nerve was quantitated as an index of ischemic damage occurring during cardiopulmonary bypass.

important in altering the development and progression of neurological dysfunction in cardiac surgical patients.

In clinical studies, several investigators [6–8] have found that a significant correlation exists between cerebral blood flow during hypothermic CPB and $PaCO_2$ levels, with higher blood flows reported using the pH-stat technique of $PaCO_2$ management. Our results after 30 minutes of hypothermic bypass are not consistent with these clinical studies. Because cerebral blood flow decreases with time during hypothermic bypass in patients [23], this finding may reflect a greater time lapse in the present study. After 30 minutes of hypothermic CPB, any apparent responsiveness to $PaCO_2$ management was lost, and cerebral perfusion remained relatively constant and similar between groups. As hypothermia reduces the cerebral metabolic rate, perfusion falls proportionately. Using the pH-stat technique of $PaCO_2$ management during hypothermic bypass could result in a state of relative cerebral hyperperfusion, which could increase the delivery of microemboli to the cerebral circulation [6]. However, we found that any differences in cerebral perfusion between the alpha-stat and pH-stat techniques were minimal and disappeared rapidly with the duration of bypass, so that cerebral blood flow remained constant over 150 minutes. In addition, a similar degree of ischemic retinal injury was found independent of $PaCO_2$ management. Because comparable levels of retinal blood flow occurred in the two groups during hypothermic bypass, a similar degree of retinal histopathology would have been expected.

Clinical studies have documented that cerebral hypoperfusion occurs after hypothermic CPB. After deep hypothermia and circulatory arrest for repair of congenital defects, Greeley and co-authors [24] reported that cerebral blood flow failed to increase in infants after rewarming and discontinuation of bypass. Cerebral perfusion after rewarming in those studies was similar to the depressed level found in the present study. These findings indicate that the magnitude of hypothermia, whether profound [24] or prolonged [present study], may be important in the pathogenesis of reduced cerebral perfusion after rewarming. Although metabolic studies were not performed in these studies to assess cerebral oxygen supply-and-demand balance, rewarming may represent a particularly vulnerable period for cerebral ischemia, as also suggested by Croughwell and co-workers [25]. During

Table 5. Distribution of Retinal Damage in Dogs After Cardiopulmonary Bypass

Group	None	Mild	Moderate	Severe
Alpha-stat (n = 10)	4	4	0	2
pH-stat (n = 8)	2	3	2	1

rewarming, 23% of patients showed significant jugular bulb desaturation, as the cerebral metabolic rate remained disproportionately elevated relative to cerebral blood flow. Further study of the pathogenesis and resolution of this perfusion deficit is clearly warranted for complete understanding of CPB-induced neurological dysfunction.

The significant decrease in cerebral blood flow during CPB after rewarming may be related to a decrease in cerebral perfusion pressure or to an increase in cerebral vascular resistance. Intracranial pressure increased with the duration of bypass in both groups of animals independent of which $PaCO_2$ management technique was used during hypothermia. That intracranial pressure increases during CPB was reported by Janicki and colleagues [10], in whose study the tonicity of the prime solution and the duration of bypass significantly potentiated the development of intracranial hypertension in sheep. In that study, acute hypercarbia caused a marked increase in intracranial pressure during CPB, but our measurements of steady-state hypercarbia in the pH-stat group showed no differences compared with the alpha-stat group. These findings reflect the fact that cerebral blood flows during hypercarbia were similar and independent of $PaCO_2$ management. Because intracranial compliance and brain water content were not altered by CPB, the progressive increment in intracranial pressure may be secondary to venous dilation, which has been reported to occur during nonpulsatile perfusion [26]. Although we found a significant increase in intracranial pressure with the duration of bypass, the magnitude of this increase was sufficient to significantly reduce cerebral perfusion pressure by 9% to 13% but could not account for the 59% to 65% decrease in cerebral blood flow after rewarming. Consequently, the persistent reduction in cerebral perfusion pressure after rewarming appears secondary to a progressive increase in cerebral vascular resistance.

Lees and associates [27] reported that the duration of CPB correlated closely with an increment in cerebral vascular resistance ($r = 0.68$; $p < 0.01$). Our results indicate that the major increase in cerebral vascular resistance during CPB occurred after rewarming. Because cerebral metabolism was not assessed in the present study, our comments on the etiology of any alteration in cerebral vascular resistance after hypothermic CPB are observational and speculative. Potential causes could include (1) plugging of arterioles and capillaries by formed elements (ie, platelets and leukocytes activated by the rewarming process), (2) swelling of pial and endothelial cells in the cerebral capillary bed, (3) capillary leakage causing interstitial edema and luminal compression, (4) air or particulate microembolization, or (5) vasoconstriction. Although aggregates of leukocytes and platelets participate in postischemic hypoperfusion in other organs, formed elements have not been reported in cerebral vessels after CPB [28, 29]. In the present study, leukocyte and platelet plugs were found in retinal vessels in only 1 animal. Although cerebral vascular resistance may increase secondary to endothelial cell swelling and luminal compression, morphological examinations of human and canine brains after CPB have revealed scattered cerebral capillary aneurysmal dilatations without narrowing or occlusion of the vascular lumen [29]. Further, the present study found neither an increase in brain water content in the animals that underwent bypass compared with animals that did not nor any change in intracranial compliance. Consequently, these three potential mechanisms—arteriolar plugging by formal elements, endothelial cell swelling, and perivascular edema formation—may not be responsible for the persistent increment in vascular resistance after hypothermic CPB.

Using retinal fluorescein angiography during bypass, Blauth and colleagues [13] found that 100% of patients showed evidence of retinal microembolism, which correlated with postoperative neuropsychometric deficits ($p = 0.075$). These microemboli may be particulate in nature, which rapidly dissolve, or alternatively, may be gaseous [12, 13]. Our group [29] has previously documented the presence of small arteriolar and capillary dilatations in experimental animals and patients dying after CPB. Although their exact cause is unknown, we believe they represent a microvascular injury secondary to gaseous or soluble particulate (ie, fat) microembolization. Fat microemboli in the cerebral circulation have been described after CPB [30], although their presence appeared independent of the duration of CPB and may have been secondary to blood transfusions. Cerebral embolization secondary to microbubbles occurring during CPB, especially during rewarming, may be responsible for the persistent cerebral hypoperfusion seen in our animals. In rabbits, cerebral blood flow progressively decreased after air microembolism disproportionate to the amount of capillary obstruction, and this decrease correlated closely with depressed neuronal function evaluated by cortical somatosensory evoked responses [31]. In addition to causing transient vascular stasis in the cerebral capillary bed, bubble microemboli can promote platelet aggregation, cause direct endothelial damage, and initiate the release of vasoactive substances, all of which can adversely affect cerebral perfusion.

Consequently, these two possible mechanisms (ie, gaseous microemboli and secondary cerebral vasoconstriction) may account for the significant reduction in cerebral perfusion after rewarming. Our comments remain speculative, as the potential recruitability of cerebral perfusion after CPB was not assessed in this study. However, our findings are compatible with these theories. Rewarming may predispose to the number and size of air microemboli in the cerebral vasculature because of changes in gas solubility as blood temperature increases from 28° to 37°C [18]. Or the period of hypothermia may mask a steady and progressive insult to cerebral perfusion from repetitive microembolization occurring throughout CPB, which becomes apparent only after rewarming. Regardless, this level of cerebral hypoperfusion may predispose to cognitive dysfunction postoperatively by causing direct neuronal damage or by retarding normal reparative processes.

Shaw and associates [2] found neuroophthalmological complications consisting of visual acuity decrease, field defects, and temporary blindness in 25% of patients after CPB. In our study, the persistent decrease in retinal perfusion and the amount of ischemic degeneration of bipolar cells were alarming. Nuclear pyknosis of bipolar cells represents an early marker of ischemic damage, although its contribution to visual impairment after CPB could not be evaluated in these experiments. The inner nuclear layer is located in the vascular watershed boundary area between choroidal and retinal artery circulations and has the lowest tissue oxygen tension of any retinal cell layer [20]. Consequently, these cells degenerate rapidly with hypoperfusion and hypoxia and represent a sensitive indicator of retinal ischemia. In contrast, cells in other retinal layers fail to show consistent changes with ischemia. With mild hypoxia, bipolar cells shrink, with clearing in the cytoplasm and clumping of the nuclear chromatin; with severe hypoxia, nuclear pyknosis results [19, 20] (see Fig 7B).

Our incidence of ischemic degeneration of bipolar cells is similar to that reported by Blauth and colleagues [13]. In their study, ischemic changes were found in retinal ganglion cells in 77% of animals after CPB, although arterial filtration was not used. The 25-μm arterial filter and bubble trap used in the present study failed to reduce retinal hypoperfusion after rewarming and the amount of ischemic retinal damage. These data indicate that the reductions in cerebral perfusion during CPB may be associated with damaging effects on other organs (ie, retina, cerebellum) as well. The type of oxygenator used in this study has been shown to release more gaseous microemboli than other bubble oxygenators and than membrane oxygenators [32]. Clinically, the use of membrane oxygenators has significantly reduced the incidence of retinal embolism [12]. Whether the alterations in cerebral blood flow and retinal histopathology can be attenuated by the type of oxygenator used during CPB warrants further study.

In conclusion, in a canine model of prolonged CPB, we found that the technique of $PaCO_2$ management during hypothermia had no effect on intracranial pressure, compliance, or cerebral perfusion. Intracranial pressure increased progressively during bypass but not to a critical level that compromised cerebral perfusion. Intracranial compliance and water content remained unchanged, which excludes cerebral edema formation by prolonged CPB as a cause of the increase in intracranial pressure. Evidence of ischemic degeneration in the bipolar cells of the retinal inner nuclear layer was found in 67% of animals, indicating that prolonged bypass may cause substantial cellular injury despite maintenance of systemic oxygenation and flow. Cerebral blood flow failed to increase after rewarming, a phenomenon that may be important in the pathogenesis of neurological dysfunction after CPB. The decrease in cerebral perfusion pressure secondary to intracranial hypertension does not account for the persistent cerebral hypoperfusion after rewarming. Rather, the increase in cerebral vascular resistance may be secondary to microembolization, which in turn causes direct cerebral endothelial damage.

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The Healing Sternum: A Comparison of Osseous Healing With Wire Versus Rigid Fixation

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Although median sternotomy is used for most cardiac procedures, postoperative dehiscence remains a serious and persistent problem. This investigation was designed to assess new bone formation and sternal healing across the linear osteotomy of the sternum and to determine if rigid fixation would enhance bony healing and thus decrease unfavorable sequelae. To test this hypothesis, 14 skeletally mature baboons (*Papio anubis*) underwent standard median sternotomy; seven sternotomies were closed with interrupted 24-gauge cerclage wires, and seven, with thin Vitallium compression miniplates and transverse lag screws. The sterna from each group were harvested en bloc at 4 and 8 weeks, radiographed, processed, and serially sectioned and stained for histomorphometry to assess the quantity of new bone across the linear osteotomy. Clinical stability was superior with the plated and lag screw group at 4 weeks; however, by 8 weeks, no clinical difference between treatments was apparent. Histomorphometric analysis indicated that the linear osteotomy gap treated with plates and screws was less than the gap associated with the wire group. Rigid fixation of the sternum resulted in earlier union with primary osseous healing, suggesting greater inherent stability. These factors may decrease adverse sequelae for this procedure.

(Ann Thorac Surg 1991;52:490–4)

Median sternotomy, with its broad exposure of the heart, great vessels, and mediastinum, is the incision of choice for most cardiac operations [1]. However, postoperative dehiscence continues to be a persistent and serious problem [2–4]. Failure of the sternotomy to heal can be related to many factors, including local infection, excessive motion, or bony instability [5]. One study documented increased dehiscence after the use of bilateral internal mammary artery (IMA) implants for coronary arterial bypass procedures, implicating local ischemia as the cause [6]. As a corollary, it has been shown that sternal blood flow falls more than 90% after IMA mobilization [7].

Despite the importance of this incision and its associated problems, the events leading to secure healing remain speculative, and little is known about the physiology of the wound itself. Specifically, there are few experimental data on the healing sternum or on the methods of stabilization related to bony union. The purpose of this investigation was to study sternal healing in an appropriate model after sternotomy and closure by rigid fixation versus wires.

Material and Methods

Fourteen skeletally mature baboons (*Papio anubis*) (mean weight, 19.6 kg) were selected for the study because their sternal size and anatomy were most analogous to humans, with the upper extremities of this model being non-weight bearing. All animals received humane care in compliance with the guidelines published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). The subjects were placed under general endotracheal anesthesia induced with thiopental sodium, maintained with isoflurane and nitrous oxide/oxygen, and monitored with arterial lines and continuous electrocardiography. A standard median sternotomy was performed on each subject. Seven animals underwent closure with interrupted cerclage 24-gauge stainless steel wires; an additional 7 subjects underwent closure with a single thin Vitallium compression miniplate for the manubrium and four Vitallium lag screws (oriented transversely across evenly spaced intervals) for the sternal body. At 4 and 8 weeks postoperatively, the sterna from each group were atraumatically harvested en bloc. The specimens were immediately radiographed, after which the specimens were then placed in 70% ethanol, transferred to absolute ethanol, and embedded in polymethylmethacrylate for undecalcified sectioning. The histological specimens were transversely sectioned across the manubrium and four consecutive segments of the sternal bodies. From each of these units, serial sections were cut to 4.5 μm in thickness and were stained using modified Masson-Goldner trichrome and von Kossa stains. Stained sections

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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were assessed for inflammation, and cellular and stromal elements were scrutinized.

Histomorphometry

A Cambridge 970 Image Analysis System interfaced with a Zeiss Axiophot microscope and Newvicon video camera were used to quantitate the amount of new bone formation across the linear osteotomies [8]. Each histological slide was evaluated at a ×6.25 magnification that enabled the examiner to assess four fields per histological slide. A standardized rectangular measuring frame of known area allowed the examiner to relate the acquired video image of the new bone across the osteotomy to the frame. The Image Analysis System was programmed to translate this video relationship into trabecular bony volume in units of cubic millimeters per cubic millimeter.

Statistics

Trabecular bone volumes were compared between treatments at different times and at the same time using a one-way analysis of variance computer model. A multiple comparison analysis (Scheffé *F* test) was used to determine if significant differences ($p < 0.05$) existed between each treatment at each time period.

Results

Plates, lag screws, and wires were removed at the time of harvesting of each group of sterna, and clinical assessment was made at the operated sites for either presence or absence of infection and instability across the linear osteotomy plane. All animals at both time periods were free from postoperative infection. In addition, all the rigidly fixed sterna were healed as demonstrated by a stable union across the osteotomy gap when the sites were manipulated after plate removal. In contrast, the 4-week wire group had unstable sterna with movement at the osteotomy site on manipulation. However, by 8 weeks, clinical manipulation did not cause motion across the sternotomies that had been wired. Histological assessment of the sterna at 4 and 8 weeks showed the presence of typical cellular elements and stromal tissue. The distinction between the wire and the plates and lag screws at 4 weeks was the amount of new bone seen histologically across the osteotomy gap (Fig 1). Typically, at this time period, woven bone was apparent in the rigidly fixed 4-week group, whereas the wire group displayed little or no new bone. However, by 8 weeks, the two treatments were virtually indistinguishable. Radiographic examination of the sterna corroborated the subjective histological appearance (Fig 2). Regardless of the treatment, there were no differences in the quantity of bone formation within the sternotomy wounds of the manubrium and the sternal body segments at 4 weeks. Histomorphometric quantitation of the amount of bone across the linear osteotomy wounds showed more bone across the manubrium of the wired group at 8 weeks than across the rigidly fixed group ($p < 0.02$) (Fig 3).

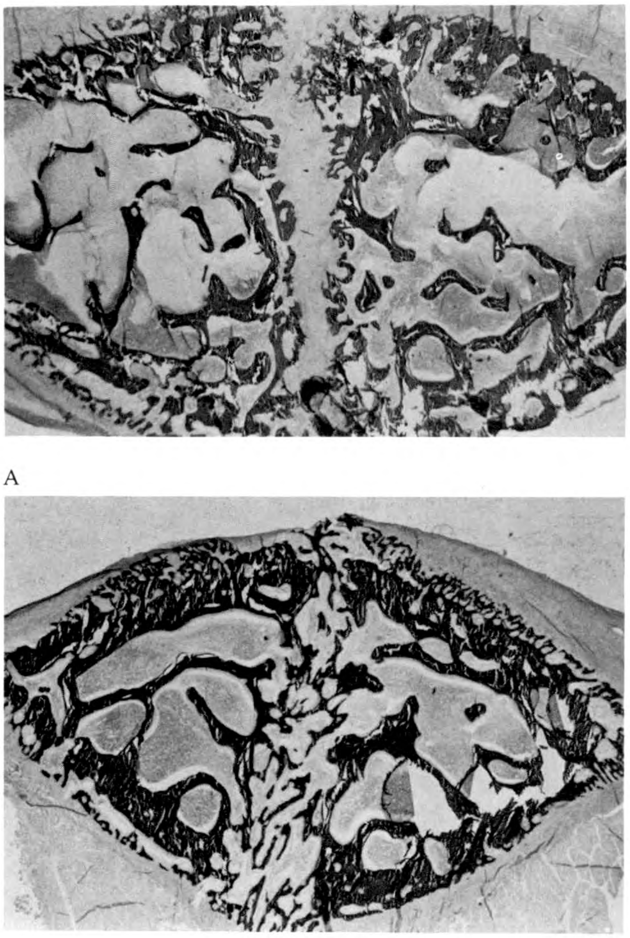

Fig 1. Healing manubrium at 4 weeks. (A) The gap across the cerclage wire manubrium shows little new bone formation. (B) In contrast, primary bone formed across the plated manubrium. (von Kossa stain, ×125 before 50% reduction.)

Comment

For more than 30 years, the median sternotomy, as described by Julian and colleagues [1], has been the preferred operative approach to the heart and mediastinum. The complications of this incision are often associated with prolonged ventilatory support, reoperation for bleeding, low-flow states, and disruptive forces such as external cardiac massage [5, 6]. Infection and dehiscence substantially increases the mortality as a result of this approach [9].

It is possible that the use of bilateral IMAs as coronary arterial bypass grafts also predisposes the wound to complications secondary to local ischemia [6]. Arnold's [10] classic studies showed that the primary blood supply to the sternum is from segmental branches of the IMA and that collaterals from other vessels are insignificant and nonexistent. Culliford and associates [6] noted a significantly increased rate of infection and dehiscence when both IMAs were harvested. However, other investigators were unable to confirm these findings [11, 12]. Our studies in primates have shown that although the median

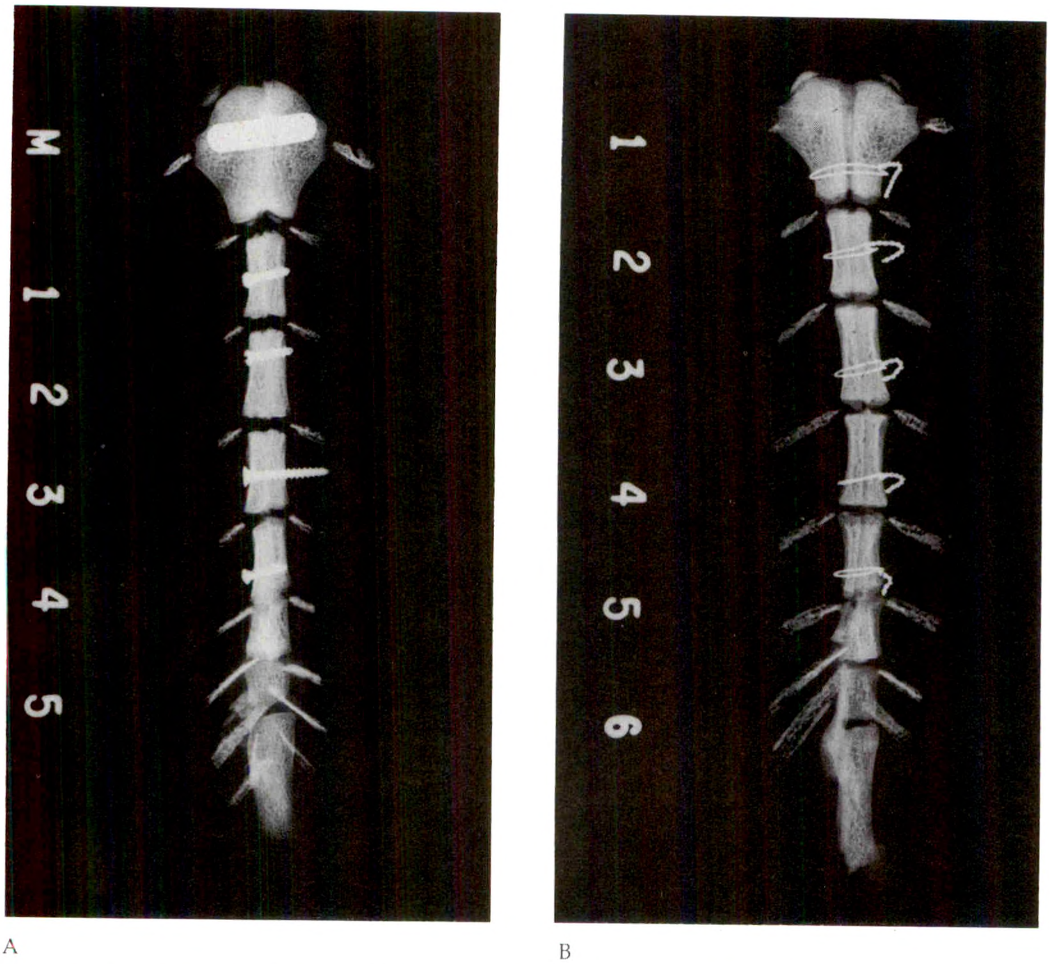

Fig 2. At 8 weeks postoperatively, the radiographs of the manubria and sternal bodies show radiolucent gaps in the wired group (B), whereas the plated and lag-screw group (A) demonstrated radiographic evidence of osseous healing.

sternotomy itself does not interfere with sternal circulation, there is a significant and immediate decrease in blood flow to the sternal half from which the IMA is mobilized [7].

Although many methods of sternal closure have been described, interrupted wire sutures remain the most commonly used technique [13]. Several variations of the wire technique have been proposed. Taber and Madaras [14] used figure-of-8 wire sutures that relied, not on the cortical bone of the sternum, but on the costal cartilages. Santos and colleagues [15] used threaded Kirschner wires, and Moghissi [16] used a series of tension sutures. Parasternal continuous sutures were proposed by Robicsek and co-workers [17], and a similar technique using absorbable sutures was described by Roy and associates [18]. Although these studies advocated the merits of achieving sternal stability, the techniques can be difficult and time consuming.

Through the years, there also have been efforts to develop a closure technique that would be more stable than the wire technique. Nylon bands were advocated by Le Veen and Piccone [19] but a subsequent study by Sanfelippo and Danielson [20] found this method to have an unacceptable complication rate. Kalush and Bonchek [21] used stainless steel bands, and Del Campo and Heimbecker [22] reported the successful use of metal plates in addition to parasternal wires. Several procedures have been reported that used rigid internal fixation and compression devices [23–25].

An important consideration in the design of an osteotomy procedure is the postoperative sequence of events that will produce bone healing. Stabilization of the osteotomized fragments by rigid fixation should ensure rapid bone union. New techniques have attempted to exploit the benefits of rigid fixation to produce sternal reapproximation [13–25]. Our study showed that at 4 weeks the miniplates and lag screws produced a more stable sternal wound than did cerclage wiring. At 4 weeks, there was a lack of histological and histomorphometric evidence to corroborate the clinical and radiographic findings. However, the typical difference between the two groups at 4 weeks was the presence of woven bone across the clinically healed osteotomy site in the rigidly fixed group with little or no new bone in the unstable wired group. At 8 weeks, the clinical determination showed no apparent differences in stability between plate-lag screw and wire

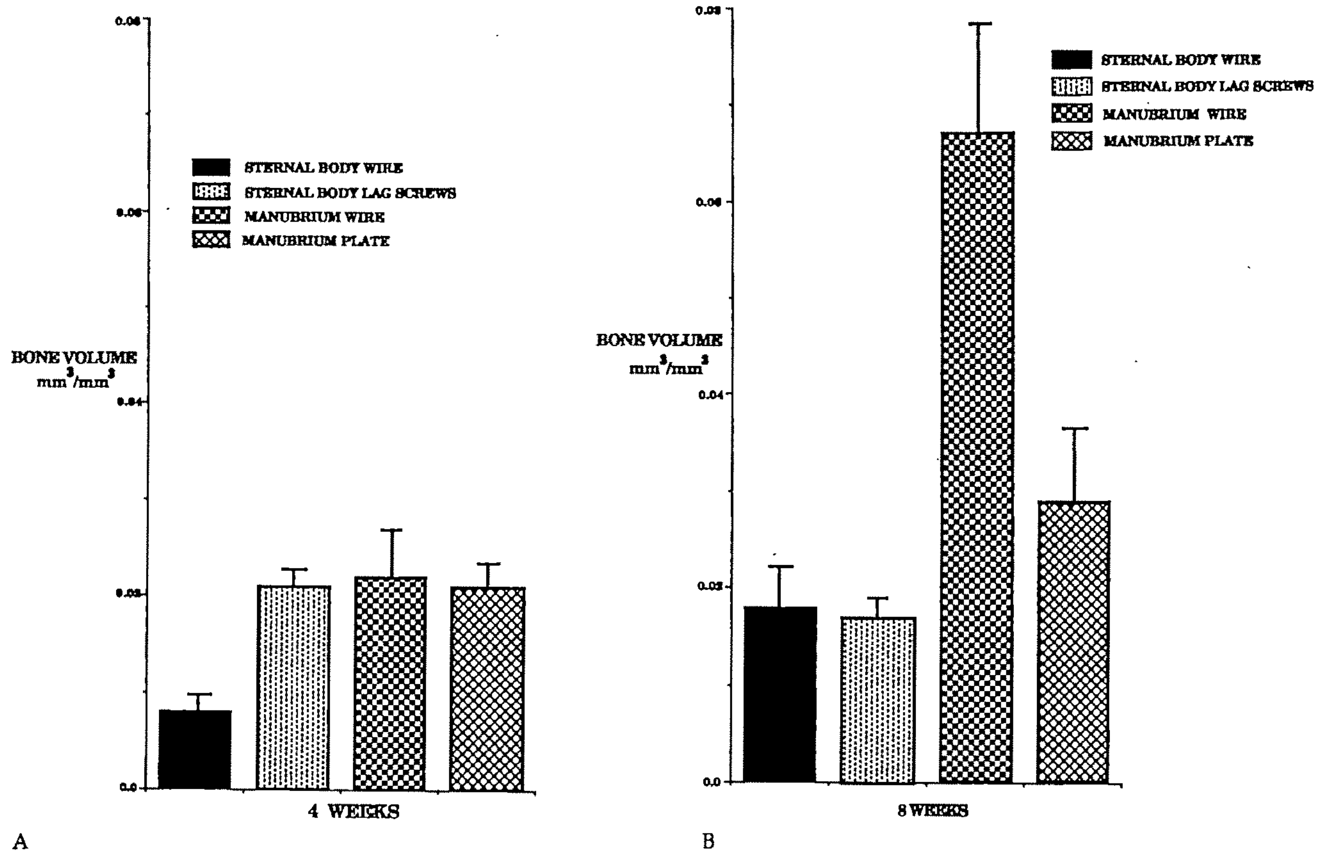

Fig 3. Bone volume at the sternotomy gap (A, 4 weeks; B, 8 weeks). Each bar *on the graph is the mean of 4 histological fields from four serially sectioned slides. There were 7 manubrium and sternal body specimens for each treatment; therefore, the data for each bar on the graph for the manubria are based on a mean of 112 fields. For the sternal body segments, the data for each bar are based on a mean of 448 fields.*

groups. Histomorphometry at 8 weeks showed that more bone had formed across the linear osteotomy gap of the manubrium treated with wire than of the manubrium treated with the miniplate. This seems logical as both were healed, and the gap across the wire-treated manubrium was greater than the gap across the miniplate manubrium; therefore, there was more room for bone to form.

The overall impression of healing across the linear osteotomy wound was that stability with bony healing was achieved earlier with miniplate and lag screw treatment than with cerclage wire. Moreover, the healing by primary bone formation without callus also typified the rigid-fixation treatment. In this regard, rigid fixation may be superior to wires owing to faster healing and earlier stability of the linear osteotomy treated with plates and screws.

Important clinical factors that must be considered in evaluating different sternotomy closures include ease of application, operative time, patient comfort, and removal of the fixation device if emergency reentry must be accomplished. As with many new techniques, there may be a reluctance to try this as it may seem too time-consuming or cumbersome. However, the miniplates and screws that provided rigid fixation in this study have been used extensively in the stabilization of facial osteotomies and fractures, providing distinct advantages over wire fixation [26]. Initially, operative time may be increased with the use of plates and screws. However, as the surgeon gains experience in rigid fixation, the time to properly place and secure the plates will approach that of wire fixation. Reentry can be accomplished in one of two ways. The screws can be removed with a screwdriver, or if more rapid entry is necessary, a small plate cutter can cut the hardware. In addition, postoperative patient comfort may be enhanced by rigid fixation owing to the immediate stabilization of an osteotomy subject to continuous movement.

In conclusion, plates and lag screws appear clinically to offer earlier bone healing and greater stability than cerclage wiring for fixing sternotomy wounds. However, by 8 weeks there does not seem to be a clinical difference between the two methods. Histomorphometrically, the quantity of bone formation across the rigidly fixed osteotomy is approximately the same as that of the wire group at 4 weeks. By 8 weeks, both of our groups demonstrated bony healing. The use of compression plates and screws produced an immediate rigid fixation of the median sternotomy with a compression osteosynthesis resulting in primary bone healing. Wiring resulted in an initially less stable form of fixation with a wider bony gap across the osteotomy. As would be expected, the wider bone gap yielded more new bone growth than the compressed osteotomy site of the miniplate and lag screw group.

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Effect of a Cyclic Adenosine Monophosphate Phosphodiesterase Inhibitor, DN-9693, on Myocardial Reperfusion Injury

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A new cyclic adenosine monophosphate phosphodiesterase inhibitor, DN-9693, was examined to see whether myocardial reperfusion injury could be reduced in a setting of cardioplegic arrest through its antiaggregation effect on leukocytes. Isolated rabbit heart models with whole blood perfusion were used, and 18 hearts were divided into three groups according to the reperfusion method: control (G-1, n = 5), DN-9693 (G-2, n = 7), and leukocyte depletion (G-3, n = 6). The hearts were subjected to 120 minutes of cold global ischemia under crystalloid cardioplegia followed by 30 minutes of reperfusion. A dose of 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of DN-9693 was administered in G-2, and a leukocyte removal filter was used in G-3 during reperfusion. Ultrastructural changes in mitochondrial injuries, intracellular edema, and capillary injuries of the myocardium showed worse changes in G-1 than in G-2 and G-3. Under microscopic study, the intracapillary leukocyte count was significantly higher in G-1 than in G-2 and G-3. Recovery of rate-pressure product, left ventricular developed pressure, and coronary flow were significantly better in G-2 and G-3 than in G-1. There were no significant differences between G-2 and G-3 for all these indices. These results indicate that reperfusion with leukocyte-depleted blood attenuates reperfusion myocardial injury and DN-9693 has a comparable myocardial protective effect with possible inhibition of leukocyte aggregation.

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Recently, considerable evidence has shown that leukocytes, especially neutrophils, are one of the causes of myocardial reperfusion injury (MRI), and their role has been described in such aspects as activation and capillary plugging [1–6]. From the standpoint of prevention of MRI, manipulations including depletion of leukocytes [7, 8] and pharmaceutical inhibition of leukocyte activity [9, 10] have been reported to attenuate MRI, considering the positive evidence of neutrophil involvement in MRI. In cardiac operations, this attenuation of leukocyte activity at reperfusion may be worthwhile in preventing MRI. Previous studies on the effects of neutrophil attenuation such as depletion have been mainly concerned with regional ischemia [11, 12], and the manipulation of neutrophils has been rarely investigated in global ischemic models except for a few reports [7, 13]. Recently, cyclic adenosine monophosphate (cAMP) phosphodiesterase inhibitor has been evaluated for antileukocyte function [14]. In this study, we evaluated the effect of a new cAMP phosphodiesterase inhibitor, DN-9693, on prevention of MRI associated with global ischemia.

Material and Methods

Eighteen white adult rabbits (2.5 to 3.0 kg) were anesthetized with sodium pentobarbital (15 to 20 mg/kg) and heparinized (300 IU/kg). The heart was isolated and perfused through the aorta under a modified Langendorff system. In this experimental circuit, the isolated heart as well as a supporting rabbit were perfused with diluted recirculating whole blood (hemoglobin level, >70 g/L [>7 g/dL]) withdrawn from 3 rabbits before each experiment (Fig 1). A membrane oxygenator (effective surface area, 0.3 m^2; Mera, Tokyo, Japan) and two roller pumps were used. All procedures from the isolation of the heart to perfusion were finished within 30 seconds. The perfusion pressure of the aorta was 100 cm H_2O.

After 15 minutes of normothermic perfusion (37°C), the control hemodynamic data were measured. Then, the aorta was clamped and 120 minutes of global ischemia was induced by infusing crystalloid cardioplegic solution (K^+, 18 mEq/L; Na^+, 8 mEq/L; glucose, 57 g/L; pH, 7.8; osmolarity, 380 mOsm/L) at 4°C with a initial dose of 10 mL/kg and 5 mL/kg every 30 minutes throughout the arrest period. The heart was immersed in cold (10° to 15°C) lactate Ringer's solution. Reperfusion (37°C) was performed for 30 minutes after ischemia.

The hearts were divided into three groups: control (G-1), DN-9693 (G-2), and leukocyte depletion (G-3). During the reperfusion period, in G-2 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of DN-9693 was infused into the perfused blood from 10

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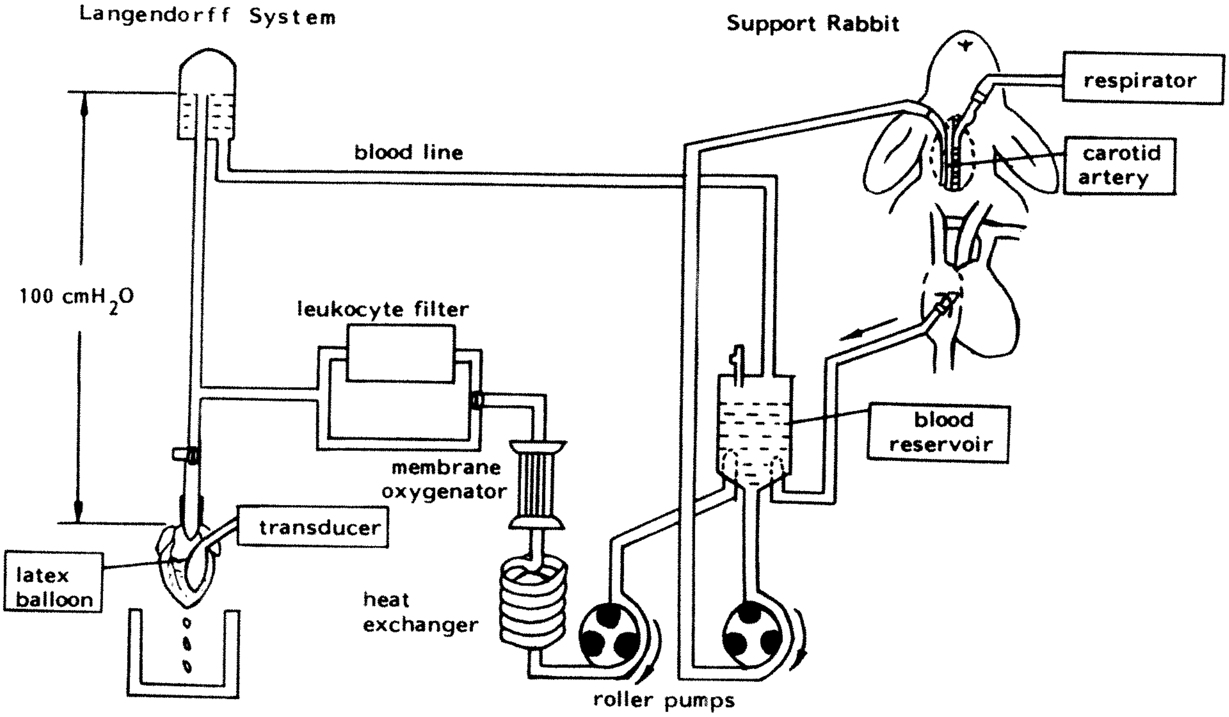

Fig 1. Scheme of experimental circuit.

minutes before reperfusion until the end of reperfusion, and in G-3 a leukocyte removal filter (Sepacell; Asahi Medical Co Ltd, Tokyo, Japan) was used. In the preliminary study, we confirmed that this filter trapped more than 95% of the leukocytes in rabbit blood. In addition, we have observed that this filter reduced platelets to the same degree.

Heart rate, coronary flow, left ventricular pressure, and its first derivative were measured. Left ventricular pressure was measured through a latex balloon placed in the left ventricle through the left atrium. The balloon was connected with a transducer to a blood pressure amplifier (AP-641G; Nihon Kohden, Tokyo, Japan). First derivative of left ventricular pressure was measured with a differentiator (ED-601G; Nihon Kohden). Left ventricular developed pressure was calculated as systolic pressure minus diastolic pressure. Rate-pressure product was calculated by multiplying the heart rate and the systolic pressure of the left ventricle. Coronary effluent was collected and malonyldialdehyde concentration was measured at the end of reperfusion.

Myocardial samples were taken at the end of the experiment. Myocardial water content was calculated from wet and dry heart weights with pieces of samples. These samples were fixed with 3% glutaraldehyde solution for 2 hours and were prepared for ultrastructural assessment. Semiquantitative assessments of ultrastructural changes were performed in a blinded fashion by a single observer with the method described previously [15, 16], namely, mitochondrial score, intracellular edema score, and capillary endothelial score. The leukocyte number of the capillaries was counted with light microscopic observation for the specimens fixed after the coronary arteries were flushed through the aorta under 100 cm H_2O pressure with lactate Ringer's solution for 15 minutes at the end of experiment. The leukocyte number of the capillaries was averaged in 20 fields of each micrograph (×200).

The Newman-Keuls test with adjustments for multiple comparisons was used to compare the data among groups. A p value less than 0.05 was considered statistically significant. All the data were described as mean ± standard deviation of the mean.

Results

Hemodynamic Results

There were no significant differences among the three groups in terms of preischemic values for all variables. The results at the end of reperfusion are shown in Table 1. The percentage of recoveries of rate-pressure product, left

Table 1. Hemodynamic Data at the End of Reperfusion

Index	G-1	G-2	G-3
%RPP	73 ± 28	127 ± 9^a	108 ± 36^a
%LVDP	74 ± 22	97 ± 28^a	101 ± 29^a
%+dp/dt	71 ± 29	124 ± 11^a	107 ± 32^a
%CF	88 ± 30	137 ± 28^a	125 ± 18^a

[a] $p < 0.05$ versus G-1. There were no significant differences between G-2 and G-3.

CF = coronary flow; +dp/dt = positive first derivative of left ventricular pressure; LVDP = left ventricular developed pressure; RPP = rate-pressure product.

Table 2. Ultrastructural Findings at the End of Reperfusion

Index	G-1	G-2	G-3
MtS	1.8 ± 0.3	0.8 ± 0.4[a]	0.4 ± 0.2[a]
EdS	1.1 ± 0.2	0.8 ± 0.3[a]	0.4 ± 0.1[a]
CeS	1.7 ± 0.4	0.8 ± 0.3[a]	0.5 ± 0.3[a]

[a] $p < 0.05$ versus G-1. There were no significant differences between G-2 and G-3.

CeS = capillary endothelial damage score; EdS = intracellular edema score; MtS = mitochondria score.

ventricular developed pressure, positive first derivative of left ventricular pressure, and coronary flow in G-2 and G-3 were significantly greater than those in G-1, whereas there were no significant differences between G-2 and G-3 for these variables.

Histological Results

The scores of mitochondrial score, intracellular edema score, and capillary endothelial score in G-2 and G-3 were significantly less than those in G-1 at the end of reperfusion. These indices did not show significant differences between G-2 and G-3 (Table 2, Fig 2). The leukocyte number of the capillaries was significantly greater in G-1 than that in G-2 and G-3 (G-1, 18 ± 7 counts/field; G-2, 3 ± 1 counts/field; G-3, 6 ± 5 counts/field; $p < 0.05$ for G-1 versus G-2 and G-3). The leukocyte number of the capillaries did not show significant differences between G-2 and G-3.

Water Content of Myocardium and Malonyldialdehyde in Coronary Effluent

The water content of myocardium at the end of reperfusion did not show a significant difference among the three groups (G-1, 83.1% ± 0.3%; G-2, 82.6% ± 2.4%; G-3, 81.0% ± 2.0%). The malonyldialdehyde content of the coronary effluent in G-2 (0.6 ± 0.3 nmol/mL) and G-3 (0.8 ± 0.2 nmol/mL) was relatively low compared with that in G-1 (2.2 ± 0.9 nmol/mL), but the differences were not significant.

Comment

This study demonstrated that reperfusion with leukocyte-depleted blood or with the addition of DN-9693 into the reperfusing blood was effective in attenuating MRI to a comparable degree after 120 minutes of cold cardioplegic global ischemia. The effects were confirmed in terms of the functional recovery of the heart, the attenuation of the no-reflow phenomenon suggested by coronary flow measurement, and the reduction of myocardial injury during reperfusion. These results also confirmed that leukocytes had a causative role in the development of MRI.

In this study, it was hypothesized that the depletion of leukocytes or the inhibition of leukocyte activity during reperfusion might influence the production of oxygen free radicals. We measured the blood level of malonyldialdehyde, which is a by-product of free radical–mediated lipid peroxidation, in coronary effluent. However, malonyldialdehyde levels did not show a significant change in the treated group in spite of significant differences in morphological and hemodynamic changes. Although malonyldialdehyde has potential limitations in indicating the presence of oxygen free radicals, it may be suggested that leukocyte-derived oxygen free radicals have less involvement in the setting of MRI. On the other hand, it is conceivable that the rabbit heart does not have a detectable concentration of either xanthine oxidase or xanthine dehydrogenase, which are known to be important enzymes in producing free radicals. This issue about the role

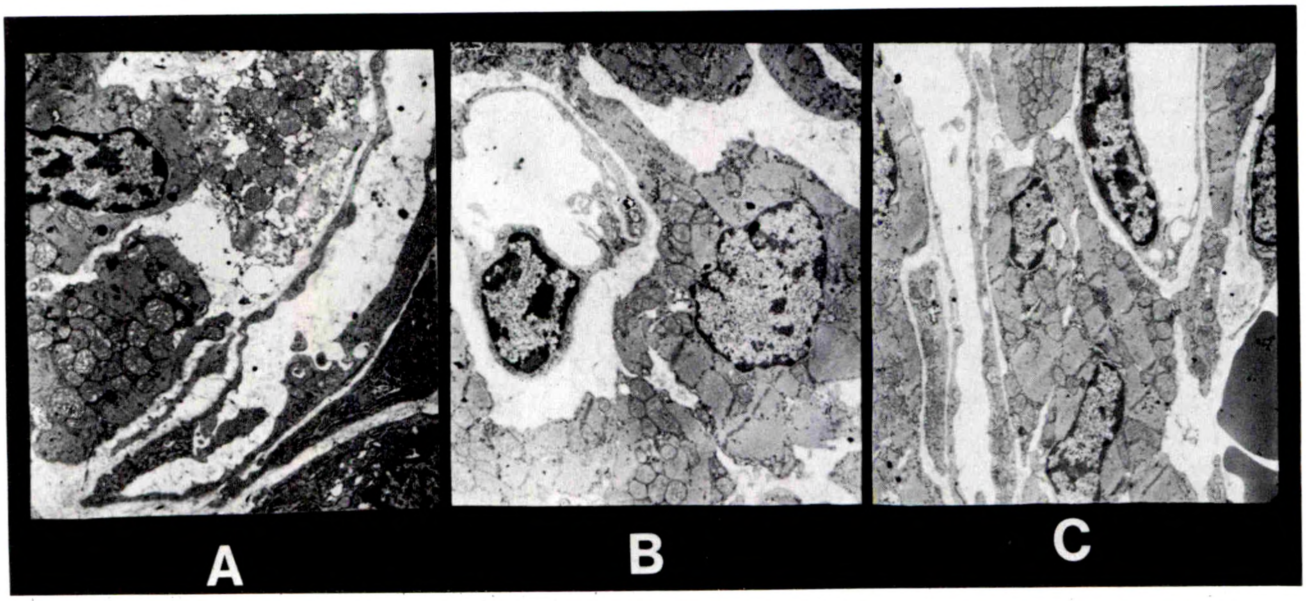

Fig 2. Representative photomicrographs of tissue samples: (A) G-1 (control group), (B) G-2 (DN-9693 group), (C) G-3 (leukocyte-depleted group).

of oxygen free radicals in MRI requires further studies with a reliable method of detection.

Agents that can increase cAMP levels in the leukocytes either by stimulation of adenylate cyclase or by the inhibition of phosphodiesterase are known to inactivate leukocytes [17]. A new cAMP phosphodiesterase inhibitor, DN-9693, has antileukocyte function that is mediated by increased cellular cAMP level [14]. In addition, we previously reported that this agent prevented leukocyte sequestration induced by extracorporeal circulation [18]. The present study showed that DN-9693 had a myocardial protective effect that was at least as good as that of leukocyte depletion. The antileukocyte function of DN-9693 was also confirmed by the finding that the leukocyte number of the capillaries in the DN-9693 group was significantly less than that in the control group, suggesting that DN-9693 can protect myocardium from MRI by inhibiting leukocyte activities with less aggregation and plugging of the capillaries.

Previous reports [5, 6] have shown that capillary plugging by leukocytes appeared to be a mechanism involved with the no-reflow phenomenon. In our global ischemic model of the isolated rabbit heart, the recovery of coronary blood flow after 120 minutes of cardioplegic arrest was significantly less in the control group than in the leukocyte-depleted group and the DN-9693 group. Concomitantly, light microscopy showed a reduced number of leukocytes in the capillaries in the leukocyte-depleted group and the DN-9693 group. Although coronary blood flow is affected by perfusion pressure in general, isolated hearts in our experiment were perfused at constant pressure. So, the findings observed in this study suggest that leukocytes were entrapped in the capillaries with possible effects on the no-reflow phenomenon, which is defined as the reduction in coronary flow at reperfusion. The DN-9693 group showed apparent prevention of this leukocyte trapping, possibly because of its antiaggregatory effect.

Another mechanism or DN-9693 to protect myocardium may be its potential property of inhibiting platelet aggregation [14, 19, 20]. Considering the involvement of platelets in this study, we assumed that the possible inhibition of platelets by DN-9693 might not be as important as inhibition of leukocytes in protection from MRI. This was also suggested by an early report that depletion of leukocytes with antiserum suppressed platelet accumulation into the myocardium, suggesting that the activation of platelets was secondary to a leukocyte-mediated event [21]. However, leukocyte depletion in this study also accompanies platelet depletion because the filter we used traps both, and a further study may be required to settle this issue.

Cyclic nucleotide phosphodiesterase inhibitors have some systemic effects such as reducing mean blood pressure and increasing arterial blood flow. A selective cAMP phosphodiesterase inhibitor, DN-9693, has more hemodynamic effects than a nonselective one such as isobutylmethylxanthine in renal and coronary circulation [22]. Regarding cardiac function, DN-9693 has a positive inotropic effect because it can also increase the cAMP level in myocytes [22]. In the preliminary study, a single bolus of 20 μg/kg did not result in significant inotropic effects in canine hearts. We used continuous administration of 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and it showed some tendency to increase heart rate. Therefore, it should be taken into account that the results regarding cardiac function were influenced by the drug itself, although we believe this may be less significant. In addition, this effect may not have direct effects on the other findings such as those of the ultrastructural study.

The results of this study have two clinical implications. First is the possibility of applying leukocyte-depleted blood reperfusion or DN-9693 at reperfusion to prevent MRI after cardioplegic arrest when it is prolonged or inadequate. Warm induction or hot shot with hyperkalemic reperfusion has been used as one of the strategies in cardiac operations, and incorporation of a leukocyte filter can be easily applied. The administration of DN-9693 at reperfusion can be made either locally to the heart or by systemic infusion. In the former method, one can select between the two methods of applying the leukocyte filter or administering DN-9693 through the side line for warm reperfusion. The second implication from these results is the significant involvement of leukocytes in MRI. In the basic and clinical studies, the effects of leukocytes in reperfusion injury should be further investigated.

In conclusion, the administration of DN-9693 during reperfusion significantly reduced the reperfusion injury and improved the recovery of heart function to a degree comparable with that of reperfusion with leukocyte-depleted blood after 120 minutes of cardioplegic arrest. The main mechanism of DN-9693 to prevent myocardial reperfusion injury may be related to the inhibition of leukocyte aggregation in microcirculation.

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Conventional Blood Conservation Techniques in 500 Consecutive Coronary Artery Bypass Operations

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With use of a nonpharmacological, simple, and inexpensive program for blood conservation, 500 consecutive patients underwent elective coronary artery bypass grafting without need of homologous red cell transfusions in 493 (98.6%). At least one internal mammary artery was grafted in all but 1 patient, with supplemental saphenous vein grafts. Intraoperatively, autologous heparinized blood was removed before bypass and retransfused at the conclusion of extracorporeal circulation. The volume remaining in the oxygenator and tubing set was returned without cell processing or hemofiltration. Using the hard-shell cardiotomy reservoir from the heart-lung machine, autotransfusion of the shed mediastinal blood was continued hourly up to 18 hours after operation. The mean postoperative mediastinal blood loss was 643 ± 354 mL, whereas 624 ± 296 mL was autotransfused. Thirteen patients (2.6%) needed reexploration for bleeding, of whom 7 (7/500, 1.4%) received homologous blood. No other patients required red cell transfusions. In addition, 9 patients were given a mean of 2.6 units of fresh frozen plasma because of suspected coagulopathy. No platelets were transfused, and no cryoprecipitate therapy was undertaken. Thus, in total, 484 patients (96.8%) were not exposed to any homologous blood products during the hospital stay. At discharge, the mean hemoglobin concentration was 121 ± 14 g/L (12.1 ± 1.4 g/dL) and the hematocrit, 0.36 ± 0.04. Postoperative complications were few. There was one in-hospital death (0.2%).

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Restriction of homologous blood transfusion in open heart operations should decrease the risk of infective contamination and antigenicity. Several blood conservation practices have evolved, with major accomplishments achieved from predonation of autologous blood [1–3], removal of autologous blood before bypass with isovolumetric substitution [4–7], reinfusion of the volume remaining in the extracorporeal circuit [8–10], and autotransfusion of the shed mediastinal drainage blood [7, 8, 11–13]. Other techniques using various devices for intraoperative hemofiltration [10, 14] and hemoconcentration with cell separators [7, 10, 15, 16] have been shown to decrease homologous blood usage in cardiac operations. Even processing of the shed mediastinal blood before reinfusion has been reported to have favorable blood-saving effects [17]. More recently, drugs for reducing postoperative blood loss have gained popularity [18–21]. In most of the reported series using one technique or different combinations of blood conservation techniques, however, the homologous blood usage was still considerable [3, 6, 16, 17, 22], even for elective coronary artery bypass grafting [13, 14, 23].

The present study reports the effects of a simple blood conservation protocol designed without the practical and financial inconvenience of special devices or equipment for blood cell processing. The program is easily applicable in routine cardiac procedures and aims to eliminate the need of homologous blood transfusions while maintaining adequate hemoglobin concentration.

Material and Methods

Between February 1989 and August 1990, 500 consecutive patients underwent primary elective myocardial revascularization. This series represents the initial patient material from our department, which is a newly formed unit for elective coronary artery bypass grafting procedures. Two thirds of the patients were treated by the same team (all of us). There were 436 men and 64 women with a mean age of 58.0 (± 8.1) years, with 120 (24.0%) 65 years old or older. The demographic data for the patients are presented in Table 1. Most of the patients had normal left ventricular function. However, 98 patients (19.6%) had an ejection fraction less than 0.50 or a left ventricular end-diastolic pressure greater than 24 mm Hg. The patients were operated on consecutively, and no patients were excluded from the study.

The protocol for blood conservation in elective coronary artery bypass procedures is as follows:

1. Discontinuation of antiplatelet drugs 7 days before operation

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Table 1. Preoperative Data on 500 Patients Undergoing Elective Coronary Artery Bypass Grafting[a]

Variable	Value
Female/male	64/436
Age (y)	58.0 ± 8.1 (33–75)
Patients ≥65 years old	120 (24%)
Previous myocardial infarction	293 (58.6%)
Left main stenosis	55 (11%)
Ejection fraction	0.67 ± 0.12 (0.30–0.91)
Ejection fraction < 0.50	64 (12.8%)
Left ventricular end-diastolic pressure (mm Hg)	15.2 ± 5.9 (2–36)
Relative heart volume (mL)	453 ± 74 (270–770)

[a] Where applicable, data are shown as the mean ± the standard deviation with the range in parentheses.

2. Removal of autologous blood before bypass for retransfusion after bypass in patients with normal preoperative red cell volume
3. Intraoperative retransfusion of oxygenator and tubing contents
4. Adequate rewarming of patient (rectal temperature, 36°C) and control of systemic blood pressure
5. Postoperative autotransfusion of shed mediastinal blood up to 18 hours postoperatively.

Premedication consisted of diazepam (0.1 to 0.2 mg/kg) and morphine-scopolamine (0.1 to 0.2 mg/kg). The general anesthesia protocol was designed to permit early extubation and was mainly a combination of diazepam (0 to 0.2 mg/kg), midazolam hydrochloride (0.1 to 0.2 mg/kg), fentanyl (8 to 10 μg/kg), and pancuronium bromide supplemented with isoflurane and nitrous oxide.

Extracorporeal circulation was performed with a bubble oxygenator (BEN 10 Plus; American Bentley Laboratories, Irvine, CA) in 335 patients and a membrane oxygenator (Bentley BCM-7) in 165. A membrane oxygenator was preferred when the extracorporeal bypass time was expected to exceed 90 minutes, ie, in operations on elderly patients, patients with impaired left ventricular function, patients requiring multiple grafts, and patients requiring endarterectomy. The heart-lung machine was primed with 2,000 to 2,500 mL of a crystalloid solution (Ringer's acetate). Anticoagulation was established with heparin sodium to achieve an activated clotting time of more than 480 seconds. This level was maintained throughout the period of cardiopulmonary bypass, with supplementary heparin as indicated by repeated determination of the activated clotting time.

Autologous blood was withdrawn from the patient after heparinization and insertion of the cannulas. From the two-stage venous cannula, blood was drained by gravity into blood bags and stored at room temperature for retransfusion at the conclusion of cardiopulmonary bypass. If hypotension occurred, the blood volume removed was replaced with Ringer's acetate from the pump through the aortic cannula. The hematocrit during bypass should exceed 0.20 to 0.22, and patients with a red cell volume (blood volume multiplied by preoperative hematocrit) less than 1,600 mL had no blood withdrawn [12]. Low ejection fraction or left main stenosis was not a criterion for exclusion from preoperative blood removal. Pulsatile flow at a rate of 2.4 $L \cdot m^{-2} \cdot min^{-1}$ and moderate hypothermia (blood temperature, 28° to 30°C) were used.

At least one internal mammary artery was grafted in all but 1 patient, supplemented with saphenous vein grafts. The left pleura was opened routinely. The aorta was cross-clamped during performance of the distal anastomoses, and myocardial preservation consisted of crystalloid cardioplegia (St. Thomas' II) and local cooling with ice slush. The proximal anastomoses were sutured during partial occlusion of the ascending aorta. The patients were rewarmed to a rectal temperature exceeding 36°C. The cardiotomy sucker was used throughout the period of heparinization, and surgical hemostasis was completed before the infusion of protamine sulfate. At the end of bypass, anticoagulation was reversed with protamine chloride at a ratio of 1.3 mg:1 mg of heparin to obtain an activated clotting time of less than 130 seconds.

The blood remaining in the oxygenator and the tubing set was drained into blood bags and retransfused in the operating room or in the intensive care unit or both places. No cell separation or hemofiltration was undertaken.

Attention was directed to surgical hemostasis, both in the chest and in the legs after vein harvesting. The leg wound was closed as soon as possible, without drainage, and wrapped with a compressive bandage. Before closure of the sternum, mediastinal and pleural tubes were placed and connected to the hard-shell cardiotomy reservoir (Bentley BCR 3538) used during cardiopulmonary bypass. The reservoir was taken to the intensive care unit, and a vacuum of 15 cm H_2O was applied. The shed mediastinal blood was retransfused hourly in a closed system [24] until the tubes were removed or up to 18 hours postoperatively.

At repeat sternotomy for postoperative bleeding, a Sorenson drainage system (Sorenson Research, Salt Lake City, UT) for suction in the surgical field and autotransfusion was available. In general, a hematocrit of 0.25 or less was the main guideline for giving homologous blood after the operation.

Hemoglobin concentration and hematocrit were recorded preoperatively, during extracorporeal circulation, 3 hours postoperatively, on postoperative day 1, and at discharge on day 7 to 10.

Postoperative hypertension was managed with intravenous administration of nitroglycerin or sodium nitroprusside, with the aim of a mean systemic blood pressure of less than 90 to 100 mm Hg in the first hours after operation. Eventual hypovolemia was treated with Ringer's acetate. In patients with unimpaired kidney function, diuretics were restrictively used to avoid homeostatic imbalance.

Table 2. Results of Blood Conservation Procedures in 500 Patients[a]

Variable	Value
Autologous blood removed before bypass (mL) (n = 445)	799 ± 314 (300–1,570)
Bleeding postoperatively (mL)	643 ± 354 (165–5,230)
Autotransfusion postoperatively (mL)	624 ± 296 (80–2,760)
Reexploration for bleeding	13 (2.6%)
Homologous blood transfusion, 1 to 9 units	7 (1.4%)
Fresh frozen plasma, 1 to 4 units	9 (1.8%)
Patients not exposed to any homologous blood products	484 (96.8%)

[a] Where applicable, data are shown as the mean ± the standard deviation with the range in parentheses.

Table 4. Operative and Postoperative Results in the 500 Patients[a]

Variable	Value
Internal mammary artery graft	499 (99.8%)
Endarterectomy	62 (12.4%)
No. of distal anastomoses	3.6 ± 1.6 (1–8)
Aortic cross-clamp time (min)	33.8 ± 13.7 (6–105)
Extracorporeal bypass time (min)	68.1 ± 20.6 (16–148)
Time on ventilator postop (h)	1.4 ± 1.1 (0–120)
Inotropic support	8 (1.6%)
Walking stairs/outdoors before postop day 4	412 (82.4%)
Walking stairs/outdoors before postop day 7	492 (98.4%)

[a] Where applicable, data are shown as the mean ± the standard deviation with the range in parentheses.

Results

Data relevant to the blood conservation program are summarized in Tables 2 and 3. In 445 patients (89%) an average of 799 ± 314 mL of autologous blood was withdrawn before bypass and retransfused after bypass. As the blood was removed after cannulation, infusion of the prime solution from the pump could prevent any situation of hypotension, and in fact, no complications during blood withdrawal were observed. No blood was withdrawn in 55 patients, as the red cell volume was less than 1,600 mL as a result of small body size, preoperative anemia, or both. The mean bleeding from the chest tubes postoperatively was 643 ± 354 mL (median amount, 587 mL), and 97% of the shed mediastinal blood was autotransfused. The amount of bleeding was monitored from closure of the sternal wound until the chest tubes were removed.

Thirteen patients (2.6%) needed repeat sternotomy for bleeding. During reoperation, 500 to 4,200 mL (mean amount, 1,636 ± 253 mL) was autotransfused, using both the hard-shell reservoir and the Sorenson system. Seven of these patients were given homologous bank blood. These were the only patients in the entire group who received homologous red cells. An additional 9 patients were given 250 to 1,000 mL of fresh frozen plasma because of suspected coagulopathy, which was mostly due to ingestion of anticoagulation drugs. Thus, in total, 484 patients (96.8%) were *not* exposed to any homologous blood products during the hospital stay.

Table 3. Perioperative Hematocrit and Hemoglobin Concentration[a]

Time Measured	Hemoglobin (g/L)	Hematocrit
Before operation	146 ± 12 (102–184)	0.44 ± 0.04 (0.30–0.54)
Minimum on bypass	. . .	0.23 ± 0.03 (0.15–0.31)
After operation		
3 Hours	124 ± 13 (88–162)	0.37 ± 0.04 (0.27–0.49)
18 Hours	128 ± 15 (78–166)	0.38 ± 0.05 (0.27–0.49)
At discharge (day 7 to 10)	121 ± 14 (77–159)	0.36 ± 0.04 (0.24–0.50)

[a] Data are shown as the mean ± the standard deviation with the range in parentheses.

In 8 patients the lowest hematocrit registered during cardiopulmonary bypass was 0.15 to 0.17, a result chiefly of infusion of an unexpectedly large amount of cardioplegic solution. No red cell transfusions were given, and the mean hemoglobin concentration postoperatively of 110 ± 11 g/L (11.0 ± 1.1 g/dL) was slightly less than average. For all patients, the mean hemoglobin concentration and hematocrit at discharge were 121 ± 14 g/L (12.1 ± 1.4 g/dL) and 0.36 ± 0.04, respectively.

The clinical results are summarized in Table 4. The patients were extubated a mean of 1.4 ± 1.1 hours after arrival in the intensive care unit. Eight patients needed inotropic support for more than 30 minutes (including intraaortic balloon pumping in 2) during the first postoperative day. The patients were energetically rehabilitated so as to regain optimal physical capacity, and within 1 week, 98.4% were able to move freely in the hospital area.

Postoperative complications included pulmonary embolism (1 patient, 0.2%), mediastinitis (1 patient), and wound infection (two sternal, four leg) (6 patients, 1.2%). Eighteen patients (3.6%) sustained perioperative myocardial necrosis as judged by new Q waves on the electrocardiogram and elevation of the enzyme levels. Most patients were asymptomatic. Five patients (1.0%) had transient neurological symptoms. One patient, a 70-year-old woman with unstable angina and left main stenosis, died of multiple-organ failure on postoperative day 4.

Comment

Despite a widespread interest in reducing blood use in cardiac procedures [2–4, 6, 7, 9–16, 25–28], the homologous blood requirement remains considerable [3, 6, 16, 17, 22], even in elective myocardial revascularization [7, 13, 23]. In 1988, Russell and colleagues [22] reported a mean homologous blood usage of 5.07 units per operation at 24 cardiac surgical centers in the United Kingdom. The risks associated with transfusions of homologous blood and blood products [29] prompted us to design a blood conservation program that should be optimal in performance and financial costs, be safe, and aim at a total avoidance of homologous transfusions. The protocol should be easy to apply in daily routine cardiac operations. Stimulated by recent reports on current blood conservation techniques [27] and suggestions of guidelines for transfusion support in coronary artery bypass operations [28], we reviewed our initial experiences.

The most important steps contributing to a decrease in blood usage during cardiac operations take place intraoperatively. Careful surgical techniques must include meticulous hemostasis and avoidance of washout or suction of red cells from the surgical field. Preferably the cardiotomy sucker is used during the entire period of heparinization.

Removal of autologous blood before bypass provides the immediate postoperative availability of fresh blood with nontraumatized platelets. Since 1972 when Hallowell and co-workers [5] reported the first series of fresh autologous blood transfusion in open heart surgery, this technique has significantly reduced postoperative bleeding in several studies [6–9, 12, 25]. Kaplan and associates [25] evaluated three different methods of removing autologous blood before initiation of cardiopulmonary bypass, and the best results concerning blood-saving effect and platelet conservation were obtained by removing heparinized blood from the venous cannula immediately before extracorporeal circulation. In our patients with a red cell volume exceeding 1,600 mL [12], a mean amount of 799 ± 314 mL of autologous blood was withdrawn, and in most instances, the volume was replaced by Ringer's acetate from the pump, thus avoiding hypotension and potential myocardial ischemia. The lowest hematocrit recorded during cardiopulmonary bypass was a mean of 0.23 ± 0.03. After reversal of heparin and optimal surgical hemostasis, the autologous blood was returned to the patient.

In a small group of 8 patients, hematocrit decreased to 0.15 to 0.17 during cardiopulmonary bypass, but this was well tolerated without homologous red cell transfusions. Postoperative autotransfusion and spontaneous hemoconcentration resulted in restoration of the hemoglobin concentration. Patients with a low ejection fraction or left main stenosis were not excluded from autologous blood removal, and the procedure was undertaken without any signs of myocardial decompensation.

Autologous predonation weeks before the surgical procedure has been reported to reduce the requirement for homologous blood [1–3]. However, this technique carries some risks, especially in patients with coronary artery disease, and Britton and colleagues [2] reported that 12% of 104 patients had to discontinue the donation because of increasing angina. In addition, autologous blood is more expensive to collect than volunteer homologous blood because of the additional nursing care and administrative coordination required [2].

Transfusion of the blood remaining in the oxygenator at the conclusion of bypass has proved to be a safe and reliable technique to save blood [7–10]. Additional refinement of this method using the Cell Saver (Haemonetics Corporation, Braintree, MA) [7, 10, 16] or hemofiltration [10, 14] to increase the hematocrit of the residual volume in the oxygenator has proved effective. However, the costs are not negligible, and the processing of the blood is time-consuming [10]. In the present series, the diluted blood from the oxygenator was retransfused to all patients either in the operating room or in the intensive care unit. Despite the absence of hemoconcentration or ultrafiltration, there were no clinical signs of volume overloading, and the patients could be extubated with adequate blood gases early in the postoperative period (mean time, 1.4 ± 1.1 hours after arrival in the intensive care unit).

Autotransfusion of shed mediastinal blood has become widely popular since the 1978 report of Schaff and associates [11]. They demonstrated a 50% reduction in blood use, and this has been confirmed by other investigators [7, 8, 12, 13]. The clinical safety of this technique has been proved by the lack of septic, pulmonary, or hepatic complications [8]. Like others [9, 13], we routinely use a hard-shell cardiotomy reservoir for retransfusion in a closed-circuit system, and we were able to save as much as 97% of the shed blood.

Griffith and co-workers [17] found that reinfusion of shed mediastinal blood was associated with laboratory evidence of disseminated intravascular coagulation. They were using the Sorenson drainage system, and they recommended routine washing of the infusate. Their findings are not supported by Hartz and colleagues [26], who compared two types of hard-shell reservoirs and concluded that the mediastinal blood could safely be infused without inducing fibrinolysis or disseminated intravascular coagulation and that the use of the cardiotomy reservoir was a safe and inexpensive method for autotransfusion after cardiac operations. Furthermore, a simple postoperative autotransfusion system provides an element of safety in situations of sudden bleeding, as hemodynamic stability can be maintained by continuous reinfusion of the shed blood [12].

In 1 patient in the present study, as much as 2,000 mL was autotransfused from the reservoir during preparation for reexploration. Excessive bleeding from a vein graft side branch was present, but there was no hemodynamic instability during the entire procedure. Nine patients (1.8%) received 1 to 4 units of fresh frozen plasma for an increased bleeding tendency. However, the effect of fresh frozen plasma on postoperative bleeding has been disputed [9], and the indication for this blood component should possibly be even more restrictive.

Repeat sternotomy for postoperative bleeding is a significant predictor of donor blood transfusion [12], and all efforts should be directed to careful operative hemostasis. The only patients in the present series who received homologous blood were in the group of 13 who underwent reoperation for surgical bleeding.

During postoperative care, hemodynamic stability without hypertensive crisis contributes to reduce mediastinal bleeding. We have found that having rewarmed the patient to a rectal temperature exceeding 36°C makes it easier to maintain hemodynamic control. Hypertension is otherwise treated with nitroglycerin or nitroprusside to keep the mean arterial pressure less than 90 to 100 mm Hg during the first postoperative hours.

Drugs to reduce perioperative blood loss in open heart operations have been introduced chiefly during the last decade [11–21, 30]. Desmopressin acetate has been shown to decrease blood loss and transfusion requirements in patients undergoing complex cardiac operations [20], but no beneficial effects were seen in routine primary coronary artery bypass operations [31]. Prophylactic treatment with ϵ-aminocaproic acid, a synthetic antifibrinolytic agent, resulted in less postoperative bleeding and reduction in homologous blood transfusions from 4.2 ± 2.3 to 2.8 ± 2.0 units [19]. Similar results were demonstrated using tranexamic acid, another antifibrinolytic drug [21]. However, the potential adverse effects on coagulation and risks of thrombosis using agents inhibiting fibrinolysis have not yet been clarified.

Reductions in postoperative bleeding and homologous blood usage have also been achieved in patients receiving high doses of the protease inhibitor aprotinin [18, 30]. It has been documented that kallikrein and plasmin are effectively inhibited [30], and the adhesive capacity of the platelets is preserved [32]. Clinically, the most pronounced effect on postoperative bleeding and blood usage has been seen in patients at high risk for bleeding complication, that is, patients having a redo operation and patients with endocarditis [18]. As high doses of aprotinin represent a substantial cost, it should possibly be restricted to these patients. In the present series, our combination of conventional blood conservation techniques excluded demands for pharmacological intervention to reduce blood loss. In fact, no patient needed donor blood transfusions except in the subset of patients undergoing repeat sternotomy for surgical bleeding.

In conclusion, a simple and inexpensive blood conservation program, mainly combining autologous blood removal before bypass, retransfusion of the volume remaining in the oxygenator, and consistent autotransfusion of mediastinal shed blood, has enabled us to avoid infusion of homologous red cells in 98.6% of patients. A total of 484 patients (96.8%) were not exposed to any donor blood products. The present combination of blood conservation techniques does not carry any extra costs except for blood bags and infusion lines at a total price not exceeding $10.00. The patients were discharged with a mean hemoglobin level of 121 ± 14 g/L (12.1 ± 1.4 g/dL), and the clinical results were favorable. No side effects or complications could be related to the blood conservation program.

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Treatment of Cardiogenic Shock With the Hemopump Left Ventricular Assist Device

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A multiinstitutional study is in progress to evaluate the Hemopump in the treatment of cardiogenic shock. Fifty-three patients with refractory cardiogenic shock were selected for Hemopump assistance. The hemodynamic definition of cardiogenic shock included (1) a cardiac index of less than 2.0 L $\cdot$ min^{-1} $\cdot$ m^{-2}, (2) pulmonary capillary wedge pressure of greater than 18 mm Hg, and (3) a systolic blood pressure of less than 90 mm Hg or a left ventricular work index of less than 1,500 g-m $\cdot$ m^{-2} $\cdot$ min^{-1}. The Hemopump was successfully inserted in 41 of 53 patients (77.3%). A significant improvement in the hemodynamic status was seen during Hemopump assistance. A minimal level of hemolysis was observed. No leg ischemia was observed. The 30-day overall survival of the Hemopump group was 31.7%. Criteria establishing indications for use and clinical utility are proposed. We conclude that the Hemopump provides significant hemodynamic support of the patient in cardiogenic shock allowing for recovery from ventricular stunning in marginal ventricles, and that in select patients the Hemopump may offer a major improvement in survival over conventional therapy.

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Cardiogenic shock is estimated to occur 150,000 times a year in the United States. Current patient management, including pharmacologic therapy, intraaortic balloon pump (IABP) counterpulsation, and emergent revascularization, has done little to change the natural course of this life-threatening condition. At present, the mortality of cardiogenic shock is still reported to be 80% to 90% [1–4]. However, experimental mechanical circulatory assist devices offer hope of improving survival [5–9]. The theoretical basis for the use of temporary mechanical support in the treatment of cardiogenic shock is to reduce the work of noncontractile but viable or "stunned" myocardium allowing time for contractility to return [10–12]. Mechanical circulatory assist devices include the IABP and a variety of experimental left ventricular assist devices (LVADs).

The IABP is widely used in the United States because it is readily implemented and its use is associated with relatively low risk. However, the IABP has a very limited ability to improve the hemodynamic status of a patient and requires some left ventricular contractility to operate effectively. Although the IABP is useful in treating patients with a moderate severity of shock, the majority of patients in severe cardiogenic shock still die in spite of IABP therapy.

A variety of LVADs have been applied on a limited basis to the treatment of postcardiotomy shock patients. Most LVADs have the capacity to support the majority (80% to 100% of the work load) of the hemodynamic requirements of the patient [10, 13] but require a major surgical procedure and an associated high risk. Existing LVADs have demonstrated the ability to improve the salvage of patients with cardiogenic shock in the postsurgical setting [6, 8, 9, 14]. However, the attendant high surgical risk and complexity of existing LVADs have usually precluded their use in patients suffering from cardiogenic shock secondary to acute myocardial infarction (AMI).

It would be useful to have a device capable of supporting the majority of the hemodynamic work of the left ventricle while exposing the patient to a degree of risk similar to that attendant with the use of the IABP. The Hemopump is a new circulatory assist device that provides such a capability [15–18].

The Hemopump consists of (1) a continuous-flow intravascular pump that provides direct circulatory assistance by pumping blood from the left ventricle to the aorta, (2) a purge set assembly that provides for blood seal integrity and hydrodynamic lubrication for the pump and drive assembly, and (3) a console that provides power to the pump assembly and purge set assembly (Fig 1). The

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Hemopump is a registered trademark of Johnson and Johnson Interventional System Co, Rancho Cordova, CA. Nimbus Medical, Inc, is the developer of the Hemopump and Johnson and Johnson Interventional Systems Company owns the rights to the Hemopump. Dr Wampler is the inventor of the device, an employee of Nimbus Medical, Inc, and a consultant to the Johnson and Johnson Interventional Systems Company.

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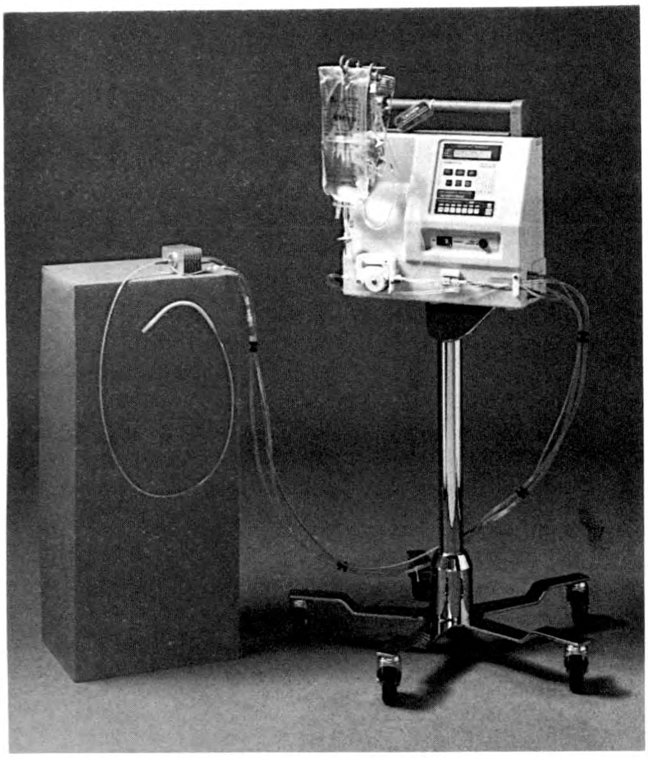


Fig 1. The Nimbus Hemopump system.

Hemopump can be placed through peripheral vascular access and support up to 80% of the left ventricular work load. The Hemopump can provide up to 3.5 L/min of flow without the need for a contribution from the left ventricle or synchronization. Because the inflow cannula is placed within the left ventricle, Hemopump assistance results in a significant degree of left ventricular decompression [13, 19].

A clinical investigation was initiated at seven centers (Appendix 1) to evaluate the safety and effectiveness of the Hemopump for use in the treatment of cardiogenic shock. The population selected for Hemopump treatment was composed of patients in cardiogenic shock who were prospectively selected for Hemopump assistance based on hemodynamic admission criteria when possible. The results of the first 53 patients selected for treatment with the Hemopump are described.

Method

Fifty-three patients with clinical evidence of cardiogenic shock were prospectively selected for treatment with Hemopump assistance. Human use was approved by the Food and Drug Administration under an Investigational Device Exemption and by the institutional review board at each clinical site. When available, hemodynamic measurements were used to identify candidates for assistance. The hemodynamic definition of cardiogenic shock was (1) a cardiac index (CI) of 2.0 $L \cdot min^{-1} \cdot m^{-2}$ or less, (2) pulmonary capillary wedge pressure (PCWP) of 18 mm Hg or greater, and (3) systolic blood pressure (SBP) of 90 mm Hg or less or a left ventricular work index (LVWI) of 1,500 $g\text{-}m \cdot m^{-2} \cdot min^{-1}$ or less (LVWI = CI × [MAP − PCWP] × 13.6, where MAP = mean arterial pressure). In addition, patients were required to demonstrate a refractoriness to volume and pharmacologic therapy.

Forty-one patients (33 male, 8 female) were supported for a mean time of 52.8 hours (range, 1 to 194 hours). Cardiogenic shock was secondary to AMI in 17 (41.5%), postcardiotomy shock in 17 (41.5%), and other causes in 7 (17.0%). The mean age was 54.7 years (range, 8.6 to 76.4 years). The pump could not be inserted in 12 patients (9 male, 3 female). Failure of insertion was secondary to severe atherosclerosis of the iliofemoral system, small native femoral artery (especially in women), and inability to cross the aortic valve. Cardiogenic shock in the noninsert group was secondary to AMI in 4 (33.3%), postcardiotomy shock in 5 (41.7%), and other causes in 3 (25%). Patients with serious blood dyscrasia, prosthetic aortic valve, severe aortic stenosis or regurgitation, or known dissecting aneurysm were not considered for Hemopump support. The IABP had been used immediately before Hemopump insertion in 75.5% of the patients assisted.

Study Protocol

APPLICATION OF THE HEMOPUMP. The Hemopump (Fig 2) was placed through surgical exposure of the femoral or external iliac artery (2 patients underwent retroperitoneal placement to the abdominal aorta). The device was then introduced into the arterial system and advanced under fluoroscopic imaging retrograde across the aortic valve such that the inflow cannula aspirated blood from the left ventricle and returned blood to the descending thoracic aorta. Patients were maintained on heparin anticoagulation to a therapeutic range of 1.5 to 2.0 times control of either the activated clotting time or the partial thromboplastin time. Anticoagulation was not initiated in postsurgical patients until the cessation of active bleeding.

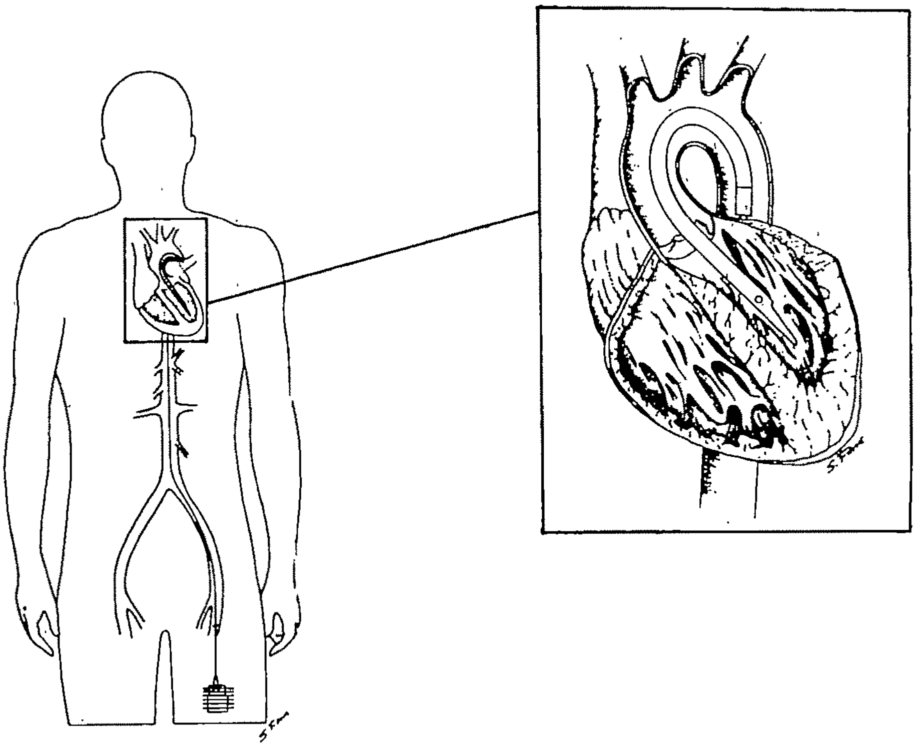
HEMODYNAMIC AND LABORATORY STUDIES. Samples for analysis of plasma free hemoglobin, complete blood count, platelets, serum haptoglobin, and routine chemistries were drawn before pump insertion, during pump assistance, and after pump removal. Cardiac index, PCWP, SBP, and MAP were recorded before pump insertion, at selected intervals during pump assistance, and after pump removal.

A 30-day follow-up study included physical and cardiologic examination covering cardiac functional status, echocardiography, vascular examination of the lower extremities, complete blood count, platelets, and plasma free hemoglobin level. Postmortem examination was performed on 23 patients.

Statistics

EVALUATION OF HEMODYNAMIC RESPONSE. The mean and standard error of the CI, PCWP, SBP, MAP, and LVWI before insertion were compared with hemodynamic variables during pump operation and after pump removal using an unpaired t test. The mean value, standard error, and p value are presented in the results.

Fig 2. Femoral artery insertion.



SURVIVAL OF HEMOPUMP VERSUS HEMODYNAMICS AT STUDY ADMIT. If a relationship between survival and preinsertion hemodynamics could be established, such a relationship could serve, clinically, as a basis for defining an indication for use of the Hemopump. The Hemopump patients for whom preinsertion data were available were stratified according to hemodynamic variables. The CI, PCWP, MAP, and LVWI were stratified into three or four sequential, equally sized groups based on the observed range of the respective hemodynamic variable. Survival for each strata was then graphically displayed to identify trends that might be clinically useful predictors of survival.

Results

Overall Patient Survival

Twenty-six (63.4%) of the Hemopump patients died on support or immediately after pump removal, and 15/41 (36.5%) were successfully weaned from support; 30-day survival for all patients supported by the Hemopump was 13/41 (31.7%). Patients with cardiogenic shock secondary to AMI demonstrated a 30-day survival of 7/17 (41.1%) compared with 4/17 (23.5%) for patients with postcardiotomy shock and 2/7 (28.6%) for those with other causes. The survival of the noninsertion group was 2/12 (16.6%), broken down as follows: acute myocardial infarction, 0 of 4 (0%); postcardiotomy shock, 1 of 5 (20%); and other causes, 1 of 3 (33%).

Hemodynamic Response

Values of CI, PCWP, SBP, MAP, and LVWI are presented in Table 1 for (1) before insertion, (2) at 24 hours, (3) mean during assistance, and (4) after removal. Due to the emergency nature of the procedures, complete hemodynamic data were not available on all patients, particularly before pump insertion. Testing for statistical significance compared the preinsertion value versus the value at 24

Table 1. Hemodynamic Variables

Variable	Before Insertion	24 Hours	Mean During Assistance	After Removal
CI (L · min^{-1} · m^{-2})	1.63	2.19	2.09	2.92
n	31	23	31	15
p Value[a]		0.005	0.011	<0.001
PCWP (mm Hg)	27.0	16.6	17.2	16.5
n	24	24	30	14
p Value		<0.001	<0.001	<0.001
SBP (mm Hg)	74.0	79.4	72.5	100.5
n	41	25	39	17
p Value		NS	NS	<0.001
MAP (mm Hg)	55.8	66.6	61.8	70.9
n	40	24	39	17
p Value		<0.001	0.054	<0.001
LVWI (g-m · m^{-2} · min^{-1})	841	1,620	1,663	2,249
n	27	23	21	13
p Value		0.024	0.001	0.0178

[a] Versus before insertion.

CI = cardiac index; LVWI = left ventricular work index; MAP = mean arterial pressure; NS = not significant; PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure.

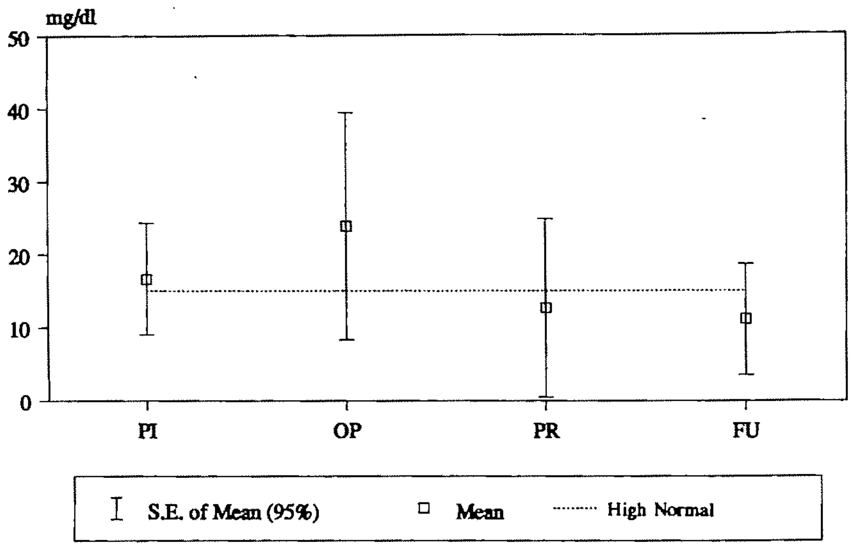


Fig 3. Plasma free hemoglobin. (FU = *30-day follow-up;* OP = *operation at 24 hours;* PI = *before insertion;* PR = *after removal at 24 hours;* S.E. = *standard error.)*

hours of operation, the mean during assistance, and the value after removal.

Plasma Free Hemoglobin

Plasma free hemoglobin levels before insertion, after insertion, before pump removal, and at 30-day follow-up are shown in Figure 3. The mean level of plasma free hemoglobin during pump operation was 170 mg/L (17 mg/dL) for the AMI and other group and 350 mg/L (35 mg/dL) for the postcardiotomy group. During pump operation the plasma free hemoglobin level ranged from 10 mg/L (1 mg/dL) to an isolated high of 2,030 mg/L (203 mg/dL). Characteristically, a transient increase was seen in most patients during the first 24 hours of operation, after which the level of plasma free hemoglobin decreased and stabilized for the duration of pump assistance. Twelve patients had at least one free plasma level greater than 500 mg/L (50 mg/dL) during pump operation; 8 of these were patients who were on cardiopulmonary bypass before Hemopump insertion, 3 suffered AMIs, and 1 was a child (body weight, 28 kg). The plasma free hemoglobin level decreased substantially in 8 of 9 patients who survived at least 24 hours. No patient injury or death was reported to be related to intravascular hemolysis.

Platelets

The mean value for platelets for all patients is presented in Figure 4. Before pump insertion, the mean value was 222 × 10^9/L (222 × $10^3/\mu L$), which decreased to a mean of 115 × 10^9/L (115 × $10^3/\mu L$) during operation. The platelet level began increasing quickly after pump removal and was 308 × 10^9/L (308 × $10^3/\mu L$) at 30 days. The range during pump operation was 13 to 412 × 10^9/L (13 to 412 × $10^3/\mu L$). Platelet transfusion was administered in 3 patients. The reduction in platelets occurring during Hemopump assistance was not associated with spontaneous bleeding.

Complications and Adverse Events

The patient complications and adverse events during hospitalization reported by the principal investigators are

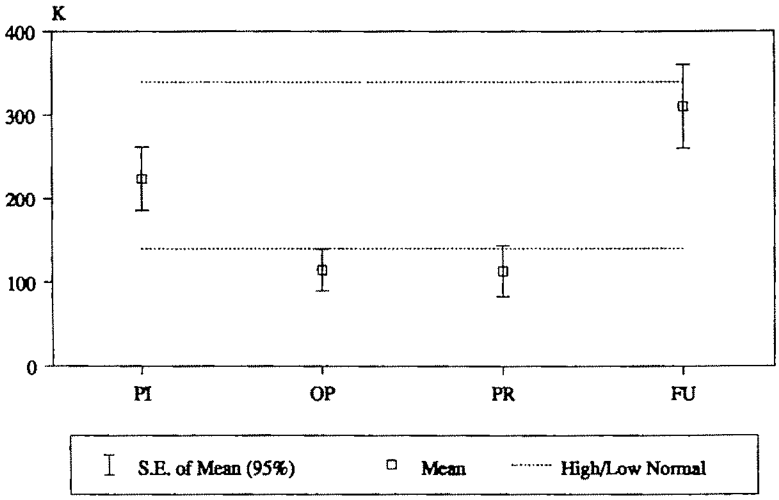


Fig 4. Platelets (abbreviations as in Figure 3).

Table 2. Complications and Adverse Events

Adverse Event	Incidence	Comment
Failure to insert	12/53 (22.6%)	. . .
Fracture of drive cable	4/41 (9.8%)	Related to early design problem, now resolved
Septicemia	3/41 (7.3%)	Not device related
Insertion site infection	1/41 (2.4%)	. . .
Thrombocytopenia	3/41 (7.3%)	Platelet transfusion
Dysrhythmia	11/40 (27.5%)	Not associated with hemodynamic sequelae
Systemic thromboembolism during assistance	1/41 (2.4%)	Small renal infarct at postmortem examination
Neurological event	2/41 (4.8%)	Hemiparesis 14 and 18 days after pump removal; preexisting mural thrombi
Peripheral embolism	1/41 (2.4%)	Two days after pump removal, associated with preexisting mural thrombus

listed in Table 2. No patient death was reported by a principal investigator as being a result of the Hemopump. No leg ischemia was observed. Aspiration of a mural thrombus resulted in an acute loss of assistance and death in 1 patient. A severe right to left shunt resulted in profound hypoxemia and death in a patient with biventricular failure and an unrecognized patent foramen ovale.

A total of four embolic events were recorded in 3 patients. A small renal infarction was found at postmortem examination in 1 patient, which likely occurred during pump operation (pump related). Two patients suffered hemiparesis (14 and 18 days) after pump removal, associated with atrial fibrillation in 1 and preexisting mural thrombus in the other. One late femoral embolism was related to preexisting mural thrombus and was successfully managed with thrombectomy. The total incidence of systemic thromboembolism during and after pump operation was 9.6%. A pump-related thromboembolism rate of 2.4% was observed.

Factors That Influence Survival

The preinsertion CI, PCWP, and MAP had little clinical value in predicting survival. However, the LVWI may provide a means of defining the severity of cardiogenic shock. If the LVWI values of patients before insertion of the Hemopump are compared, the impact of patient selection and timely intervention can be seen. Figure 5 shows survival as a function of the LVWI at study admit for Hemopump patients. At relatively low initial work indices (less than 500 g-m $\cdot$ m^{-2} $\cdot$ min^{-1}), the survival was 1 of 7 (14.3%). With a work index from 500 to 1,000 g-m $\cdot$ m^{-2} $\cdot$ min^{-1}, survival was 3 of 9 (33.3%). If the work index was between 1,000 and 1,500 g-m $\cdot$ m^{-2} $\cdot$ min^{-1}, the survival was 7 of 11 (63.6%).

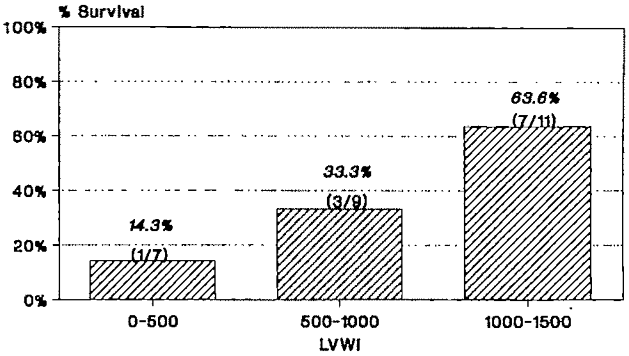


Fig 5. Survival (%) versus left ventricular work index (LVWI) before insertion.

Cardiac Index Versus PCWP

The hemodynamic response to Hemopump assistance and the prognosis for left ventricular recovery and survival can be correlated with the CI and PCWP. The prognosis after initiation of therapy may be evaluated using the CI and PCWP after 24 hours of assistance. Figure 6 is a display of CI versus PCWP of all Hemopump patients alive at 24 hours. It can be seen that if the CI was less than 2.0 L $\cdot$ min^{-1} $\cdot$ m^{-2} or the PCWP was greater than 18.0 mm Hg, the 30-day survival was only 10%. However, if the hemodynamic condition improved such that the CI was greater than 2.0 L $\cdot$ min^{-1} $\cdot$ m^{-2} and the PCWP was less than 18.0 mm Hg, 12 of 13 patients (92.3%) could be weaned from support and 10 of 13 (76.9%) survived to 30 days.

Comment

Overall Survival

Survival of cardiogenic shock is reported in the literature to range from 10% to 20% [2–4]. Because 75.5% of Hemopump patients were failing on the balloon pump with

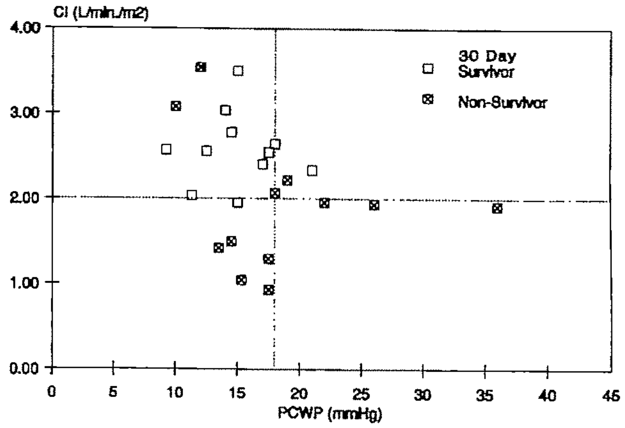


Fig 6. Cardiac index (CI) versus pulmonary capillary wedge pressure (PCWP) at 24 hours.

maximal pharmacologic therapy before Hemopump assistance, the overall Hemopump survival of 31.7% potentially demonstrates a substantial improvement over treatment with the IABP. The survival of patients who were selected for Hemopump assistance but who did not have successful insertion of the Hemopump was 2 of 12 (16.6%). The LVAD registry reported an overall survival of 25.5% in 1989 [20], which compares favorably with our overall results.

Complications

The general safety of the Hemopump was shown by demonstrating (1) acceptable level of injury to blood-borne elements such as erythrocytes and platelets, (2) acceptable incidence of intracardiac and vascular injury observed at autopsy, (3) minimal adverse effects on the coagulation system beyond that expected from systemic heparinization, (4) acceptable incidence of thromboembolic events, and (5) demonstration of reliable operation of the Hemopump.

HEMOLYSIS AND THROMBOCYTOPENIA. One of the major safety concerns related to the use of the Hemopump was the potential for injury to blood cellular elements, particularly erythrocytes. Normal levels of plasma free hemoglobin are less than 150 mg/L (15 mg/dL), although elevations to 1,000 mg/L (100 mg/dL) can be observed after cardiopulmonary bypass. Multiple transfusions, particularly in combination with cardiopulmonary bypass, can also contribute to intravascular hemolysis. Two thirds (8/12) of the patients who demonstrated at least one plasma free hemoglobin level of greater than 500 mg/L (50 mg/dL) had been on cardiopulmonary bypass. The highest level observed, 2,030 mg/L (203 mg/dL), was in a patient who had been on cardiopulmonary bypass. The persistent elevation of 550 mg/L (55 mg/dL) observed in the small (28 kg) child was likely related to a small circulating plasma volume. All three patients with AMI who had an initial transient elevation greater than 500 mg/L (50 mg/dL) experienced a rapid decrease after the first 24 hours of assistance. Attrition of senescent erythrocytes could explain this transient increase during the first 24 hours of operation.

Plasma free hemoglobin is relatively nontoxic unless it precipitates in the renal tubules. The renal threshold for plasma hemoglobin is nominally 1,000 mg/L (100 mg/dL). The mean levels of plasma free hemoglobin during Hemopump operation were well below 1,000 mg/L (100 mg/dL) and were considered to pose a minimal risk to the patient (see Fig 3).

A significant decrease in platelets was observed during Hemopump assistance. The decrease in platelets was more severe in patients after cardiopulmonary bypass than in patients with acute myocardial infarction or other causes. Decreases in platelets to levels associated with spontaneous bleeding were effectively treated with platelet transfusion (3 patients).

THROMBOEMBOLISM. The reported incidence of thromboembolic events in patients suffering from AMI or mural thrombus or undergoing LVAD implantation ranges from 5% to 15% [21]. The pump-related thromboembolism rate in this study was 2.4%. Patients with AMI may have a mural thrombus; thus the potential for aspiration of a mural thrombus into the pump must be weighed.

VENTRICULAR DYSRHYTHMIA. The Hemopump cannula is a large foreign body that, when positioned in the ventricular cavity, might be expected to produce some degree of ventricular ectopy. The clinical significance of ventricular ectopy and malignant rhythms relates to the hemodynamic decay normally associated with these rhythms. In general, it has been observed during the trials that the cardiac output and MAP are much less sensitive to dysrhythmia when supported by the Hemopump. The ability to maintain adequate hemodynamic support during periods of severe dysrhythmia during Hemopump assistance has been documented in patients. However, not all patients tolerate dysrhythmia during Hemopump assistance, and on occasion cardioversion was required to maintain viable hemodynamics. Dysrhythmias observed include sustained ventricular tachycardia and, on one occasion, ventricular fibrillation. The patient with ventricular fibrillation was supported for 45 minutes until successfully defibrillated. This patient maintained acceptable cardiac output and was conscious during this interval.

DEVICE OPERATION AND SAFETY. The Hemopump was, in general, reported to function reliably and was easily integrated within the environment of the intensive care unit. The small size and simplicity of operation of the Hemopump, compared with an LVAD or IABP, is not anticipated to require additional personnel, such as a perfusionist, to monitor operation. The most clinically significant device failures reported were drive-cable failures.

The high speed of rotation of the flexible drive cable, 25,000 rpm, imposes limits on the geometry and handling of the drive cable in the implant environment. Fracture of the drive cable results in complete loss of assistance. A nonfunctioning, correctly placed Hemopump may produce some degree of aortic stenosis and insufficiency and requires either pump removal or replacement. A high incidence of cable fracture (60%) was observed early in the trial and necessitated a redesign of the inflow cannula to minimize stress concentration. Since implementation of the redesign, a cable fracture rate of 5.2% has been observed; the majority of these fractures have been at least partially related to improper insertion techniques or failure to keep the drive cable free of acute bend radii. Cable fractures at this time can largely be avoided by strict adherence to proper handling and attention to implant geometry. Patient injury or death have not been reported as a sequela of cable fracture. A new drive cable has been shown to exhibit superior durability and handling and will be available in 1992.

RIGHT TO LEFT SHUNTING. Patients with severe biventricular failure who also have a patent foramen ovale or acquired ventricular septal defect secondary to acute myocardial infarction are at risk for right to left shunting when supported by any device that employs continuous flow pumping, ie, centrifugal or axial flow devices. The incidence of an anatomically patent foramen ovale is estimated to be 10% to 20%, but only 1 patient (2.4%) demonstrated serious shunting during Hemopump assistance. The presence of a right to left shunt should be suspected if desaturation of the blood occurs with the initiation of assistance. If desaturation is found during pump assistance, a reduction in pump speed may reduce the size of a shunt.

Hemodynamic Efficacy

A clinically and statistically significant improvement of the CI, PCWP, SBP, and LVWI occurred in response to Hemopump assistance. Although improvement in these hemodynamic variables would be expected to facilitate peripheral perfusion, organ function, and left ventricular recovery, many patients did not experience hemodynamic improvement sufficient to permit survival to weaning or survival to 30 days. Patients who could not achieve both a cardiac index of greater than 2.0 $L \cdot min^{-1} \cdot m^{-2}$ and a pulmonary capillary wedge pressure of less than 18 mm Hg were, primarily, in a state of biventricular failure with concomitant volume loading. In addition, patients with multiorgan failure and other complications of cardiogenic shock often did not survive in spite of an adequate hemodynamic response and, in many cases, left ventricular recovery. Preinsertion variables that might be useful in identifying patients likely to benefit from Hemopump assistance are discussed in the next section.

Factors in Favor of Survival

Predictably, survival diminishes as the severity of illness increases. Thus, patient selection and early intervention would be expected to result in a substantial increase of overall survival of patients in cardiogenic shock treated with the Hemopump. Unfortunately, a convention useful in measuring the severity of cardiogenic shock and prognosticating survival awaits definition and universal acceptance. In the Hemopump study, analysis of survival to weaning and to 30 days with respect to CI, PCWP, or MAP did not reveal clinically useful trends. However, calculated left ventricular work functions do appear to have value as prognosticators of survival based on analysis in this study and on reports by other investigators [2–4].

Evaluation of any one of the hemodynamic variables (CI, SBP, PCWP) may not present an accurate picture of the "global" hemodynamic condition of the patient and, thus, has little prognostic value. The left ventricular work index (LVWI), when applied to Hemopump patients, appears to mirror the global hemodynamic condition of the patient and demonstrates a good correlation with survival to weaning and 30 days.

The LVWI is a reflection of the hydraulic work, as power, that takes place when blood is moved from the left ventricle into the aorta. During Hemopump assistance the LVWI is a reflection of the combined work of the Hemopump and the left ventricle. The LVWI can be useful clinically because (1) it provides an assessment of the rate at which work is generated across the aortic valve and (2) it is an indicator of the severity of left ventricular dysfunction and can be used as a prognostic indicator of survival. A normal LVWI is approximately 3,000 to 3,500 $g\text{-}m \cdot m^{-2} \cdot min^{-1}$. Based on Hemopump inclusion criteria, a Hemopump candidate would have an LVWI of 1,500 $g\text{-}m \cdot m^{-2} \cdot min^{-1}$ or less. When a large improvement in the LVWI was seen in response to Hemopump assistance, a large clinically important trend toward left ventricular recovery and improvement in 30-day survival was observed.

The LVWI may be useful clinically to aid the physician in (1) assessing the severity of cardiogenic shock and timing of Hemopump intervention and (2) assessing the adequacy of Hemopump assistance and the level of left ventricular recovery.

Limitations of the Study

Of the 41 patients who underwent successful pump insertion, 27 had sufficient hemodynamic data before pump insertion to support the calculation of LVWI. Patients for whom preinsertion data were not available were in a group in which clinical circumstances precluded full measurement of the hemodynamic status of the patient. These circumstances included, in many cases, simultaneous cardiopulmonary bypass (failure to wean) and patients undergoing acute resuscitation in whom the inherent delay of invasive monitoring would have diminished the probability of survival.

The small sample size and heterogeneity of the patient cohort limited a rigorous statistical analysis between the different diagnostic groups and the subsets of patients.

Conclusion

The clinical study has demonstrated the hemodynamic effectiveness and safety of the Hemopump when used in the treatment of cardiogenic shock. Hemopump therapy resulted in a significant decrease in PCWP and an increase in the SBP, MAP, CI, and LVWI. Patients who underwent successful insertion of the Hemopump demonstrated improved survival approaching statistical significance when compared with patients in whom the Hemopump could not be placed. The hemodynamic effectiveness of the Hemopump may result in substantial increase in patient survival in select patients and is associated with acceptable risk. It is concluded that the Hemopump is an effective, safe, and practical LVAD and may play a major role in the treatment of cardiogenic shock.

The invaluable help of Karen Correa in the preparation of the manuscript is gratefully appreciated.

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Appendix 1.

The Hemopump clinical study is currently ongoing at 7 institutions: (1) St. Luke's Episcopal Hospital (The Texas Heart Institute) in Houston, TX; (2) Humana Hospital Audubon in Louisville, KY; (3) The University of Michigan Medical Center in Ann Arbor, MI; (4) Hermann Hospital (The University of Texas Medical School) in Houston, TX; (5) Emory University and Crawford Long Hospitals in Atlanta, GA; and (6) The Cleveland Clinic in Cleveland, OH. Clinical trials were also conducted at Mercy Hospital Medical Center in Des Moines, IA, from May 1988 to December 1988.

Investigators who have been involved in the Hemopump clinical study, by institution, include the following physicians: *The Texas Heart Institute, Houston, TX*: Principal Investigator, O. Howard Frazier, MD; Co-Investigators, Wayne Dear, MD, J. Michael Duncan, MD; *Humana Hospital Audubon, Louisville, KY*: Principal Investigator, Allan Lansing, MD, PhD; Co-Investigators, Ronald Barbie, MD, Zahi Masri, MD; *The University of Texas Medical School, Houston, TX*: Principal Investigator, Richard W. Smalling, MD, PhD; Co-Investigator, Michael Sweeney, MD; *The University of Michigan Medical Center, Ann Arbor, MI*: Principal Investigator, John Nicklas, MD; Co-Investigators, G. Michael Deeb, MD, Michael Shea, MD, Steven Bolling, MD, Jeffrey Lieden, MD; *Mercy Hospital Medical Center, Des Moines, IA*: Principal Investigator, Steven J. Phillips, MD; Co-Investigators, Robert Zeff, MD, Cham Kongtahworn, MD, William Wickemeyer, MD, Mark D. McGaughey, MD, Liberato Iannone, MD, Prasad Palakurthy, MD; *Emory University and Crawford Long Hospitals, Atlanta, GA*: Principal Investigator, Robert Guyton, MD; Co-Investigators, Spencer King, MD, Patrick Curling, MD, Kirk Kanter, MD, Omar Lattouf, MD, Henry A. Liberman, MD; *The Cleveland Clinic Foundation, Cleveland, OH*: Principal Investigator, Leonard Golding, MD; Co-Investigators, Patrick Whitlow, MD, Russell Raymond, DO.

Sepsis After Coronary Bypass Grafting: Evidence for Loss of the Gut Mucosal Barrier

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Postoperative infections may originate from a patient's gastrointestinal tract. We studied infections after coronary artery revascularization. Three hundred twenty-nine patients underwent coronary artery revascularization from January 1987 to March 1990. Eight of the 329 (2.4%) died; none of the deaths were infection related. Fifty-five culture-proven infections were identified in 22 of 321 survivors (6.8%); 9 infections (16%) were gram-positive, 5 (9%) were fungal, and 41 (75%) were gram-negative. Site of infections were respiratory tract, 58%; urinary tract, 18%; blood, 13%; and mediastinum, 11%. Ninety-six percent of respiratory tract and all urinary tract infections were gram-negative or fungal. There was no significant difference between infected and noninfected groups in sex, age, smoking history, preoperative hematocrit or leukocyte count, serum albumin level, or time on extracorporeal bypass. The infected group required intubation and nasogastric suction for a significantly longer time than the noninfected group ($p < 0.001$). Time to enteral alimentation was significantly longer in the infected group ($p < 0.02$). We were unable to correlate the number of infections with the lengths of intubation, nasogastric suction, or time to enteral alimentation. This study supports the concept of postoperative infections arising from bacterial translocation across the patient's gastrointestinal tract. The most significant risk factor is the length of the gastrointestinal tract disuse.

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Patients requiring cardiopulmonary bypass support occasionally have a postoperative course complicated by bacteremia or sepsis. Predisposition to sepsis is variously attributed to general ill health, malnutrition, immunosuppressive effects of extracorporeal bypass, prolonged intubation, or use of indwelling appliances (eg, drains). Recent studies suggest iatrogenic gastric alkalinization or prolonged gastrointestinal (GI) inactivity may compromise normal GI barrier systems and allow autoinfection by GI tract flora [1].

We review our experience with sepsis after cardiopulmonary bypass for coronary revascularization and present an analysis of etiology.

Material and Methods

Patients undergoing cardiopulmonary bypass at the United States Air Force Keesler Technical Training Center Medical Center from January 1987 to March 1990 were retrospectively reviewed. Patients with culture-proven septic complications were identified and stratified considering standard epidemiologic data, smoking history, length of time on bypass, gastric decompression and alkalinization, GI tract disuse, and bacteriologic profiles.

Statistical comparisons of infected and noninfected groups were accomplished by χ^2 analysis for epidemiologic data and by linear regression with analysis of variance testing for comparison of time with the number of observed infections. Significant differences between groups regarding time intubated, time with nasogastric suction, and time to feeding were confirmed by repeat analysis after log transformation.

Results

Three hundred twenty-nine patients underwent coronary artery revascularization with intraoperative cardiopulmonary bypass from January 1987 to March 1990, inclusive. There were 277 men (84%) and 52 women, ranging in age from 37 to 80 years (mean, 59.9 years). Eight patients (2.4%) died (6 male, 2 female). There were no infection-related deaths. Complete chart data were available for 321 surviving patients; culture-proven infections were identified in 22 (6.8%). Of the 55 infections, 9 (16%) were gram-positive, 5 (9%) were fungal, and the remainder (75%) were gram-negative. The majority of infections were of the respiratory tract (58%), followed by urinary tract (18%), blood (13%), and mediastinum (11%). The preponderance of respiratory tract infections (96%) and all of the urinary tract infections were gram-negative or fungal. Gram-positive bacteremia was identified in five of six productive blood cultures; 3 of these patients required indwelling percutaneous cardiac assist devices in the immediate perioperative period. Four mediastinal infections were gram-positive; 2 of these patients had been reexplored for postoperative bleeding.

There was no significant difference between the in-

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The opinions expressed herein are those of the authors and not necessarily those of the Air Force or the Department of Defense.

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Table 1. Comparison of Coronary Artery Bypass Patients With and Without Postoperative Infections

Variable	Noninfected	Infected	p Value
Male	84%	78%	NS
Female	16%	22%	NS
Mean age (y)	58.4 (38–80)[a]	61.6 (40–75)	NS
Elective operation	88%	73%	NS
Smoking (pack-years)	39.8 (0–187)	35 (0–100)	NS
Bypass time (min)	92.8 (30–231)	92.6 (48–203)	NS
Hemoglobin (g/L)	411 (319–474)	397 (251–547)	NS
Leukocyte count ($\times 10^6$/L)	8.1 (2.2–17.9)	8.2 (4.8–14.3)	NS
Serum albumin (g/L)	37 (20–53)	35 (20–49)	NS
Time intubated (h)	23.7 (0–190)	69.6 (9–360)	<0.001
Time with NGT (h)	24.1 (4–187)	77.6 (9–350)	<0.001
Time to PO (h)	55 (24–192)	107.8 (48–360)	<0.02

[a] Numbers in parentheses are range.

NGT = nasogastric tube; NS = not significant; PO = volitional feeding.

fected and noninfected groups with regard to sex, age, smoking history, preoperative hematocrit or leukocyte count, serum albumin level, or time on extracorporeal bypass (Table 1). The infected group required intubation and nasogastric suction for a significantly longer period of time than the noninfected group ($p < 0.001$). The time to enteral alimentation was also significantly longer in the infected group ($p < 0.02$). We were unable to demonstrate a significant correlation between the number of infections observed and the length of time patients were intubated and treated with nasogastric suction or the length of time to enteral alimentation.

Comment

Coronary artery revascularization is an effective treatment of myocardial ischemia due to atherosclerotic coronary artery disease [2]. Coronary artery bypass grafting (CABG) is a standard surgical procedure with a mortality risk of 2.3% (within 30 days) and an infectious complication risk of 6.4% within 6 months of operation [2–4]. Infectious complications most often include wound infection (3.4%), urinary tract infections (1.8%), and pulmonary infections (1.2%). Perioperative factors identified as influencing the development of postoperative sepsis include the patient's age, nutritional status, smoking history, length of bypass, use of gastric acid neutralizing agents, prolonged intubation, and prolonged GI tract disuse [5–9]. We critically analyzed culture-proven infectious processes within our CABG experience to identify etiology as a guide to prevention.

Our infectious complication rate (6.8%) is comparable with reported series. Eleven percent of our infections were at sternal or groin wounds. Two cutaneous wounds grew gram-positive bacteria (staphylococcus), whereas the remainder grew enteric organisms (*Enterobacter*, *Pseudomonas*). The 2 patients with gram-positive cultures had been reexplored in the immediate postoperative period for bleeding. Fifty-eight percent of the infections were of the respiratory tract. Cultures from all of the patients with respiratory tract infections grew enteric organisms (*Escherichia coli*, *Klebsiella*, *Enterobacter*, *Haemophilus*, *Candida*). In a collected review of 7,559 patients undergoing cardiac operations we found 148 patients with perioperative infections; 49% were enteric [9–11].

Extracorporeal cardiopulmonary bypass provides nonphysiologic pulsatile tissue perfusion, which may stimulate release of complement and histamines with subsequent impairment of the semipermeable properties of basement membranes. Bypass has been shown to be followed by expansion of the extracellular water compartment, a significant decrease in the colloid oncotic pressure, and movement of fluid across capillaries into the interstitium [12]. Cellular metabolism is affected, with reduced bicarbonate levels and increased lactate levels, but overall intracellular metabolism is apparently unaffected as evidenced by normal postbypass levels of intracellular adenosine triphosphate, water, and pH [13, 14]. Logistic regression analysis of multiple variables in bypass patients with infections has implicated length of extracorporeal bypass to be contributory in development of postoperative infections [9]. The relatively short bypass runs required for CABG do not seem to disrupt GI barrier function at the cellular level. We found no significant difference in the length of bypass between our reference population and those with sepsis.

Early observations of critical care patients undergoing gastric acid neutralization (antacids and histamine blockers) to prevent stress gastritis and hemorrhage demonstrated that nosocomial pulmonary infections were usually of enteric origin [5, 6]. Gastric alkalinization may add to postoperative infections by disrupting normal enteral sterilization performed by gastric acid. Our patients are all treated with antacids, as standard procedure, in the immediate postoperative period to prevent stress gastritis. Each patient receives 30 to 90 mL of a commercially available suspension of aluminum hydroxide, magnesium hydroxide, and simethicone every 2 hours for a measured gastric pH greater than 4.5. Cimetadine (300 mg intravenously every 8 hours) is added if the antacids fail to keep gastric pH greater than 4.5. There were no deviations of prophylaxis between the infected and noninfected groups in our series. Gastric alkalinization therefore could not be implicated as a risk factor to sepsis.

Recent laboratory and clinical studies have defined a mucosal barrier that is responsible for separation of the GI tract exterior milieu from the somatic interior milieu [15]. The barrier is multifactorial, consisting of the mucus lining, enterocyte competence, normal enteric bacterial ratios, prevention of bacterial endoadherence, and GI secretory immunoglobulin production [16, 17]. Disruption of the GI barrier is directly related to length of hypotensive episodes during shock, GI tract disuse, and poor nutritional support [15, 18].

A significant difference between our septic and nonseptic groups was identified in the length of time that the GI tract was not used. Similar observations have been made in patients undergoing GI operations. The longer a patient goes without enteral alimentation, the greater the risk of disruption of the GI barrier and subsequent enteric autoinfection [7, 19]. Maintenance of the barrier in critically ill patients requires full caloric support, preferably through the enteral route [20, 21]. The amino acid glutamine is essential for enterocyte survival, GI tract immunoglobulin production, and prevention of bacterial translocation across the GI barrier [22].

With growing evidence underscoring the importance of a competent GI barrier in preventing postoperative sepsis, we stress aggressive enteral nutritional support in our CABG patients. Volitional enteral nutrition is begun within the first 24 hours of convalescence in the uncomplicated patient. Nasojejunal feeding tubes are placed within 48 hours in those patients who cannot eat because of prolonged intubation or postoperative mechanical support. Nonvolitional enteral alimentation is begun with half-strength chemically defined, low fat formulas that are supplemented with glutamine and branched-chain amino acids. Chemically defined formulas are easily absorbed, even in the patient with early postoperative ileus. Glutamine is important in enterocyte and GI immune system competence. The branched-chain amino acids are preferentially used as an energy source within the skeletal muscle and help prevent skeletal muscle catabolism during recuperation. Long-chain ω-6 fatty acids (intralipids) have been implicated as immunosuppressive in critically ill patients by impairment of chemotaxis, cytokine production, and antigen processing by leukocytes. Fats also impair respiratory activity by causing bronchial constriction and increased bronchial mucus production. Maximizing overall immune competence and tissue oxygen delivery is essential in intensive care unit patient management, so we limit fat administration to twice-weekly intravenous lipids for essential fatty acid supplementation only [23, 24].

The rate of continuous drip formula delivery is advanced to full fluid requirements over 2 to 3 days. The concentration of formula is then increased over 1 to 2 days to meet full caloric needs. Those hypoalbuminemic patients who are intolerant of enteral support (bloating, distention, watery diarrhea) are placed on full parenteral caloric support; the albumin deficit is calculated, and the albumin is replaced over 3 days. Using these guidelines we have been able to achieve full enteral support in more than 90% of our patients.

Since instituting our early-alimentation program, we have treated 64 patients with CABG. Four patients have had postoperative infections. Two of those patients had preoperative, culture-proven infections (suppurative thrombophlebitis, 1; pneumonia, 1) and underwent CABG on an emergency basis. The post-CABG cultures produced bacteria identical in species to those of the preoperative cultures (staphylococcus, 1; *Klebsiella*, 1). A third patient, with a history of previous CABG, underwent a procedure on the orthopedic service and did not receive postoperative alimentation for 192 hours. During his postoperative intensive care unit hospitalization, myocardial ischemia developed because of thrombosed coronary bypass grafts, and he required emergent redo CABG. A *Klebsiella* pneumonia developed in him.

One patient is considered an early feeding protocol failure. He underwent emergent CABG after profound myocardial ischemia developed during cardiac catheterization and required perioperative intraaortic balloon pump support. He was intubated for 190 hours and was supported with early postoperative enteral alimentation, but a *Haemophilus* pneumonia developed in him.

Early postoperative enteral alimentation by volitional or nonvolitional means may be an important adjunct in preventing bacterial translocation from the GI tract and subsequent postoperative infections.

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Early Postoperative Sternal Approximation After ITA Harvesting: Computed Tomographic Evaluation

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Between November 1989 and February 1990, 66 randomized sternotomized patients underwent aortocoronary bypass and were subjected to a sternal scanner in the early postoperative period. Each examination included a manubrial and a sternal body print. At each level, we studied the occurrence of spacing or misalignment of the sternal layers. The 66 patients were subdivided into four groups according to the type of conduit harvested (single left internal thoracic artery or saphenous vein) and the type of material used for the sternal closure (steel wires or nylon yarns). In all cases, adequate early sternal approximation, which is represented by a good alignment as well as by an excellent contact of the sternal layers, was infrequently demonstrated. Moreover, the two abnormalities most often observed were manubrial spacing and sternal body misalignment. The sternal closure technique and internal thoracic artery harvesting had no significant effect on the sternal approximation. To minimize manubrial spacing and sternal body misalignment, we propose that the surgeon should apply three threads through the manubrium, withdraw the shoulder roll beforehand, elevate both of the patient's shoulders, and maintain the two xyphoid layers in the same plane and in fairly close contact during the tightening of the wires.

(Ann Thorac Surg 1991;52:518–22)

Since 1957 [1], median sternotomy has become the standard incision used for most cardiac surgical procedures. Many authors have studied the frequency and severity of complications related to morbidity and mortality. The incidence of complications ranges between 0.5% and 5%. Table 1 lists the frequency of complications that we found in the literature between 1973 and 1990. Mediastinitis seems to appear in less than 1% of cases. In our department, a retrospective study (between January 1987 and May 1989) on 1,073 patients showed a 2.42% complication rate, with a mediastinitis rate of 0.37%.

Many factors have been mentioned as predisposing to these complications, such as prolonged intubation and respiratory assistance, reexploration for hemorrhage, low postoperative cardiac output, postoperative external cardiac massage, diabetes, tobacco smoking, and chronic renal failure. Regarding the basic concepts of traumatology, the quality of the early postoperative sternal approximation seems to be an important factor that needs to be studied carefully.

Adequate healing of fractured bone depends on an anatomically perfect setting, a dynamic compression at the fracture site, and a strong approximation material. If these principles are not respected, instability, deep-seated infection, or both could arise at the sternal level.

Internal thoracic artery (ITA) harvesting imposes a vigorous sternal retraction and a sternal raising. Does it influence the early postoperative sternal approximation? To answer this question, the present study was undertaken.

Material and Methods

Patient Profile

Sixty-six randomized patients underwent a median sternotomy for aortocoronary bypass grafting between No-

Table 1. Review of Literature

Author	n	Patients with Complication (%)	Complication
Engelman et al [2]	1,042	1.6%	Mediastinitis
Culliford et al [3]	2,594	1.5%	Mediastinitis
Stoney et al [4]	4,531	0.8%	Sternal dehiscence
Serry et al [5]	4,125	1.8%	Global
Breyer et al [6]	870	2.9%	Global
Sarr et al [7]	824	0.7%	Mediastinitis
Grossi et al [8]	7,949	1.0%	Mediastinitis
Scully et al [9]	5,032	1.0%	Mediastinitis
Ottino et al [10]	2,579	1.9%	Mediastinitis
Johnson et al [11]	883	2.7%	Mediastinitis
Hazelrigg et al [12]	2,582	0.8%	Global

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Table 2. Characteristics of the Population[a]

	ITA		SV	
Variable	Steel (n = 21)	Nylon (n = 12)	Steel (n = 12)	Nylon (n = 11)
Age (y)				
<70	18 (85.7)	17 (77.3)	6 (50.0)	4 (36.4)
>70	3 (14.3)	5 (22.7)	6 (50.0)	7 (63.6)
Male	14 (66.7)	17 (77.3)	12 (100.0)	8 (72.7)
Female	7 (33.3)	5 (22.7)	0 (0.0)	3 (27.3)
Weight (kg)				
<80	19 (90.5)	17 (77.3)	11 (91.7)	11 (100.0)
>80	2 (9.5)	5 (22.7)	1 (8.3)	0 (0.0)
Height (cm)				
<180	20 (95.2)	21 (95.5)	12 (100.0)	11 (100.0)
>180	1 (4.8)	1 (4.5)	0 (0.0)	0 (0.0)
Smoker				
No	15 (71.4)	11 (50.0)	5 (41.7)	7 (63.6)
Yes	6 (28.6)	11 (50.0)	7 (58.3)	4 (36.4)
Diabetes				
No	17 (81.0)	20 (90.9)	8 (66.7)	7 (63.6)
Yes	4 (19.0)	2 (9.1)	4 (33.3)	4 (36.4)
Emergency				
No	20 (95.2)	21 (95.5)	9 (75.0)	11 (100.0)
Yes	1 (4.8)	1 (4.5)	3 (25.0)	0 (0.0)
First procedure	21 (100.0)	21 (95.5)	11 (91.7)	8 (72.7)
Ventilation (h)				
<24	20 (95.2)	21 (95.5)	10 (83.3)	8 (72.7)
>24	1 (4.8)	1 (4.5)	2 (16.7)	3 (27.3)
Reexploration				
No	20 (95.2)	18 (81.8)	12 (100.0)	8 (72.7)
Yes	1 (4.8)	4 (18.2)	0 (0.0)	3 (27.3)

[a] Numbers in parentheses are percentages.

ITA = internal thoracic artery; SV = saphenous vein.

vember 1989 and February 1990. After operation, the patients were divided into four groups according to the method of harvesting, ie, single left ITA or saphenous vein harvesting, and the type of material used for the sternal closure. The four groups are as follows: group 1 (n = 21), ITA steel closure; group 2 (n = 22), ITA nylon closure; group 3 (n = 12), saphenous vein steel closure; and group 4 (n = 11), saphenous vein nylon closure. The population's characteristics are indicated in Table 2.

Operative Procedures

Substantia spongiosa bleeding was controlled by bone wax. The sternal closure technique was the same in all cases, including two threads through the manubrium and four threads through the sternal body. The stainless steel wires (Deknatel No. 5) were twisted first manually and then with a needle holder in some patients. In others, nylon threads (Tevdek No. 5) were tightly knotted. Subcutaneous closure and skin closure were each completed in an uninterrupted fashion.

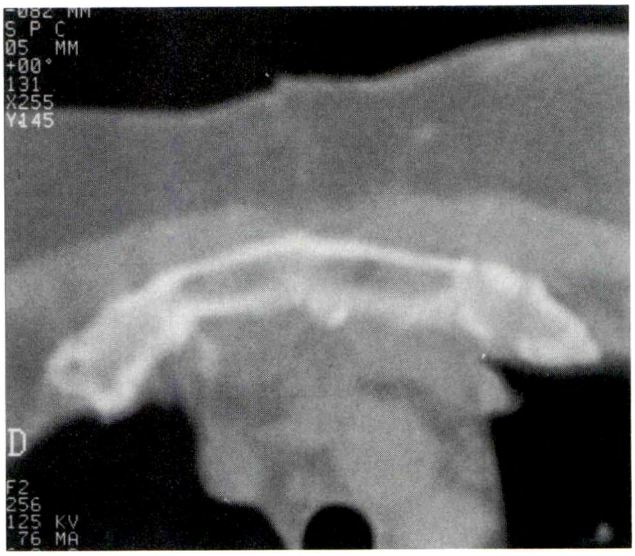

Fig 1. Adequate early sternal approximation at the manubrial level.

Follow-up Methods

EARLY FOLLOW-UP. Each patient was subjected to sternal scanning, including a manubrial and a sternal body print, 3 to 8 days after leaving the intensive care unit. After optical magnification (×6), each print was studied to evaluate the quality of sternal approximation. The evaluation was made as follows: adequate = sternal approximation without spacing or misalignment as illustrated in Figures 1 and 2; inadequate = sternal approximation with spacing or misalignment or both as illustrated in Figures 3 and 4. Spacing was expressed in millimeters.

LATE FOLLOW-UP. Late follow-up (5 ± 1 months) was based on clinical examination of the state of the skin and sternal stability.

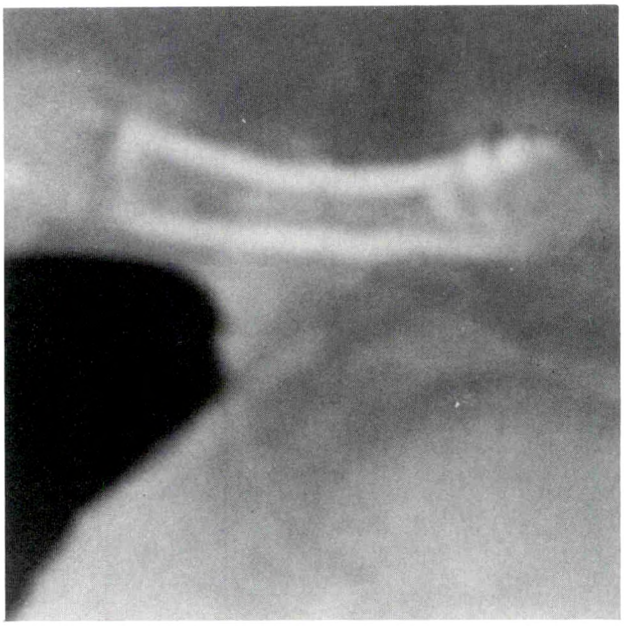

Fig 2. Adequate early sternal approximation at the sternal body level.

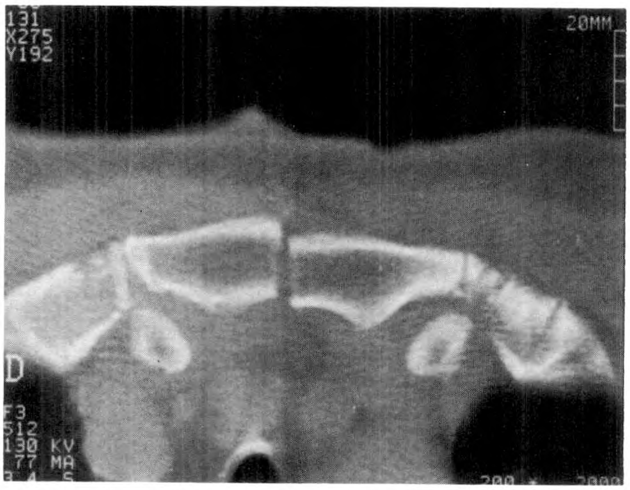

Fig 3. Inadequate early sternal approximation at the manubrial level.

Statistical Methods

The qualitative analysis of the sternal approximation was made by the χ^2 test adjusted with Yate's correcting factor. The results were considered significant at a p level less than 0.05. The nonparametric test of Mann and Whitney was used for the quantitative analysis of the spacing.

Results

Sternal Approximation

No adequate approximation could be found in groups 2 and 4. Adequate approximation was observed in 14.3% and 16.7% of patients in groups 1 and 3, respectively; however, the results were not significant (Table 3). In all groups, the two most frequently encountered abnormalities were manubrial spacing and sternal body misalignment.

No significant relationship could be found between sternal approximation and the type of sternal closure. Moreover, there was no significant influence of ITA harvesting on the sternal approximation. It should be noted that all 5 patients who were reoperated on for sternal wound complications (mediastinitis, 2; sternal dehiscence, 2; and cutaneous disunion, 1) underwent ITA harvesting. Sternal closure was made in 3 of them with nylon threads and in the other 2 with stainless steel wires. One patient in group 2 died of mediastinitis.

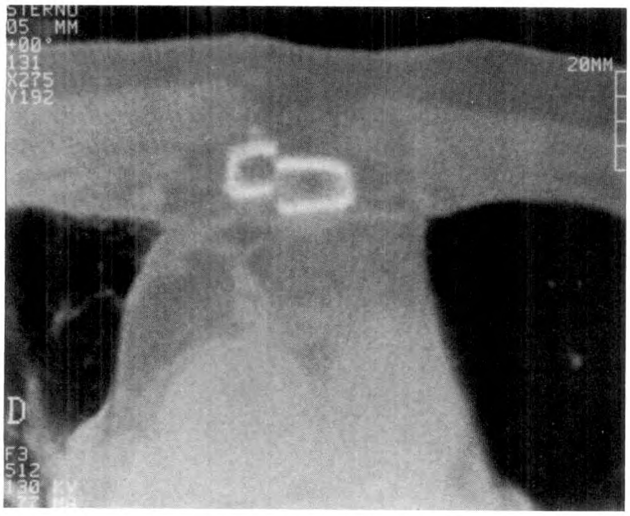

Fig 4. Inadequate early sternal approximation at the sternal body level.

Table 3. General Results[a]

	ITA		SV	
Variable	Steel (n = 21)	Nylon (n = 22)	Steel (n = 12)	Nylon (n = 11)
Adequate Ap	3 (14.3)	0 (0.0)	2 (16.7)	0 (0.0)
Inadequate Ap				
M misalignment	4 (19.0)	7 (31.8)	2 (16.7)	3 (27.3)
M spacing	17 (80.9)	20 (90.9)	8 (66.7)	8 (72.7)
SB misalignment	11 (52.4)	14 (63.6)	6 (50.0)	9 (81.8)
SB spacing	4 (19.0)	8 (36.4)	5 (41.7)	4 (36.4)
Sternal reoperation	2 (9.6)	3 (13.6)	0 (0.0)	0 (0.0)
Death	0 (0.0)	1 (4.6)	0 (0.0)	0 (0.0)

[a] Numbers in parentheses are percentages.

Ap = approximation; ITA = internal thoracic artery; M = manubrial level; SB = sternal body level; SV = saphenous vein.

Sternal Spacing

The largest spacings were found in group 2 at the manubrial (2.5 ± 0.14 mm) and at the sternal body (2.0 ± 0.64 mm) levels, whereas the smallest spacings were observed in group 3 at the manubrial level (1.6 ± 0.18 mm) and in group 4 at the sternal body level (1.1 ± 0.25 mm) (Table 4). However, the difference in the size of spacings between these groups was not significant. Internal thoracic artery harvesting and the type of sternal closure did not significantly influence the size of spacing.

Comment

Problems related to ITA harvesting are usually expressed in terms of sternal devascularization. It is believed [13] that before ITA harvesting, the ITA and its branches provide the only substantial arterial blood supply to the

Table 4. Mean Spacing Values

	Manubrial Spacing		Sternal Body Spacing	
Group	n	Mean ± SEM (mm)	n	Mean ± SEM (mm)
ITA steel (group 1)	17	2.2 ± 0.25	4	1.3 ± 0.30
ITA nylon (group 2)	20	2.5 ± 0.14	8	2.0 ± 0.64
SV steel (group 3)	8	1.6 ± 0.18	5	1.6 ± 0.67
SV nylon (group 4)	8	2.1 ± 0.34	4	1.1 ± 0.25

ITA = internal thoracic artery; SEM = standard error of the mean; SV = saphenous vein.

Table 5. Mean Spacing Values in Patients With Internal Thoracic Artery Harvesting

	Reoperated		Nonreoperated	
Level	n	Mean ± SEM (mm)	n	Mean ± SEM (mm)
M				
Steel	2	2.55 ± 1.4	15	2.16 ± 0.20
Nylon	3	3.27 ± 0.35	18	2.33 ± 0.12
SB				
Steel	1	0.8	3	1.45 ± 0.30
Nylon	3	3.33 ± 1.70	6	1.2 ± 0.15

M = manubrial level; SB = sternal body level; SEM = standard error of the mean.

sternum. After harvesting, this blood supply is destroyed. An anastomosis between perforating branches of the ITA and the thoracoacromial artery has also been described [14]. Should a collateral system be present to provide blood supply to the sternum after ITA harvesting, then the difference in the incidence of complications between ITA and saphenous vein grafting needs to be explained.

According to Culliford and associates [3], sternal wound complications occurred in 1.1% of patients after saphenous vein bypass and in 2.3% and 8.5% of patients after single and bilateral ITA grafting, respectively. Hazelrigg and co-workers [12] have indicated that excluding any other risk factor, the chance of wound complications observed after bilateral ITA grafting is nearly five times greater than that after saphenous vein bypass grafting and three times greater than that after single ITA grafting. These differences could be explained by the direction of the force exerted on the retracted sternum during exposure and dissection of the ITA.

We were surprised by the fact that the 5 patients who were reoperated on for sternal reasons underwent ITA harvesting. We compared the patients who underwent reoperation versus those who did not in groups 1 and 2 with regard to the size of spacing (Table 5). No significant difference could be found between mean spacing values at the manubrial and sternal body levels when the closure was made by stainless steel wires. There was also no significant difference at the sternal body level when the sternum was closed by nylon yarns; however, the difference at the manubrial level was significant ($p < 0.05$) if we accept comparing groups of 3 and 18 patients.

In our opinion, manubrial spacing may also be explained by the weight of the sloping shoulders. It should be noted that during operation, patients are placed on a shoulder roll. When the roll is removed at the end of the procedure, a decrease in sternal spacing is observed. This spacing has the tendency to diminish when both of the patient's shoulders are raised during sternal closure.

Each sternal closure method has its difficulties: stainless steel wires are difficult to manipulate and nylon knots are difficult to tighten.

Like many authors [15, 16], we would also like to emphasize the importance of a strictly median sternotomy, which permits an equal distribution of the forces used to oppose the two sternal edges during sternal closure.

It is important to note that, excluding the eventual influence of ITA harvesting, only 15.1% of the sternal steel closures and none of the sternal nylon closures show an adequate sternal approximation ($0.05 < p < 0.10$). It is evident that this study was made on a small population, and owing to this, we may not draw conclusions but are only able to put forward some ideas. To lower the rate of complications, one should improve the method of sternal closure. One should never forget that sternotomy should always be strictly median, abuse of bone wax should be avoided, and the sternal wound should be meticulously closed.

The present study seems to indicate that sternal approximation is generally inadequate. This can be explained by technical problems such as steel wires that are difficult to manipulate, nylon yarns that are difficult to tighten, the patient's sloping shoulders and position on the shoulder roll, and the assistant's ability when making the sternotomy or the sternal closure. To improve the closing method, we propose that the surgeon should pass three threads through the manubrium especially when using nylon yarns, withdraw the shoulder roll beforehand, elevate both of the patient's shoulders, and maintain the two xyphoid layers in the same plane and in fairly close contact during the tightening of the wires.

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Notice From the Southern Thoracic Surgical Association

The Thirty-eighth Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Grand Cypress, Orlando, Florida, November 7–9, 1991. Members may register upon payment of a $50 fee. There will be a $300 registration fee for nonmember physicians except for guest speakers, speakers on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 7, 1991. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Advance registration forms, hotel reservation cards, and details regarding transportation arrangements will be mailed to Association members. Nonmembers should write to the Secretary-Treasurer, Hendrick B. Barner, MD.

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High Potassium Contents in Organ Preservation Solutions Cause Strong Pulmonary Vasocontraction

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Euro-Collins (ECS) and UCLA-formula organ preservation solutions induced strong vasocontraction in porcine pulmonary arteries when studied in organ baths at temperatures of 37°C and 30°C. At 20°C ECS induced a 30% contraction, but at 6°C no contraction (n = 5) or a weak contraction (n = 1) was elicited. Neither prostaglandin E_1 nor nifedipine caused any significant reduction of the vasocontraction elicited by ECS and UCLA. Krebs solution, enriched with potassium in amounts corresponding to those in ECS (115 mmol/L) or UCLA (30 mmol/L), induced vasocontraction comparing well with those induced by ECS or UCLA, indicating that it is the high potassium content that causes the vasocontraction. In a second experiment lung segments were stored at 4°C for 9 hours in ECS, UCLA, or Krebs solution. Pulmonary arterial segments were then studied in organ baths at 37°C. The choice of preservation solution did not significantly affect the contractile properties of potassium, noradrenaline, or the thromboxane mimic U-46619. To conclude, high potassium contents in organ preservation solutions induce strong pulmonary vasocontraction in lung temperatures greater than 20°C but not in temperatures less than 10°C. These vasocontractions are not significantly reduced by prostaglandin E_1 or nifedipine. We suggest that the initial preservation solution used to cool down the lungs should contain 4 mmol/L or no potassium. When the lung temperature is less than 10°C, a second perfusion might be done, and then a high potassium content (if thought to be essential) will not cause vasocontraction.

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Collins solution, in different modifications [1, 2], is widely used to preserve organs for transplantation. Cooper's group recommends injection of prostaglandin E_1 (PGE_1) into the pulmonary artery to counteract vasocontraction before administering their modified Collins solution to preserve lungs for transplantation [3]. Euro-Collins solution (ECS) contains a high concentration of potassium (Table 1). It is well known from vascular smooth muscle physiology that potassium-rich solutions cause strong vasocontraction, and in a recent study of the human pulmonary artery it was shown that potassium (124 mmol/L) was a stronger vasoconstrictor than noradrenaline [4].

Promising results have been demonstrated with UCLA-formula organ preservation solution (UCLA), compared with ECS, when applied in canine lung transplantation [5]. In UCLA the potassium content is less than in ECS, but it is still seven times higher than the physiological content in blood (see Table 1).

The aim of the present investigation was to evaluate whether ECS or UCLA causes pulmonary artery vasocontraction, and to test how effective PGE_1 and nifedipine (a calcium-channel blocker) are in counteracting such a vasocontraction if present.

Material and Methods

Preparation and Mounting

Lungs from healthy adult pigs were taken within minutes after slaughter. Lung segments were perfused with and immediately put into chilled (4°C) Krebs buffer solution (Krebs; for composition, see Table 1) and brought to the laboratory for immediate investigation (Fig 1).

In a second experiment (group 2), lung segments from other pigs were perfused with and put into chilled (4°C) Krebs, ECS, or UCLA and stored at 4°C for 9 hours before being investigated.

The pulmonary segments were immersed in Krebs solution and the pulmonary arteries were dissected free under an operation microscope. The arteries were divided into ring segments approximately 1 mm in diameter and 1 mm long. Each of the ring segments was suspended between two L-shaped metal holders, one of which was attached to a Grass FTO3C force displacement transducer and in turn connected to a Grass polygraph for continuous recording of isometric tension. The other holder was fixed to a movable unit allowing fine adjustments of vessel tension. The vessel segments were immersed in temperature-controlled (37°C) tissue baths containing 5 mL of Krebs bubbled with 95% O_2 and 5% CO_2 giving a pH of 7.4 (see Fig 1).

The vessels were repeatedly stretched to a basal tension of 4 mN during an equilibration period of approximately 1

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Table 1. Composition of the Preservation Solutions

Component	Krebs	Euro-Collins	UCLA
NaCl (mmol/L)	119	. . .	. . .
$NaHCO_3$ (mmol/L)	15	10	. . .
KCl (mmol/L)	4.6	15	20
NaH_2PO_4 (mmol/L)	1.2	. . .	5
KH_2PO_4 (mmol/L)	. . .	15	. . .
Na_2HPO_4 (mmol/L)	. . .	. . .	45
K_2HPO_4 (mmol/L)	. . .	43	. . .
$MgCl_2$ (mmol/L)	1.2	. . .	. . .
$CaCl_2$ (mmol/L)	1.5	. . .	. . .
Glucose (mmol/L)	11	214	278
Mannitol (mmol/L)	. . .	. . .	13.7
Insulin (U/L)	. . .	. . .	80
Verapamil (mg/L)	. . .	. . .	10
Serum (mL/L)	. . .	. . .	30
pH	7.4	7.0	7.4
mOsm/L	300	529	350
Na^+ (mmol/L)	135.2	10	57
K^+ (mmol/L)	4.6	115	30

hour. Previous experiments have shown this tension to produce optimum responses. The bath fluid was routinely changed every 15 minutes.

In group 1 the length of the vessel segments was 1.02 ± 0.08 mm (mean ± standard deviation; n = 6) and the distance between the metal holders was 1.13 ± 0.21 mm as measured by means of an operation microscope equipped with a scaler. In group 2 the length was 1.43 ± 0.44 mm and the distance was 1.32 ± 0.32 mm (n = 6).

Experimental Procedure

The ring segments were exposed to a buffer solution containing 124 mmol/L of K^+, which induced strong vasocontraction. The effect was then washed out by replacing the bath solution with Krebs. This procedure was repeated until two consecutive contractions varied less than 10%. The maximum contraction was used as an internal standard to which the agonist-induced contractions in group 2 were related.

GROUP 1. The vessel segments were contracted with buffer solutions containing 80 mmol/L and 30 mmol/L of K^+ sequentially. The bath solution was replaced after each contraction with Krebs, which was repeatedly changed for more than 30 minutes, allowing the vessels to stabilize at basal tension. The bath fluid was replaced with either warm (37°C) ECS or UCLA. The contraction thus induced was allowed to stabilize until a steady state was reached. Either nifedipine (3×10^{-12} to 3×10^{-6} mol/L) or PGE_1 (10^{-12} to 10^{-5} mol/L) was then added cumulatively, and the response to each concentration of the relaxing drugs was expressed in percent of the tension obtained at the steady state. One third of the segments with ECS-induced and UCLA-induced contractions were kept as controls where no drug was given.

To simulate the clinical donor situation, PGE_1 (10^{-7} mol/L) was added 1 minute before the addition of ECS in 12 separate individuals at either 37°, 30°, 20°, or 6°C.

GROUP 2. Noradrenaline or U-46619 was added cumulatively until the maximum contraction was reached. The contraction was regarded as maximum when two subsequent concentrations gave a response of the same amplitude, or when the subsequent concentration induced a decreased contraction amplitude. The maximum contractile force, E_{max}, was expressed in percent of the contraction induced by 124 mmol/L K^+. The potency of each agonist was expressed as the pEC_{50} value, ie, the negative logarithm of the EC_{50} value, determined graphically from each curve as the concentration giving half the maximum contraction.

Buffer Solutions

The contents of the Krebs, ECS, and UCLA are shown in Table 1. The K^+-rich buffer solutions were prepared by substituting all (124 mmol/L) or part (30 mmol/L or 80 mmol/L) of the NaCl in the Krebs solution with KCl.

Drugs

Nifedipine (0.1 mg/mL; Bayer, Leverkusen, Germany) was provided in ampoules, dissolved in a mixture of

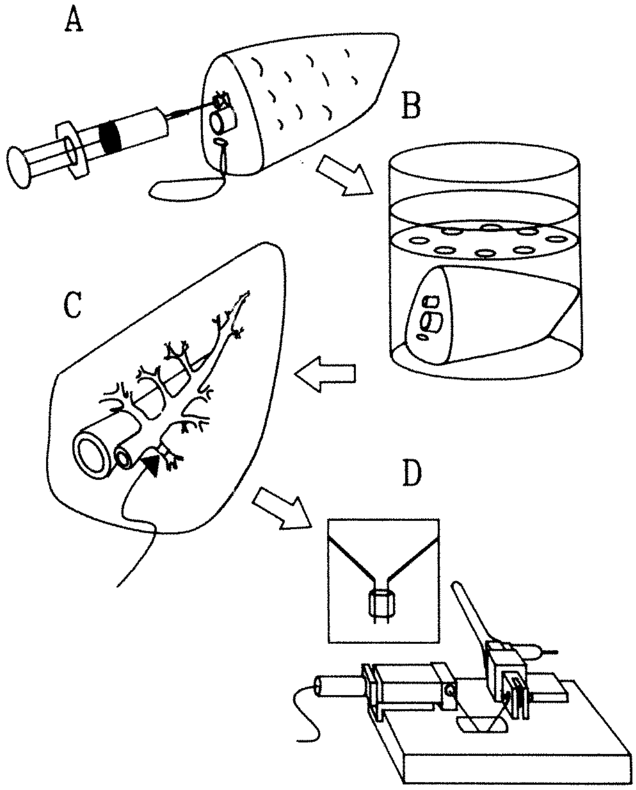

Fig 1. After perfusion with Krebs, Euro-Collins, or UCLA solution (A) the lung segments were kept under the surface of the respective preservation solution at 4°C (B). The pulmonary artery was dissected under the surface of the solution (C) and 1 × 1-mm ring segments were suspended between metal holders in organ baths for recording of the isometric tension (D).

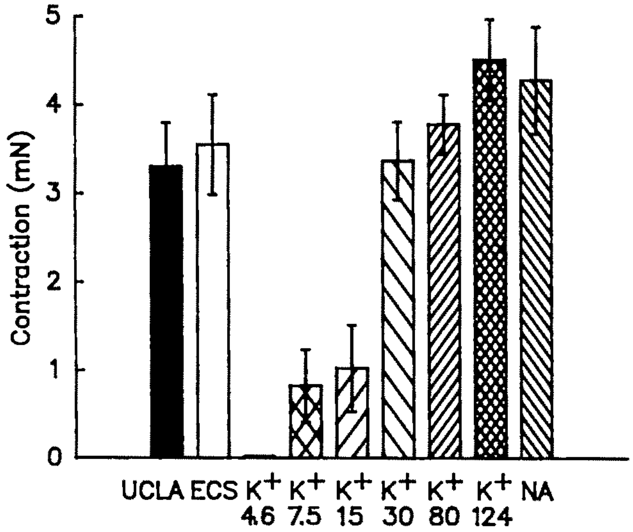

Fig 2. The contractile response in porcine pulmonary arteries exposed to UCLA, Euro-Collins (ECS), or buffer solutions containing various amounts of K^+ (in mmol/L) and the maximum effect obtained with noradrenaline (NA) are shown (mean ± standard error of the mean; n = 6).

ethanol (150 mg/mL) and polyethylene glycol (150 mg/mL) and protected from light until use. (−)-Noradrenaline hydrochloride was purchased from Sigma Chemical Co (St. Louis, MO). Stock solutions of PGE_1 and U-46619 (Upjohn, Kalamazoo, MI) were prepared in absolute ethanol and stored in −20°C. Just before use the prostanoids were diluted in phosphate buffer at neutral pH, and the other drugs in 0.9% NaCl with 1.0 mmol/L ascorbic acid.

Statistics

Student's *t* test for unpaired data was used for statistical comparisons, and a probability level less than 0.05 was regarded as significant.

Results

Group 1

The contractile responses elicited by the different preservation solutions are shown in Figure 2. UCLA induced a contraction of 3.30 ± 0.49 mN. The buffer solution with equimolar amounts of K^+ (30 mmol/L) induced tension of 3.36 ± 0.44 mN. Euro-Collins solution (115 mmol/L K^+) induced a contraction of 3.55 ± 0.56 mN (mean ± standard error of the mean) compared with the 3.77 ± 0.33 mN elicited by the buffer solution with 80 mmol/L of K^+ and the 4.50 ± 0.45 mN elicited by 124 mmol/L of K^+. The contraction induced by ECS was followed by relaxation within 15 minutes, and after 60 minutes the contraction was at a steady level of about 20% of the maximum contraction (Fig 3). The contraction induced by UCLA was followed by relaxation within 40 minutes and was at a level of about 50% of the maximum contraction after 1.5 to 2 hours. The buffer solution containing 80 mmol/L K^+ maintained a stable contraction for 2 hours, whereas the buffer solution containing 30 mmol/L of K^+ caused a contraction that slowly increased after being stable for 20 minutes (Fig 4).

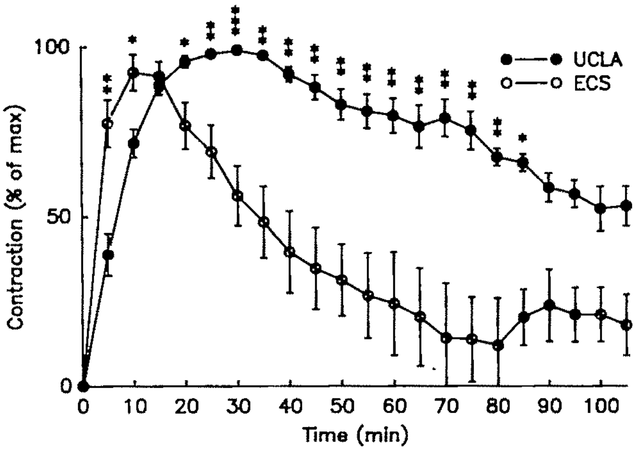

Fig 3. The contractile response (mean ± standard error of the mean) to UCLA and Euro-Collins solution (ECS) is expressed in percent of the initial maximum contraction. The abscissa denotes the time elapsed after exposure to the solutions. ($p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.)*

The effects of nifedipine and PGE_1 on contractions induced by ECS and UCLA are shown in Figure 5. In the controls the vessel tension slowly decreased, and increasing concentrations of nifedipine and PGE_1 induced no significant further relaxation.

The effects of nifedipine and PGE_1 in vessels precontracted with the buffer solutions containing 30 or 80 mmol/L of K^+ are shown in Figure 6. Nifedipine in concentrations of 3×10^{-10} mol/L or greater significantly reduced the K^+-induced contractions in both types of buffer solution, whereas PGE_1 in concentrations of 10^{-9} mol/L or greater had the same effect only in the buffer solution containing 30 mmol/L K^+.

When PGE_1 was administered 1 minute before ECS, the vasocontraction induced by ECS at 37°C (4.66 ± 1.31 mN;

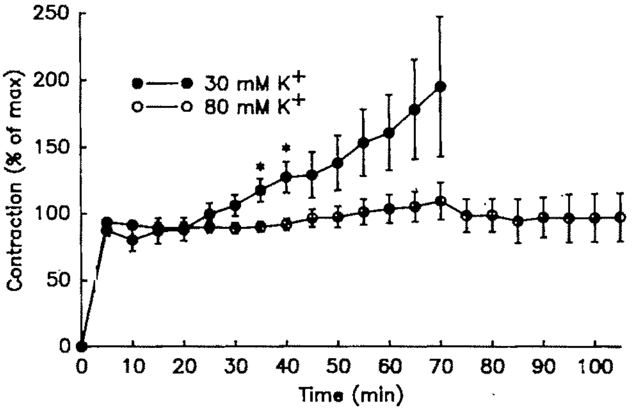

Fig 4. The contractile response (mean ± standard error of the mean) to buffer solutions containing 30 and 80 mmol/L K^+ is expressed in percent of the initial maximum contraction. The abscissa denotes the time elapsed after exposure to the solutions. ($p < 0.05$.)*

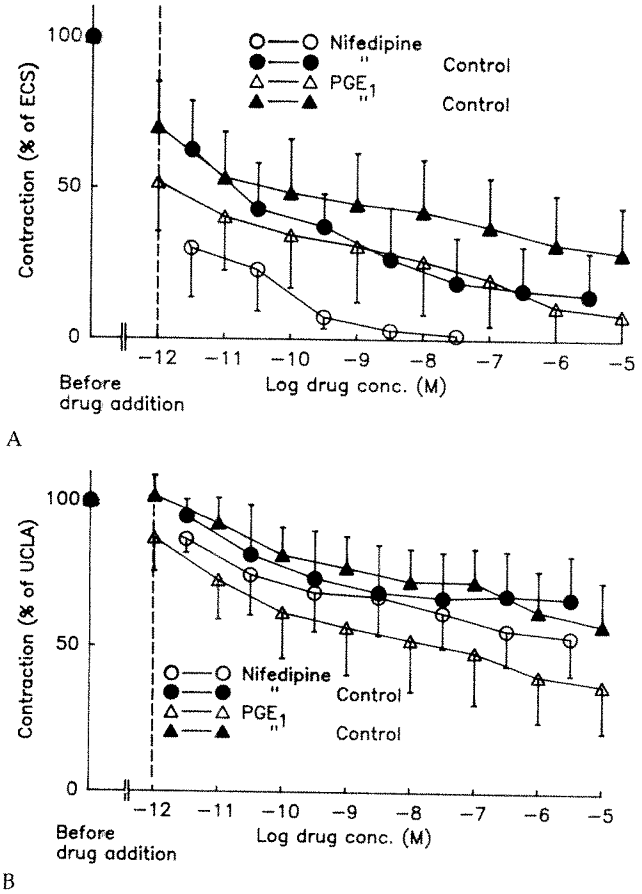

Fig 5. The effects of the cumulative addition of nifedipine and prostaglandin E_1 (PGE$_1$) *to vessels exposed to either (A) Euro-Collins or (B)* UCLA *solution.* Filled symbols *denote the contraction level in vessels where no drug was given, at the same time interval after exposure to the solutions.*

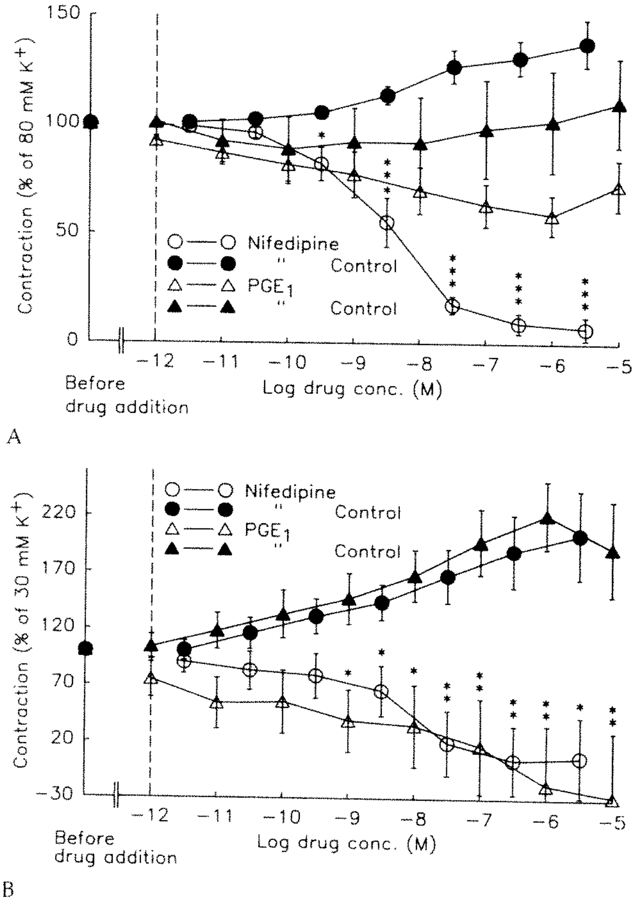

Fig 6. The effects of the cumulative addition of nifedipine and prostaglandin E_1 (PGE$_1$) *to vessels exposed to a buffer solution containing either (A) 80 mmol/L of K$^+$ or (B) 30 mmol/L of K$^+$.* Filled symbols *denote the contraction level in vessel segments where no drug was given, at the same time interval after exposure to the solutions.* (* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.)

mean ± standard error of the mean; $n = 6$) was not significantly different from when ECS was given alone (3.55 ± 0.56 mmol/L).

The contractile force developed by the exposure to 37°, 30°, 20°, and 6°C ECS is shown in Figure 7. At 6°C 5 of the 6 individuals showed no contraction at all.

Group 2

There was no significant difference between segments stored in Krebs, ECS, or UCLA in their response to either K$^+$, noradrenaline, or the thromboxane mimic U-46619 (Table 2; Figs 8, 9).

Comment

In our clinical and experimental lung transplantation program we use the preservation technique described by Egan, Kaiser, and Cooper [3]; prostaglandin E_1 (500 μg) is injected into the pulmonary artery over 60 seconds, just before inflow occlusion and cross-clamping of the aorta. Cold (4° to 6°C) ECS (50 mL/kg body weight) is used to flush the lungs; the perfusate runs in under low pressure (15 mm Hg) by adjusting the level of the bag. The tip of the left atrial appendage is excised and a cut is made in the inferior vena cava to allow for venting of the left and right heart. Both pleural spaces are flooded with cold saline

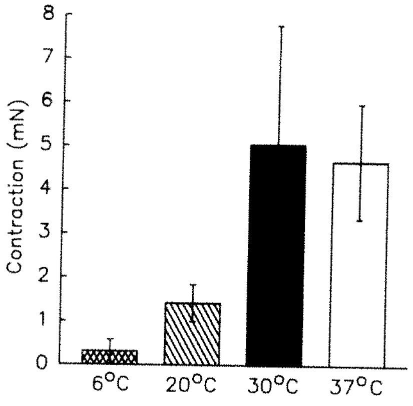

Fig 7. The contractile forces (in mN; mean ± standard error of the mean; $n = 6$) induced by Euro-Collins solution at different temperatures. Prostaglandin E_1 (10^{-7} mol/L) was added 1 minute before the vessels were exposed to Euro-Collins solution.

Table 2. Contractile Capacity After 9 Hours of Preservation[a]

		Noradrenaline		U-46619	
Solution	K^+ (mN)	E_{max} (%)	pEC_{50}	E_{max} (%)	pEC_{50}
Krebs	3.4 ± 0.8	134 ± 28	6.5 ± 0.3	247 ± 37	8.2 ± 0.3
Euro-Collins	5.1 ± 0.8	99 ± 11	6.7 ± 0.3	175 ± 22	8.9 ± 0.4
UCLA	6.4 ± 1.4	65 ± 14	6.7 ± 0.4	190 ± 30	8.6 ± 0.5

[a] The contractile capacity of 124 mmol/L K^-, noradrenaline, and thromboxane-mimic U-46619 after 9 hours of storage in the different solutions. E_{max} denotes the maximum contraction induced, expressed in percent of the contraction elicited by a buffer solution containing 124 mmol/L K^+. All values are given as mean ± standard error of the mean; n = 6.

solution, and the lungs are ventilated (100% O_2) during the pulmonary artery flush to facilitate uniform distribution of the perfusate.

When we perform this preservation procedure in adult pigs with temperature probes placed superficially and deeply into the lungs, the lung temperature drops to about 30°C after 2 minutes of perfusion, and when all the ECS (50 mL/kg) has run in (within 5 minutes) the lung temperature has fallen to 18° to 20°C. The lungs are then excised and placed under cold (4° to 6°C) saline solution. The lung temperature gradually falls to 4° to 6°C over a period of about 1 hour.

In the present study we have therefore chosen the following temperatures for our organ bath studies: 37°C (ie, the temperature in the lungs during the initial perfusion), 30°C (the lung temperature after about 2 minutes of perfusion), 20°C (the lung temperature when all the perfusate has been given), and 6°C (the final lung temperature during the storage period). When all the blood has been washed out of the lungs (ie, when about one third of the perfusate has passed through the lungs and the lung temperature is around 30°C) we have a situation similar to the in vitro conditions in our organ baths, except that the donor lungs are ventilated with 100% O_2 whereas the organ baths are bubbled with 95% O_2 and 5% CO_2. Ideally, the donor lungs should be ventilated with 95% O_2 and 5% CO_2 to give a physiological intraalveolar carbon dioxide tension; ventilating blood-free lungs without CO_2 will lower the intraalveolar carbon dioxide tension, which might be injurious to the alveolar cells [6].

Our study indicates that high K^+ contents in lung preservation solutions will cause strong pulmonary vasocontraction. This is not surprising because physiologists for a long time have been using K^+-rich buffer solutions to induce "maximum" vasocontraction to serve as an internal standard to which the vasocontracting capacity of different drugs can be related. The vasocontraction induced by 30 mmol/L K^+, which equals the K^+ content in the UCLA formula, did not differ significantly from the effects of 124 mmol/L of K^+. Concentration-response experiments with K^+ have been performed [7] showing that about 90% of the maximum contractile effect of K^+ is reached already around 60 mmol/L. This is in accordance with the findings in the present study (see Fig 2).

Prostaglandin E_1 is at present regularly used for vasodilation either before infusion of the preservation solution or in the solution itself [3, 8–10]. In this study, however, PGE_1 was not able to significantly counteract the vasocontraction induced by the preservation solutions. Nifedipine, which has previously been shown to be a potent vasodilator on precontracted blood vessels [11], seemed to be more effective than PGE_1 in ECS, although this effect was not significantly different from the controls (see Fig 5A).

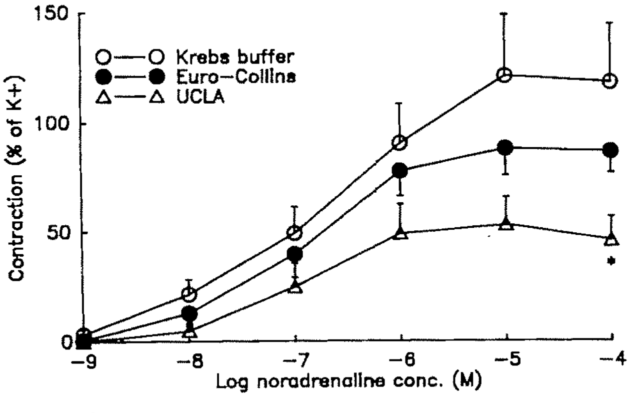

Fig 8. The concentration-response curves (mean ± standard error of the mean; n = 6) of noradrenaline in porcine pulmonary artery after 9 h storage in Krebs, Euro-Collins, or UCLA solution. The contractile responses are expressed in percent of the preceding K^+ (124 mmol/L)-induced contraction. ($p < 0.05$.)*

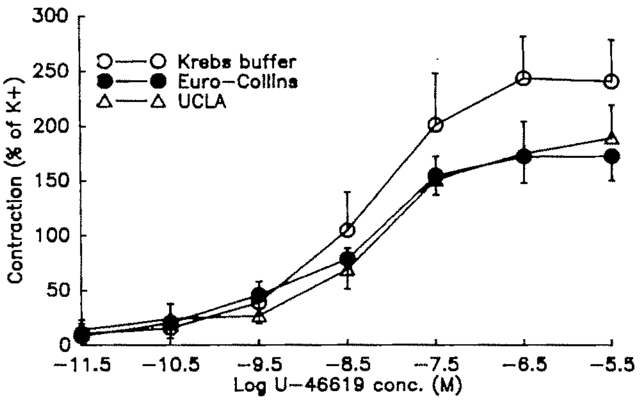

Fig 9. The concentration-response curves (mean ± standard error of the mean; n = 6) of thromboxane A_2-mimic U-46619 in porcine pulmonary artery after 9 hours of storage in Krebs, Euro-Collins, or UCLA solution. The contractile responses are expressed in percent of the preceding K^+ (124 mmol/L)-induced contraction.

As seen from Figure 7, the vasocontraction caused by ECS diminished with the decrease in temperature. At 6°C ECS caused no contraction at all in 5 individuals and a weak contraction in 1. A logical way to deal with the K^+-induced vasocontraction during lung preservation should therefore be to use a potassium-free solution for the initial perfusion. When the lung is cold, potassium loses its capacity to elicit vasospasm. If it is thought to be important for the lung preservation to use a solution with an intracellular electrolyte composition (ie, with a high K^+ content), this solution could be given as a second perfusion when the lung temperature is low.

In this study the contractile effect of noradrenaline and U-46619, a thromboxane A_2-mimic, was investigated after 9 hours of storage of the lungs in the three preservation solutions. The contractile effect of noradrenaline was reduced after storage in UCLA, although not to statistical significance. This is most likely an effect of the calcium-channel blocker verapamil, which has been shown to have α-antagonistic properties as well [12–15]. The reason why UCLA keeps the vessels in a contractile state longer than ECS might be the Ca^{2+} content in the serum in the UCLA (ECS has no Ca^{2+}); the buffer solutions containing 30 and 80 mmol/L of K^+ and physiological amounts of Ca^{2+} showed no signs of relaxation with time (see Fig 4).

To conclude, the high potassium contents in the organ preservation solutions used in the present study induce strong vasocontraction. If not adequately abolished this vasocontraction might lead to a less than perfect organ perfusion and core cooling of the lungs. Core cooling of the lungs is probably of special importance, because the airy, spongelike lungs are difficult to cool down by surface cooling. An inhomogeneous perfusion, because of vasocontraction, might furthermore inadequately preserve some lung cells [16]. Low temperature (but neither nifedipine nor PGE_1) effectively eliminates the vasospasm induced by ECS or UCLA when used for lung preservation.

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Prophylaxis of Atrial Fibrillation With Magnesium Sulfate After Coronary Artery Bypass Grafting

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Ninety-nine consecutive consenting patients were prospectively entered into a randomized, double-blind, placebo-controlled trial to determine the efficacy of postoperative magnesium therapy on the incidence of cardiac arrhythmias after elective coronary artery bypass grafting. No patient had documented or suspected arrhythmias preoperatively. Forty-nine patients received 178 mEq of magnesium given over the first 4 postoperative days, and 50 patients received only placebo. The clinical characteristics of both groups were similar. The preoperative mean serum magnesium concentration was similar in both study (1.90 mEq/L) and placebo (1.90 mEq/L) groups. The mean postoperative serum magnesium concentration in study patients was significantly elevated over postoperative days 1 through 4 when compared with preoperative levels ($p < 0.001$). The postoperative mean serum magnesium concentration in control patients declined and remained significantly depressed through postoperative day 3 ($p < 0.001$), but increased to preoperative levels by postoperative day 4. The mean serum magnesium concentration was significantly greater in the study patients as compared with the control patients over postoperative days 1 through 4 ($p < 0.001$). Although there was no significant difference between groups with respect to episodes of ventricular arrhythmias, there was a significant decrease in the number of episodes of atrial fibrillation in the group receiving magnesium therapy ($p < 0.02$). There were no recognized adverse effects of magnesium therapy. Prophylactic magnesium administration seems to lessen the incidence and severity of atrial fibrillation after coronary artery bypass grafting.

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Supraventricular tachyarrhythmias occur commonly after coronary artery bypass grafting. A variety of pharmocologic agents have been used to prevent the occurrence of supraventricular tachyarrhythmias including propranolol [1], timolol [2], digoxin [3], and verapamil [4]. Magnesium administration has proved beneficial in preventing ventricular and supraventricular tachyarrhythmias after acute myocardial infarction [5–7], and although hypomagnesemia has been reported after cardiopulmonary bypass [8–10], there is little information regarding the use of magnesium to prevent supraventricular tachyarrhythmias after coronary artery bypass grafting. This study was undertaken to determine whether prophylactic postoperative magnesium administration can reduce the incidence of supraventricular tachyarrhythmias after coronary artery bypass grafting.

Material and Methods

This study was approved by the Human Subjects Committee of St. Thomas Hospital, Nashville, Tennessee. After giving informed consent, 99 consecutive consenting patients undergoing elective first-time coronary artery bypass grafting were prospectively randomized in a double-blind fashion to receive postoperative magnesium (49 patients) or placebo (50 patients). Excluded from the study were patients with a serum creatinine level greater than 25 mg/L (2.5 mg/dL), those with a history of second- or third-degree heart block, patients with a permanent pacemaker, and those refusing to participate. Patients with any documented or suspected supraventricular or ventricular arrhythmias, including isolated atrial or ventricular premature depolarizations noted on preoperative surface electrocardiography, were excluded as well. All patients underwent left heart catheterization and selective coronary arteriography because of progressive angina pectoris and were referred for surgical myocardial revascularization. Patients requiring additional procedures, such as valve replacement or left ventricular aneurysmectomy, were excluded.

Patients were randomly assigned to receive in a double-blind fashion either (1) placebo group maintenance fluids (5% dextrose in water with 20 mEq of potassium chloride per liter) or (2) study group maintenance fluids (5% dextrose in water with 20 mEq of potassium chloride per liter plus 40 mEq of magnesium, as magnesium sulfate, per liter). These intravenous fluids were given as a continuous infusion at 100 mL/h over the first 24 hours postoperatively followed by 25 mL/h over postoperative hours 25 through 96. The study group, therefore, received 96 mEq of magnesium over the first 24 hours postoperatively (4 mEq/h) followed by 72 mEq over postoperative hours 25 though 96 (1 mEq/h).

All patients had determination of serum magnesium and potassium levels preoperatively, immediately postop-

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eratively, and on postoperative days 1 through 4. Supplemental potassium was given as needed to maintain a serum potassium level greater than 4 mEq/L.

For the purposes of defining end points in this study, postoperative cardiac arrhythmias were defined as: (1) ventricular fibrillation, (2) ventricular tachycardia (greater than 3 premature ventricular contractions in a row at a rate greater than 100 beats/min), (3) premature ventricular contractions greater than 5 per minute, multiformed, couplets, or R on T phenomenon, and (4) atrial fibrillation or flutter and supraventricular tachycardia at rates greater than 110 beats/min lasting greater than 30 seconds. Frequent premature atrial contractions were not treated unless deemed hemodynamically significant. Using these definitions, cardiac arrhythmias were treated under the direction of the attending surgeon. All patients continued the study protocol regardless of the arrhythmia noted or the treatment received. Although the patient's physicians were not blinded to the results of the serum magnesium testing, no patient received additional magnesium therapy. Each episode of arrhythmia was recorded, and for patients with a persistent arrhythmia lasting more than 24 hours, each new 24-hour period was recorded as an additional episode.

All operations were performed using hypothermic cardiopulmonary bypass with a crystalloid, non-magnesium–containing prime and a membrane oxygenator. Multidose blood cardioplegia with topical hypothermia was used in 82 patients. The cardioplegic component contained no additional magnesium. In these 82 patients (39 study, 43 control), standard surgical techniques were used with all distal coronary anastomoses performed during a single period of aortic cross-clamping. Proximal vein graft anastomoses were performed with partial aortic occlusion during rewarming. The left internal thoracic artery was used as a bypass conduit in the majority of patients. Seventeen patients (10 study, 7 control) underwent myocardial revascularization using hypothermic intermittent ischemic arrest as described by Olinger [11].

All patients underwent careful intraoperative and postoperative hemodynamic monitoring with arterial, central venous, and pulmonary artery pressure monitoring with thermodilution cardiac output determination. Atrial, atrioventricular sequential, or ventricular pacing in the early postoperative period was used as required for bradycardia. Patients receiving β-blockers had these medications continued until the day of operation. No patient received β-blockers, calcium channel blockers, or digitalis postoperatively unless a specific arrhythmia dictated their use. Lidocaine was not routinely used postoperatively unless specifically indicated for the treatment of a postoperative ventricular arrhythmia. Postoperative hypertension was managed with intravenous nitroglycerin or nitroprusside, or both. Inotropic or pressor support was used to maintain a mean arterial pressure greater than 70 mm Hg and a cardiac index greater than 2 L $\cdot$ min^{-1} $\cdot$ m^{-2}.

Apart from the study protocol involving the magnesium infusion or placebo, each patient's postoperative management was directed by a single group of cardiovascular surgeons and anesthesiologists at this institution. All patients underwent continuous electrocardiographic monitoring through postoperative day 4 using the Mennen Horizon 2000 Bedside Arrhythmia Detection System (Mennen Medical Co, Clarence, NY). At the time of termination of the study, the code was broken and the patient data were assembled into placebo and magnesium groups. The incidence of postoperative arrhythmias was analyzed for group differences by using a two-sided χ^2 analysis. The mean serum magnesium levels (both within and between the control and study groups) and the mean number of episodes of arrhythmias between groups were analyzed using a paired or two-sample t test where appropriate.

Table 1. Patient Characteristics[a]

Variable	Study Group	Control Group
N	49	50
Sex (M/F)	35/14	39/11
Age (y)	59 (43–75)	62 (42–79)
History of MI	17	21
β-Blockers	13	13
Ca^{2+} Blockers	24	22
Diuretics	5	10
Digitalis	0	7
LVEF	0.50 (0.30–0.70)	0.49 (0.25–0.70)
XC time (min)	66 (28–167)	66 (18–135)
No. of grafts	4.0 (2–7)	3.6 (1–7)

[a] Values in parentheses are ranges.

LVEF = left ventricular ejection fraction; MI = myocardial infarction; XC = aortic cross-clamp.

Results

Patient demographics are assembled in Table 1. The two groups were well matched with respect to age, sex, and history of prior myocardial infarction. There was no significant difference between groups with respect to β-blocking or calcium channel blocking medication, although preoperative digoxin and diuretic use was more common in the control group. Left ventricular systolic function was similar between groups as were operative variables, including ischemic time, number of grafts, and the method of myocardial protection.

There was one death in the study period occurring in a 79-year-old man 4 days after two-vessel coronary bypass grafting due to recurrent ventricular tachycardia degenerating to asystole from which he could not be resuscitated. This patient was in the control group. Serum magnesium level in this patient on the day of death was 2.0 mEq/L. Permission for autopsy was denied. There were four postoperative myocardial infarctions as determined by new Q waves, compatible clinical course, and increase in creatine kinase-MB level, with two in each group. Seventeen of 49 study and 27 of 50 control patients received

Table 2. Mean Serum Magnesium Levels[a]

		Postoperative Day				
Group	Preop	0	1	2	3	4
Study	1.90 ± 0.03	1.27 ± 0.03	2.44 ± 0.05	2.30 ± 0.05	2.20 ± 0.04	2.08 ± 0.05
Control	1.90 ± 0.04	1.17 ± 0.03	1.25 ± 0.03	1.44 ± 0.04	1.75 ± 0.04	1.85 ± 0.04

[a] Values are expressed in milliequivalents per liter ± standard error of the mean.

inotropic or pressor support postoperatively to maintain a mean arterial pressure greater than 70 mm Hg and a cardiac index greater than 2 L · min^{-1} · m^{-2} during the first 18 postoperative hours. Forty study and 31 control patients received nitroprusside for hypertension. Intravenous nitroglycerin was used routinely in both groups.

The mean serum magnesium concentrations for the two groups are shown in Table 2. A comparison of the changes in serum magnesium concentration over time for the two groups is shown in Figure 1. There was no difference between groups in the preoperative mean serum magnesium concentration. In both the study and control group, there was a significant decline in the mean serum magnesium concentration immediately after coronary artery bypass grafting ($p < 0.001$). Compared with preoperative levels, the mean serum magnesium concentration was significantly elevated in study patients on postoperative days 1 through 4 ($p < 0.001$).

In control patients, the mean serum magnesium concentration remained depressed as compared with preoperative levels on postoperative days 1 through 3 ($p <$ 0.001) but returned to preoperative levels by postoperative day 4. The mean serum magnesium concentration in study patients was significantly greater than that of controls on postoperative days 1 through 4 ($p < 0.001$).

The episodes of the various dysrhythmias are shown in Figure 2. There were no significant differences between groups for the various ventricular arrhythmias. The difference between groups with respect to the numbers of patients with atrial fibrillation did not reach statistical significance; however, significantly fewer episodes of atrial fibrillation occurred in the group receiving supplemental magnesium. Seven patients had 12 episodes of atrial fibrillation in the study group as compared with 14 control patients experiencing 42 episodes ($p < 0.02$). Nine of these 14 control patients experienced persistent or recurrent atrial fibrillation requiring multiple pharmacologic interventions, and 2 of these 9 required synchronized direct-current cardioversion because of hemodynamic embarrassment associated with the atrial fibrillation. All 9 were eventually discharged receiving oral digoxin. Additionally, 7 required oral verapamil and 3 patients required oral quinidine for control of the atrial fibrillation.

By comparison, only 2 patients in the study group experienced more than one episode of atrial fibrillation. No patient in the study group required cardioversion.

Two of 17 patients undergoing coronary revascularization using intermittent ischemic arrest experienced atrial fibrillation (both in the control group). Four of 7 control patients receiving preoperative digoxin experienced atrial fibrillation. The number of patients receiving preoperative β-blockers in whom postoperative atrial fibrillation developed was similar in both groups (4 study, 3 control).

No side effects could be attributed to the magnesium therapy. As noted previously, atrial and ventricular pacing was used as required and no patient in this study had artificial pacing requirements beyond 36 hours. There was no difference in pacing requirements between the two groups. The study code was not broken throughout the study.

Comment

Magnesium is the second most abundant intracellular cation and is an essential cofactor of the myocardial cell

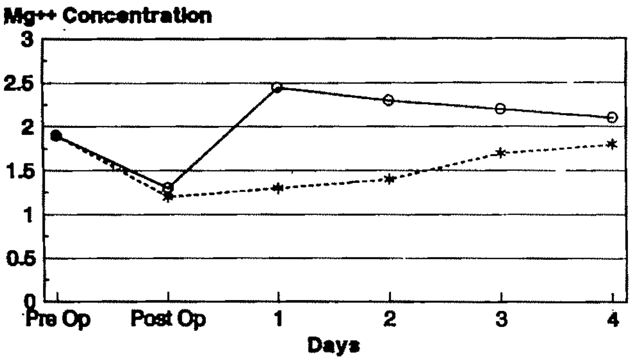

Fig 1. Magnesium concentration (mEq/L). (Solid line = *study patients;* dotted line = *control patients.)*

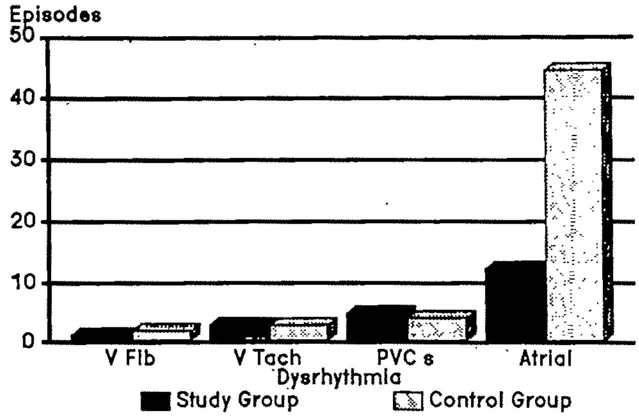

Fig 2. Episodes of dysrhythmia after coronary artery bypass grafting. (Atrial = *atrial fibrillation;* PVCs = *premature ventricular contractions;* V Fib = *ventricular fibrillation;* V Tach = *ventricular tachycardia.)*

membrane associated enzyme sodium-potassium adenosinetriphosphatase, which maintains normal transmembrane sodium and potassium gradients. Magnesium deficiency may adversely affect this ionic pump, impairing the ability of the myocardial cell to maintain normal intracellular potassium concentrations, leading to alterations in membrane potential, potassium conductance, and repolarization, and may thereby be potentially arrhythmogenic [12]. Hypomagnesemia is seen in a variety of clinical states and is commonly noted after cardiopulmonary bypass [8–10]. Although in vitro data exist to support a potential arrhythmogenic influence of hypomagnesemia [13], in vivo evidence is inconclusive and based on testimonials to arrhythmia suppression by the administration of magnesium salts [14]. This arrhythmia suppression with magnesium is, however, nonspecific and does not prove that hypomagnesemia induced the arrhythmia. Nonetheless, numerous uncontrolled observations do support the contention that exogenous magnesium suppresses a variety of tachyarrhythmias [15–19].

In humans it has been difficult to separate the effects of magnesium deficiency from other associated electrolyte abnormalities, especially potassium deficiency. As a result, much of our understanding of the cardiovascular effects of magnesium are derived from in vitro observations and the in vivo effects of exogenous magnesium administration.

Detailed electrophysiologic studies in humans [20–23] have examined the effects of magnesium on cardiac conduction. In these studies, patients have differed in baseline conduction abnormalities, presence of discernible heart disease, and degree of induced hypermagnesemia. Magnesium therapy did, however, induce consistent prolongation of atrioventricular conduction with PR interval prolongation seen on the surface electrocardiogram and AH interval prolongation detected by intracardiac electrograms. In general, there was little effect on ventricular conduction or refractoriness. These studies provide some rationale for the potential effectiveness of magnesium in the treatment of some cardiac arrhythmias, particularly supraventricular tachyarrhythmias.

Most reports attesting to the effectiveness of magnesium therapy in the treatment of cardiac arrhythmias are case reports or collections of case reports. Recently, however, a few controlled studies have shown a decrease in tachyarrhythmias after acute myocardial infarction in patients treated with magnesium as compared with controls [5–7].

These observations in the reported literature and the frequent development of supraventricular tachyarrhythmias and hypomagnesemia after coronary artery bypass prompted this study. The results of this study confirm previous observations of significant hypomagnesemia after cardiopulmonary bypass [8–10]. All patients experienced a significant decrease in serum magnesium level, which is probably explained by hemodilution and possibly a β-receptor–mediated phenomenon secondary to elevated epinephrine levels commonly noted after cardiopulmonary bypass [13, 24]. In the control group, the mean serum magnesium decreased to its nadir immediately postoperatively but then gradually increased to preoperative levels by the fourth postoperative day. By this protocol, the mean serum magnesium concentration in the study group was significantly elevated over the first 4 postoperative days. No patient had a serum magnesium concentration greater than 3.8 mEq/L, and there were no recognized adverse effects to the drug.

Although there was no significant difference between groups with respect to the numbers of patients with atrial or ventricular tachyarrhythmias, there was a significant decrease in the number of episodes of atrial fibrillation in the study group. In addition, these dysrhythmias, when they occurred in the study group, were well tolerated and more easily treated as reflected by the more frequent requirement for cardioversion and multiple drug interventions in the control group. There was, however, no demonstrable difference in the duration of hospital stay between groups reflecting the usually benign nature of supraventricular arrhythmias after coronary artery bypass grafting [25].

Interestingly, the 7 control patients receiving preoperative digoxin were afforded no protection from postoperative supraventricular arrhythmias, as 4 of 7 patients experienced 18 episodes of atrial fibrillation. Abrupt discontinuation of β-blockers in the postoperative period did not seem to influence these results. Four of 13 study patients and 3 of 13 control patients receiving preoperative β-blockers had development of atrial fibrillation in the postoperative period (seven episodes in each group). Likewise, the method of myocardial protection did not seem to influence these results. Although conceivably atrial anoxia during ischemic arrest periods could potentiate postoperative supraventricular arrhythmias, this was not observed. Only 2 of 17 patients (both control) had postoperative atrial fibrillation. Others have noted that the incidence of atrial arrhythmias is similar whether the method of myocardial protection is cold potassium cardioplegia or intermittent aortic occlusion [26].

Although the monitoring system used was not a Holter tape recording system, it automatically records bradyarrhythmias or tachyarrhythmias, allowing analysis of the arrhythmia. It is unlikely that any arrhythmias as defined by the end points in this study were missed.

When this study was initiated there were no reports on the use of magnesium in patients after cardiopulmonary bypass. Extrapolating from the existing literature, this study was based on the protocol used by Rasmussen and associates [7], which demonstrated a reduced incidence of supraventricular arrhythmias in patients with acute myocardial infarction. Admittedly this protocol is somewhat cumbersome, requiring a 4-day continuous infusion, and clearly further studies should be done both to confirm these results and to simplify the method of magnesium administration.

The observation of increased requirements for inotropes and vasopressors in control patients cannot be easily explained. The patient groups were similar with respect to measurable variables, the study was blinded, and postoperative hemodynamic management was directed by a single group of cardiovascular surgeons and anesthesiologists with a consistent philosophy regarding the use of inotropes and vasopressors. Conceivably the decreased incidence of postoperative supraventricular arrhythmias attributed to magnesium replacement could be related to increased catecholamine use in the control group. Alternatively, the decreased requirements for inotropes in the study group could be related by some undefined mechanism to magnesium administration. These two alternative explanations are speculative and should be addressed by further study.

It should be stressed that the observations in this study should not be overinterpreted. They do not prove that hypomagnesemia is the cause of supraventricular arrhythmias after coronary artery bypass grafting, but only that supplemental magnesium seems to lessen their incidence and severity. Additional studies are needed to confirm these observations and, more importantly, to establish the mechanism of the antiarrhythmic properties of magnesium.

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Bezold-Jarisch Reflex in Postoperative Pediatric Cardiac Surgical Patients

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The Bezold-Jarisch reflex is an inhibitory reflex that originates from the heart, is mediated by the vagus nerve, and is manifested by hypotension and bradycardia. We present 4 pediatric cardiac surgical patients, aged 1 day to 9 months, who exhibited cardiovascular collapse in their early postoperative course. In each patient, cardiovascular deterioration was marked by an insidious decrease in arterial blood pressure without an associated change in heart rate, central venous pressure, or airway pressure. Bradycardia followed the decrease in blood pressure. The Bezold-Jarisch reflex was suspected and atropine was administered, first as a bolus injection at 0.01 mg/kg, and later, as a continuous infusion at 0.01 mg $\cdot$ kg^{-1} $\cdot$ h^{-1}. Atropine prevented recurrent episodes of hypotension and bradycardia. We believe the Bezold-Jarisch reflex is more prevalent than previously suspected in postoperative pediatric cardiac surgical patients.

(Ann Thorac Surg 1991;52:534–6)

The Bezold-Jarisch reflex is an inhibitory reflex that originates from the heart, is mediated by the vagus nerve, and is manifested by hypotension and bradycardia. For many years, the reflex was considered a pharmacological curiosity. However, recent interest has suggested the reflex may be elicited by myocardial ischemia [1], myocardial reperfusion [2], coronary angiography [3], and aortic stenosis [4].

Four cases of pediatric cardiac operations complicated by recurrent episodes of hypotension and bradycardia are reported. Each was successfully treated with atropine. In each patient, the clinical course was consistent with the Bezold-Jarisch reflex, which suggests that it may be more prevalent than previously suspected in the pediatric cardiac surgical population.

Case Reports

Patient 1

Patient 1 was a 4-month-old, 4.0-kg infant who had congenital aortic stenosis with a 70-mm Hg gradient across the valve preoperatively. He underwent aortic valvotomy under deep hypothermia with circulatory arrest. No postoperative inotropic support was required.

Eighteen hours after operation, the child experienced a precipitous hemodynamic decline with an initial decrease in blood pressure followed by bradycardia and asystole. The sternotomy was reopened at the bedside, and the patient was successfully resuscitated. He was returned to the operating room for irrigation and closure of the sternotomy wound. Dopamine, isoproterenol, and epinephrine were required for hemodynamic maintenance. Two hours later, the infant experienced a second sudden decline in hemodynamics, again initiated by a decrease in blood pressure followed by bradycardia. There was no concomitant change in central venous pressure. He was successfully resuscitated with cardiopulmonary resuscitation. Eight hours later, the child experienced a third episode of hemodynamic collapse, once again heralded by hypotension and followed by bradycardia. Once again, he was successfully resuscitated with cardiopulmonary resuscitation. At this time, the Bezold-Jarisch reflex was suspected. The child was placed empirically on 0.01 mg/kg of intravenous atropine given every 30 minutes. There were no further episodes of hemodynamic collapse. However, the child suffered severe, anoxic, neurological, and renal injuries possibly as a consequence of the repeated episodes of hemodynamic collapse requiring cardiopulmonary resuscitation. He died 3 days later. Autopsy showed left ventricular hypertrophy with no evidence of myocardial infarction.

Patient 2

The second patient was a 2.9-kg infant with type II truncus arteriosus and a type A interrupted aortic arch. As a neonate, the child underwent end-to-end repair of the interrupted aortic arch and pulmonary artery banding. Four hours after pulmonary artery banding, the infant experienced precipitous hypotension followed by bradycardia and asystole. Cardiopulmonary resuscitation was successful in resuscitating the infant. Several hours later, unexplained hypotension developed again. There was no associated change in central venous pressure, arterial blood gases, or ventilator inspiratory pressures. The Bezold-Jarisch reflex was suspected as a possible cause. With the onset of the next episode of hypotension, 0.01 mg/kg of atropine was administered intravenously

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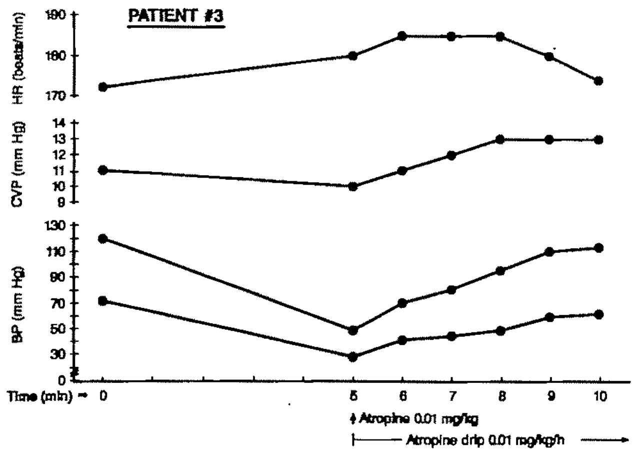

Fig 1. A decrease in blood pressure occurred with no associated change in heart rate, central venous pressure, or inspiratory ventilator pressure. Atropine alone corrected the hemodynamic situation and precluded its recurrence. (BP = *blood pressure;* CVP = *central venous pressure;* HR = *heart rate.)*

with immediate resolution of the hypotension. Thereafter, a bolus of 0.01 mg/kg of atropine was administered hourly over the next 18 hours. It was curious to note that if the administration of atropine was delayed by 10 to 15 minutes, hypotension followed by a decreasing heart rate would occur. Each time, atropine successfully returned the blood pressure to the baseline value.

Twenty-four hours after the pulmonary artery was banded, the child became hypotensive when a scheduled dose of atropine was administered approximately 15 minutes late. By the time the atropine could be administered, the child was quite hypotensive and bradycardic and ultimately became asystolic and could not be resuscitated. Autopsy showed right and left ventricular hypertrophy with evidence of an acute subendocardial myocardial infarction.

Patient 3

A 9-month-old, 7.7-kg child with tetralogy of Fallot underwent complete repair with a 19-mm pulmonary homograft. Circulatory arrest time was 39 minutes. Postoperatively, the child was in stable condition on arrival in the intensive care unit on a therapeutic regimen of 15 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of dobutamine and 1 to 4 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nitroprusside.

The patient was hemodynamically stable until 9 hours after operation. At that time, there was an insidious decrease in blood pressure without a change in heart rate, central venous pressure, or inspiratory ventilator pressures (Fig 1). Her blood pressure decreased from 120/72 mm Hg with sinus rhythm at a rate of 172 beats/min to 48/28 mm Hg with a sinus rhythm of 180 beats/min. Atropine was administered intravenously at 0.01 mg/kg as a bolus dose, and an atropine infusion was begun at a rate of 0.01 $mg \cdot kg^{-1} \cdot h^{-1}$. Within 30 seconds, blood pressure began to increase and by 5 minutes had improved to 113/62 mm Hg. There was no change in heart rate and only a minimal increase in central venous pressure with the administration of atropine. Atropine was the only medical intervention. The atropine infusion was continued for the next 48 hours with no subsequent episodes of hypotension. The remaining postoperative course was unremarkable.

Patient 4

The last patient was a 9-week-old, 5.5-kg child with a type II truncus arteriosus. The child underwent repair with a pulmonary homograft under deep hypothermia and circulatory arrest. The child's condition was quite stable for the first 45 minutes in the surgical intensive care unit, at which time the infant's systolic blood pressure decreased from 90 to 30 mm Hg over several minutes. There was no associated change in heart rate until the blood pressure reached 30 mm Hg, at which time the child became bradycardic. Central venous pressure remained stable at 10 to 14 mm Hg. Chest compressions and hand ventilation were begun. Epinephrine and calcium were administered with absolutely no change in hemodynamics. A bolus injection of 0.01 mg/kg of atropine was administered, followed by rapid improvement in blood pressure. The systolic blood pressure returned to the 90- to 100-mm Hg range, and heart rate remained stable at 145 beats/min. Because we suspected the occurrence of Bezold-Jarisch reflex, an atropine infusion was begun at 0.01 $mg \cdot kg^{-1} \cdot h^{-1}$. The child was also maintained on 5.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of dopamine and 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nitroprusside. The patient's condition remained stable for the next 4 days at which time the inotropic infusions were weaned without incident. The child was discharged home 7 days later.

Comment

The Bezold-Jarisch reflex was described in 1867 by Von Bezold and Hirt [5] and was further elucidated in 1939 by Jarisch and Richter [6]. Until recently, it was regarded as little more than a pharmacological curiosity.

The Bezold-Jarisch reflex was originally elicited by the intravenous administration of Veratridine, which resulted in hypotension and bradycardia [5, 6]. Subsequent work has demonstrated that the reflex is initiated by activation of mechanical stretch receptors located primarily in the posteroinferior wall of the left ventricle [7, 8]. The afferent limb of the reflex arc is mediated by nonmyelinated C fibers that course to the base of the heart near the origin of the left main coronary artery [9]. The afferents are then carried primarily by the right vagus nerve [10] for central integration into the brainstem, which involves at least one cholinergic synapse [11]. Activation of the reflex culminates in hypotension and bradycardia mediated by cholinergic vasodilation and withdrawal of sympathetic tone [12].

The Bezold-Jarisch reflex has been implicated in several clinical situations. Because receptors for the reflex are more densely localized in the posteroinferior aspect of the left ventricle [8], the Bezold-Jarisch reflex is noted far more frequently with posteroinferior left ventricular ischemia than with anterior wall ischemia [1]. In addition, reperfusion of ischemic myocardium may elicit the Bezold-Jarisch reflex, particularly when the ischemic region is the posteroinferior wall of the left ventricle [2]. In fact, elicitation of the Bezold-Jarisch reflex has come to be an indicator of successful coronary thrombolysis [13]. The stimulus that elicits the reflex with either myocardial ischemia or reperfusion is unclear. It has been suggested that mechanical dysfunction of the ventricular wall under these conditions may promote the reflex [14].

The Bezold-Jarisch reflex has been implicated in the mechanism of syncope associated with aortic stenosis. Mark and colleagues [4] demonstrated that in patients with severe aortic stenosis, a vasodilation of the forearm was noted in response to leg exercise rather than the normal response of vasoconstriction. This was interpreted as the activation of left ventricular receptors secondary to an increase in left ventricular pressure. It was observed that after aortic valve replacement, the forearm vasodilatory response returned to the normal vasoconstrictive response.

Bradycardia and hypotension resulting from coronary angiography may be attributable to the Bezold-Jarisch reflex [3], particularly when the contrast medium affects the inferior wall of the left ventricle [15]. The finding that atropine would effectively block the peripheral vasodilation and alleviate the hypotension associated with coronary angiography was of therapeutic value [16, 17].

This report describes 4 pediatric patients who underwent open heart operation and who experienced paroxysmal episodes of hypotension and bradycardia in the postoperative period. The children underwent different surgical procedures, yet all had serious hemodynamic deterioration after their congenital cardiac anomalies were repaired. The Bezold-Jarisch reflex can be implicated with confidence because in each situation, the first noticeable change in the patient's clinical course was a decrease in blood pressure followed by bradycardia (with no initial reduction in cardiac output secondary to bradycardia) and culminating in hypotension. The continuous administration of atropine successfully prevented recurrence of the reflex.

The common eliciting stimulus for the reflex, if any, is unknown. Myocardial ischemia may have been a stimulus because the autopsy in patient 2 did show myocardial necrosis. Stimulation of the left ventricular receptors may have occurred secondary to left ventricular distention or to increased left ventricular pressure.

Development of the Bezold-Jarisch reflex has been studied in swine, and the reflex is noted to mature between 1 week and 2 months postnatally (18). It is inferred that the reflex is present in human infants. Because 4 cases of the Bezold-Jarisch reflex were observed over a short period in patients with different congenital cardiac anomalies, we speculate that the phenomenon may be more common than previously appreciated. Our experience suggests that the Bezold-Jarisch reflex should be considered as a possible cause of otherwise unexplained hemodynamic deterioration in the postoperative pediatric cardiac surgical patient. Should the reflex be suspected, we recommend atropine infusion therapy.

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Combined Thoracic Aortic Dissection and Abdominal Aortic Fusiform Aneurysm

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Certain clinical and autopsy findings are described in 13 patients who had both aortic dissection (AD) and fusiform abdominal aortic aneurysm (AAA). All 13 patients had severe and extensive aortic atherosclerosis. The AAA was diagnosed clinically in 9 patients, and 5 had the AAA resected. The AD was diagnosed clinically in 5 patients, and 2 underwent attempted operative repair. Two patients who had the AAA resected because of suspected rupture were found later to have ruptured a more proximal AD. Thus, AD occurs occasionally in patients who have AAA. In older persons with suspected rupture of an AAA, a more proximal rupture of an AD should be ruled out. When both AAA and AD are present in the same patient, the AD is more likely the cause of cardiovascular collapse than is rupture of the AAA.

(Ann Thorac Surg 1991;52:537–40)

Abdominal aortic aneurysm (AAA) is one of the consequences of severe atherosclerosis (an intimal disease), but most persons with AAA also have or have had systemic hypertension [1]. In contrast, aortic dissection (AD) (a medial disease) is a consequence of systemic hypertension, but many persons with it also have atherosclerosis [1]. Because systemic hypertension is a precursor of both AD and AAA and because systemic hypertension is extremely common in adults in the United States (nearly 40%), the occurrence of both AD and AAA in the

Table 1. Clinical and Morphological Data in 13 Patients With Both Thoracic Aortic Dissection and Abdominal Aortic Atherosclerotic Fusiform Aneurysm

Patient No.	Age (y)	Sex	Diagnosis in Life		Operation for		Rupture		Dissection		Location of AD Entry Tear	Aorta Dissected				CA >75%	LV	
			AD	AAA	AD	AAA	AD	AAA	Acute	Healed		Asc	T	DT	Abd		F	N
1	57	F	+	+	+	0	+	0	+	0	Ascending	+	+	+	0	0	0	0
2	61	M	+	+	0	0	+	0	+	+	Asc + DT	+	+	+	0	0	0	0
3	62	F	0	0	0	0	0	0	+	0	Ascending	+	+	+	0	+	+	0
4	62	M	0	+	0	+	0	0	0	+	Ascending	+	0	0	0	0	0	0
5	63	M	0	+	0	+	+	0	+	+	Asc + DT	+	+	+	0	+	+	0
6	64	M	0	+	0	0	+	0	+	0	Transverse	+	+	0	0	0	0	0
7	65	M	+	0	+	0	+	0	+	0	Ascending	+	+	+	+	+	0	0
8	67	M	+	+	0	0	0	+	0	+	DT	0	0	+	0	+	+	0
9	71	M	+	+	0	+	0	0	+	0	DT	0	0	+	0	+	+	0
10	75	M	0	+	0	+	+	0	+	0	Ascending	+	+	+	0	+	0	0
11	78	M	0	0	0	0	+	0	+	0	Transverse	0	+	+	+	0	0	0
12	80	F	0	0	0	0	+	0	+	0	Transverse	+	+	0	0	0	0	
13	83	M	0	+	0	+	0	0	0	+	DT	0	0	+	0	+	+	0
Total or Mean	68	10 M	5	9	2	5	8	1	10	5		9	9	10	2	7	5	0

AAA = abdominal aortic aneurysm; Abd = abdominal; AD = aortic dissection; Asc = ascending; CA = coronary arterial luminal narrowing; DT = descending thoracic; F = fibrosis; LV = left ventricular; N = necrosis; T = transverse

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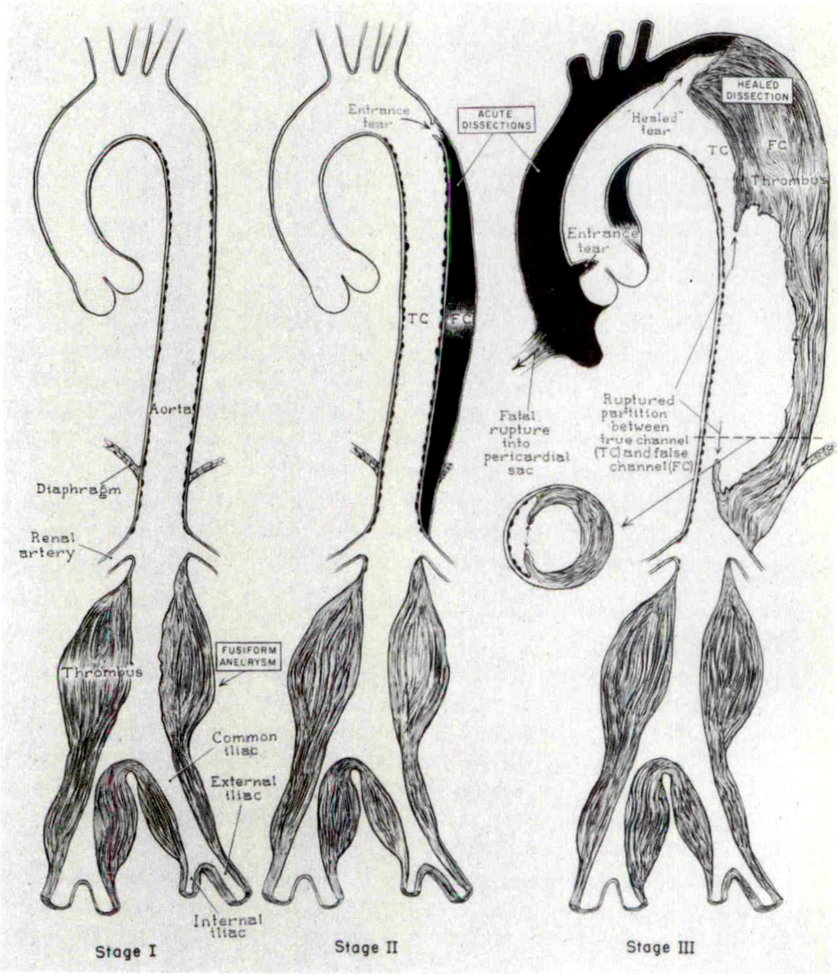

Fig 1. (Patient 5, Table 1.) Drawing of the aorta in a 63-year-old man who had an acute myocardial infarct at age 60 years that healed. At age 63 years, 7 months before death, he was hospitalized with a "virallike illness" that, in retrospect, probably represented an acute aortic dissection with an entrance tear in the descending aorta. Computed tomography at the time disclosed an abdominal aorta aneurysm measuring 5 cm in diameter. A day before death, he was hospitalized with acute chest pain. Rupture of abdominal aortic aneurysm was suspected, and laparotomy was performed. The aorta was ligated just caudal to the renal arteries, and grafts were placed from aorta just proximal to the ligation site to iliac arteries. He died shortly thereafter and was found to have massive hemopericardium (700 mL) from rupture of an acute dissection of the ascending aorta. The drawing shows the 3 stages of events that occurred.

same patient might be expected to be fairly common because both AD and AAA are not infrequent conditions. Reports describing both AD and AAA in the same patient, however, are infrequent [2–4]. In this report, we describe certain findings in 13 patients studied at autopsy and found to have both AD and AAA.

Material and Methods

Pertinent clinical and morphological findings in the 13 patients are described in Table 1. The 13 patients ranged in age from 57 to 83 years (mean, 68 years); 10 were men and 3 were women. All 13 by history had had systemic hypertension. Diagnosis of AAA was made during life in 9 of the 13 patients, and 5 of the 9 patients had the AAA operatively resected: in 3 patients (patients 4, 9, and 13, Table 1), the AAA operation was 2 to 5 years before death, and in 2 patients (patients 5 and 10), the AAA operation was on the day of death. Diagnosis of AD during life was made in 5 patients, and 2 underwent attempted operative repair. In all 13 patients, the AAA appeared to have preceded the AD. In 11 of 13 patients, the AD did not extend more caudally than just past the origin of the renal arteries, and death in 8 patients resulted from rupture of the outer wall of the false channel of the dissected aorta. The resulting hemorrhage was into the pericardial sac in 5 patients and into the left pleural space in 3 patients. Death resulted from rupture of the atherosclerotic AAA in 1 patient (patient 8). Four patients died shortly after operative intervention: patients 1 and 7, both with entrance tears in the ascending aorta, exsanguinated in the operating room after attempted repair of the dissection; patients 5 and 10 clinically were believed to have a ruptured AAA and underwent laparotomy. No rupture was found.

At autopsy, the right-to-left dimension of the AAA in each of the 8 patients in whom the AAA had not been resected was more than 5 cm and all contained intraaneurysmal thrombus. The wall of the AAA was diffusely atherosclerotic, and the arch and descending thoracic aorta also contained extensive atherosclerotic plaques.

The location of the entrance tear of the AD in the 8 patients with acute AD only was ascending aorta in 4 patients, transverse aorta in 3, and descending thoracic aorta in 1 patient. The entrance tear in the 3 patients with healed AD only was in the ascending aorta in 1 and descending thoracic aorta in 2. In 2 patients, both healed and acute dissections were present: the entrance tear for the healed AD in each was in the descending thoracic aorta, and that for the acute AD in each was in the ascending aorta.

The heart was increased in weight (>350 g in women, >400 g in men) in all 13 patients. A transmural (involve-

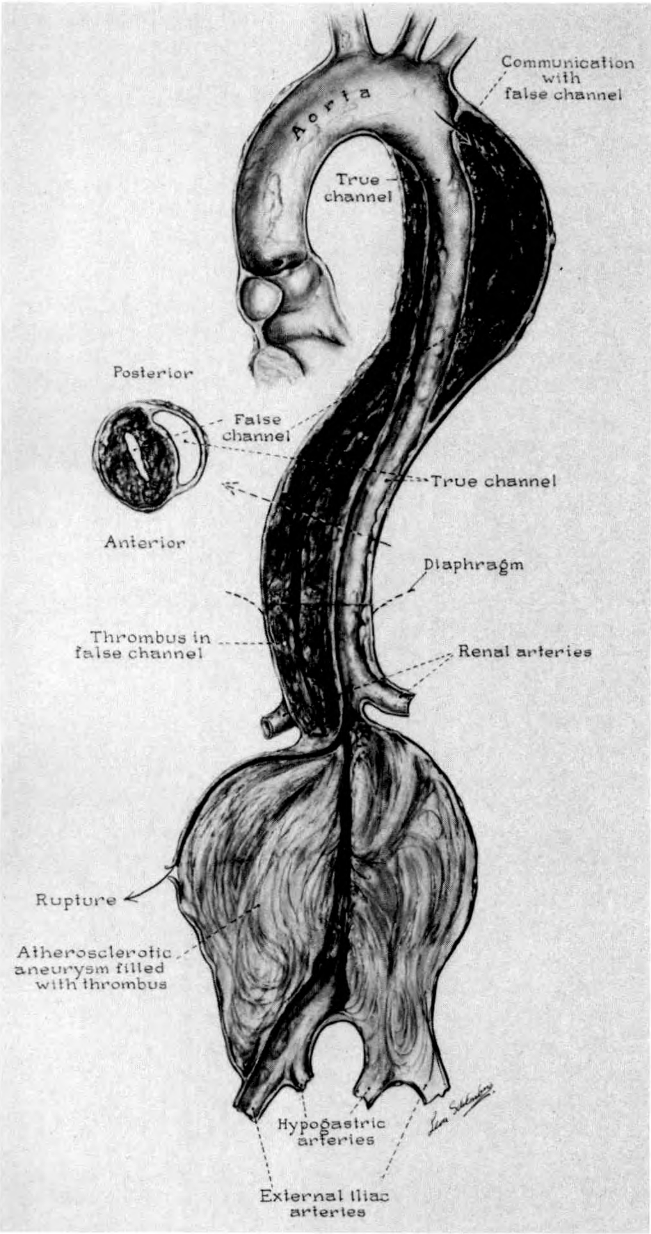

Fig 2. (Patient 8, Table 1.) Drawing of aorta in a 67-year-old man who had a healed aortic dissection with entrance tear in the descending thoracic aorta and fatal rupture of a large atherosclerotic fusiform abdominal aortic aneurysm filled with thrombus. The false channel of the dissection also contains thrombus. The dissection ends at the beginning of the abdominal aortic aneurysm. This patient was the only 1 of the 13 in whom rupture of the abdominal aortic aneurysm occurred rather than rupture of the aortic dissection. (Reproduced with permission from Roberts CS, Roberts WC. Aortic dissection with the entrance tear in descending thoracic aorta. Analysis of 40 necropsy patients. Ann Surg 1991;213:356–68.)

ment of all the inner one half of the wall and all or a portion of the outer one half) left ventricular scar was present in 5 patients, all of whom had severe coronary arterial luminal narrowing by atherosclerotic plaques. Additionally, 2 other patients without left ventricular scars also had severe coronary luminal narrowing by plaques. No patient had grossly discernible foci of left ventricular necrosis (acute myocardial infarct). Only 1 of 13 patients had a dilated left ventricular cavity. The aortic valve in all 13 patients was tricuspid. No patient had aortic isthmic coarctation, and no patient had the Marfan syndrome.

Comment

During a 30-year period, 182 patients with AD were studied at autopsy in the Pathology Branch of the National Heart, Lung, and Blood Institute. In general, the amount of atherosclerosis in the aorta in these patients was relatively mild. Some, however, had 100% intimal surface involvement of the abdominal aorta by atherosclerotic plaque, and of this latter group, 13 had large (>5 cm right-to-left) fusiform abdominal aortic aneurysms. These 13 patients represent 7% of the 182 patients we studied at autopsy.

Although the AAA was chronic in all 13 patients, the AD was acute in 10 patients in whom rupture of the acute AD was fatal in 8. Surprisingly, healed AD was present in 5 of the 13 patients, 2 of whom also had acute AD. These latter 2 patients had acute dissection of the ascending aorta, healed dissection of the descending thoracic aorta, and fusiform aneurysm (atherosclerotic) of the abdominal aorta. The entrance tear of the acute AD was in the ascending aorta in 7 patients, transverse aorta in 3 patients [5], and descending thoracic aorta in 3 patients [6].

The presence of AAA can complicate the diagnosis of AD. Of our 13 patients, only 5 had the diagnosis of AD made during life, whereas 9 had the diagnosis of AAA established during life. A patient with an AAA in whom cardiovascular collapse suddenly develops logically might be strongly suspected to have rupture of the AAA. As exemplified by our patients 5 and 10 (see Table 1), the presence of the AAA might obscure the presence of a more proximal AD. Both patients underwent laparotomy for suspected AAA rupture, but no rupture of the AAA was found; both patients had fatal rupture of the wall of the more proximal acute AD several hours after the abdominal aortic operation (Fig 1). Thus, the potential presence of a more proximal AD might be kept in mind when a patient is admitted with cardiovascular collapse and an AAA is found. On the other hand, the abdominal aortic aneurysm is not immune to rupture in the presence of aortic dissection as exemplified by patient 8 (Fig 2).

Excluding the case described by Snow [7], in which we interpret the published gross photograph of abdominal aorta not to show a fusiform aneurysm, we found only two previous publications describing concurrent AD and AAA. In 1987, Taylor and associates [3] described a 66-year-old man with a large AAA and an AD with an entrance tear in the descending thoracic aorta. It was unclear whether the AD or the AAA caused the fatal rupture. Cambria and colleagues [4] in 1988 found 18 patients with combined AD and AAA (or thoracoabdominal aneurysm) among 325 patients with AD observed at two medical centers (Massachusetts General Hospital and Yale-New Haven Hospital) during a 21-year period (1965 to 1986). Of the 18 patients, 7 had the AAA previously resected and 11 did not. The entrance tear of the AD was in the ascending aorta in only 3 patients (17%) and in the descending thoracic aorta in 15 patients (83%). In all 18 of their patients, both the AAA and AD were diagnosed during life. If they had not been diagnosed, the patients would not have been included in their study, which was a clinical one, in contrast to ours, which was primarily an autopsy one. Fortunately, the occurrence of acute AD in person with preexisting AAA is uncommon. Gomes and Wallace [8] found AD in only 1 of their 200 patients with AAA.

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Complement Activation During Bypass in Acquired C1 Esterase Inhibitor Deficiency

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Serial complement estimations during cardiopulmonary bypass are reported in a patient with acquired C1 esterase inhibitor deficiency. Although the extent of classic and alternative pathway activation appeared appropriate, exaggerated common pathway activation with massive increase in the C3d:C3 ratio occurred. A fatal hemostatic disorder, pulmonary edema, and circulatory collapse ensued despite prophylaxis and therapy.

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Acquired C1 esterase inhibitor (C1-INH) deficiency occurs when classic complement pathway activation is chronically unrestrained [1]. Overconsumption and insufficient resynthesis deplete levels of C1-INH, and angioneurotic edema may be precipitated by minor injury [2]. Although open heart operation has been reported in an asymptomatic patient with biochemical hereditary C1-INH deficiency [3], the present report details the effects of bypass on the complement cascade in a patient with clinically active acquired deficiency.

A 74-year-old man with unstable angina due to severe triple-vessel disease was referred for coronary operation. There was a 7-year history of angioneurotic edema with one episode requiring tracheostomy. Functional and quantitative abnormalities in the classic and alternative pathway components (Table 1) were consistent with acquired C1-INH deficiency. A monoclonal gammopathy was detected but no evidence of malignancy. Edema episodes were controlled with danazol (200 mg/day).

At operation, 100 mg of hydrocortisone was administered and bypass was instituted using a bubble oxygenator with crystalloid prime. Triple-vessel revascularization was performed and bypass was discontinued without difficulty. Protamine and fresh frozen plasma were given but continuous serious bleeding from all suture lines and needle holes was noted. All attempts to secure hemostasis, including administration of further coagulation factors and ϵ-aminocaproic acid, were unsuccessful. Rising airway pressures and clinical pulmonary edema developed, and eventually circulatory collapse occurred and resuscitation was unsuccessful.

With prior informed consent arterial blood samples were drawn during operation into 10 mmol/L ethylenediaminetetraacetic acid. Complement parent molecules C3, C4, and B and their split products C3d, C4d, and Ba were assayed using techniques previously reported [4]. Split product to parent molecule concentration ratios, an index of complement activation, were also calculated.

The fragment:parent molecule ratios of complement factors are displayed in Figures 1 through 3, with the 99% confidence limits of similarly timed investigations on 36 "normal" patients undergoing elective coronary artery bypass grafting. Low concentrations of parent molecules C3, C4, and B were seen preoperatively with an appropriate decrease during bypass (not illustrated). Although alternative pathway activity changed appropriately, there was an exaggeration of classic pathway activation at the end of bypass and sustained massive elevation of the

Table 1. Functional and Immunochemical Assay Values

Assay	Patient	Normal Range
Functional assays		
CH50 (U/mL)	<5	25–45
C4 (U/mL)	<25	40–120 × 10^3
C2 (U/mL)	<50	1,000–3,000
C1-INH (% normal serum)	28	60–120
Immunochemical assays		
C1q (% normal serum)	30	60–130
C1-INH (g/L)	0.06	0.18–0.26
C4 (g/L)	0.01	0.14–0.54
C3 (g/L)	0.08	0.75–1.75
C5 (% normal serum)	50	80–160
Factor B (% normal serum)	40	75–150
IgG (g/L)	17.35	6–16
IgA (g/L)	1.45	0.9–5.2
IgM (g/L)	1.15	0.25–1.6
Serum electrophoresis	Monoclonal IgG	

C1-INH = C1 esterase inhibitor; IgA = immunoglobulin A; IgG = immunoglobulin G; IgM = immunoglobulin M.

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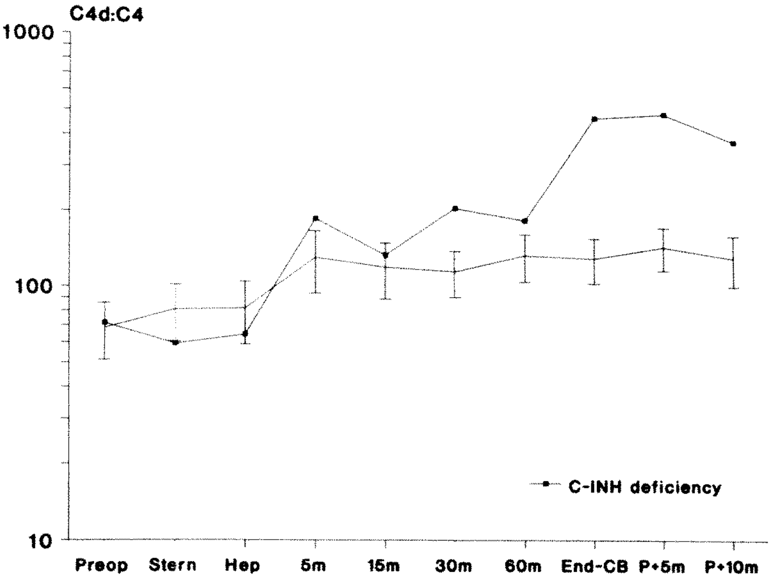

Fig 1. C4d:C4 ratios before, during, and after cardiopulmonary bypass in acquired C1 esterase inhibitor (C1-INH) *deficiency.* Error bars *indicate 99% confidence limits of the mean for similarly timed measurements in 36 "normal" patients. C4d:C4 ratios are displayed on a logarithmic scale.* (Preop = *preoperative;* Stern = *after sternotomy;* Hep = *at heparinization;* 5m, 15m, 30m, 60m = *5, 15, 30, and 60 minutes after commencing cardiopulmonary bypass;* End-CB = *end of cardiopulmonary bypass;* P + 5m = *5 minutes after protamine;* P + 10m = *10 minutes after protamine.)*

C3d:C3 ratio during and after bypass when compared with controls.

Comment

Although acquired C1-INH deficiency is often associated with low C4 concentrations but near-normal levels of C3 [5], the low preoperative levels of C3 in this patient indicate C1-INH deficiency with extensive hypocomplementemia. In an attempt to reduce the known effects of complement activation during bypass preoperative danazol and hydrocortisone and postbypass fresh frozen plasma and ϵ-aminocaproic acid were administered. These measures did not prevent classic and alternative pathway activation in parallel with "control" cases with possible massive activation of the common pathway [4]. C1 esterase inhibitor is known to inhibit plasmin, kallikrein, activated Hageman factor, and activated plasma thromboplastin antecedent, and although C1-INH deficiency is not known to be associated with hemostatic disorders it is conceivable that, in this case, the absence of circulating C1-INH and the stress of bypass led to profound disturbance of other biological cascades resulting in unarrestable hemorrhage, pulmonary edema, and death.

Certain management recommendations can be made in an attempt to avoid repetition of this experience of cardiopulmonary bypass in C1-INH–deficient patients. The continuation of low-dose danazol is probably inadequate to satisfactorily augment C1-INH levels to cover cardiopulmonary bypass. Higher doses, given for at least a week preoperatively, might raise C1-INH levels to greater than 50% of normal [6]. Preoperative fresh frozen plasma

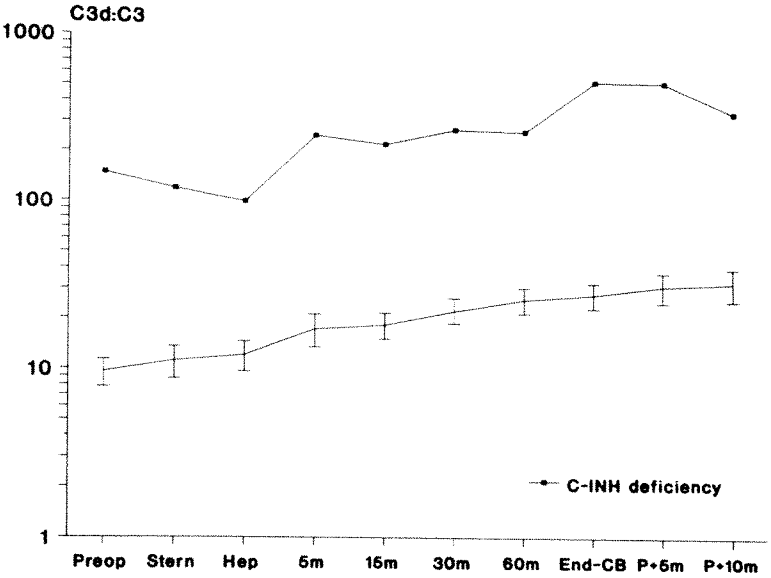

Fig 2. Ba:B ratios before, during and after cardiopulmonary bypass in acquired C1 esterase inhibitor (C1-INH) *deficiency.* Error bars *indicate 99% confidence limits of the mean for similarly timed measurements in 36 "normal" patients. (Abbreviations as in Figure 1.)*

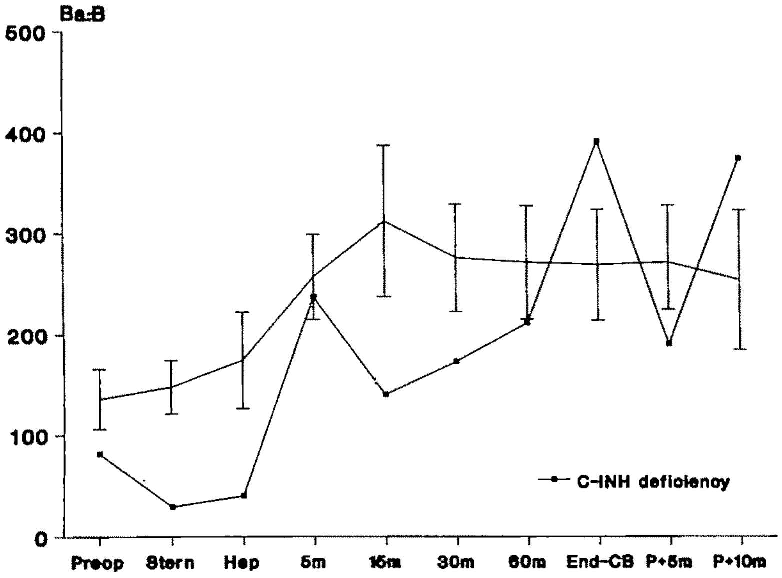

Fig 3. C3d:C3 ratios (logarithmic scale) before, during, and after cardiopulmonary bypass in acquired C1 esterase inhibitor (C1-INH) *deficiency.* Error bars *indicate 99% confidence limits of the mean for similarly timed measurements in 36 "normal" patients. (Abbreviations as in Figure 1.)*

and partially purified C1-INH would increase levels further [2, 7]. Methylprednisolone before bypass, oxygenator type, and priming constituents may also affect complement activation on bypass, and these aspects should be considered when planning cardiopulmonary bypass operation [8]. Although the plasmin antagonists tranexemic acid [9] and ϵ-aminocaproic acid [10] inhibit complement activating enzymes, they also inhibit the degradation of C5a to C5adesArg, potentially increasing the biological activity of C5a. The effects of their use in this situation may, therefore, be unpredictable.

Cardiopulmonary bypass operation in C1-INH deficiency would appear extremely hazardous, and careful preoperative evaluation and management are recommended to try and contain this risk.

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Bidirectional Cavopulmonary Anastomosis Using Vertical Vein for Right Isomerism, Pulmonary Atresia, and TAPVR

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An 8-year-old girl who was diagnosed as having right atrial isomerism, pulmonary atresia, butterfly-shaped juxtaductal pulmonary arterial stenosis, total anomalous pulmonary venous return of the supracardiac type, regurgitant common atrioventricular valve, and univentricular heart of the right ventricular type underwent bilateral, bidirectional cavopulmonary anastomosis using a vertical vein and anastomosis between the common pulmonary venous trunk and atrium. The surgical procedure in this patient is described.

(Ann Thorac Surg 1991;52:544–6)

Patients with atrial isomerism frequently have anomalies of systemic and pulmonary venous return, pulmonary atresia, arborization abnormalities of pulmonary arteries, and common atrioventricular valve, in addition to complex univentricular heart. Although experience with the Fontan operation has resulted in some modifications of the original criteria for patient selection, the presence of such anomalies often precludes the option of Fontan repair. We report a patient with these anomalies who underwent bilateral, bidirectional cavopulmonary anastomosis as an interim operation toward later Fontan repair.

The patient was an 8-year-old girl who had cyanosis since birth. When she was 4 years of age, cardiac catheterization revealed that she had right isomerism, univentricular heart of the right ventricular type, pulmonary atresia with severe juxtaductal bilateral pulmonary arterial stenosis (Fig 1, right), total anomalous pulmonary venous return of the supracardiac type (Fig 1, left), and common atrioventricular valve. Arterial oxygen saturation was 0.71. She underwent right modified Blalock-Taussig shunt, and her cyanosis improved. The second catheterization performed at the age of 8 years revealed moderate regurgitation in the common atrioventricular valve and occlusion of the upper lobe branch of the right pulmonary artery. Arterial oxygen saturation was 0.78. Ventriculography revealed moderately depressed ventricular function, and the end-diastolic pressure was 11 mm Hg.

During operation using cardiopulmonary bypass with moderate hypothermia, the previous modified Blalock-Taussig shunt was divided, and the ductus arteriosus was ligated. The vertical vein was divided, the proximal end was anastomosed to the left pulmonary artery with 6-0 polydioxanone suture, and the distal end was oversewn. Similarly, anastomosis of the superior vena cava to the right pulmonary artery was performed. The common pulmonary vein was anastomosed to the atrium with a continuous suture of 6-0 polydioxanone (Fig 2).

The patient came off bypass easily and postoperative recovery was satisfactory, without any complications. Internal jugular venous pressure ranged from 12 to 15 mm Hg, and atrial pressure was 8 to 10 mm Hg. Arterial oxygen saturation increased to 0.83. Postoperative echocardiography revealed decreased regurgitant fraction in the common atrioventricular valve and improved ventricular contraction. She was discharged from the hospital without any medication on the 15th postoperative day.

Comment

Although the Fontan-type operation has been extensively applied in patients with complex cyanotic congenital heart disease [1, 2], we considered our patient a less than ideal candidate for the Fontan procedure because of the presence of moderate regurgitation in the common atrioventricular valve and congenital or iatrogenic abnormalities of the pulmonary arteries [3]. In addition, there are difficulties in the establishment of direct systemic venous to pulmonary connections when total anomalous pulmonary venous return is present [4].

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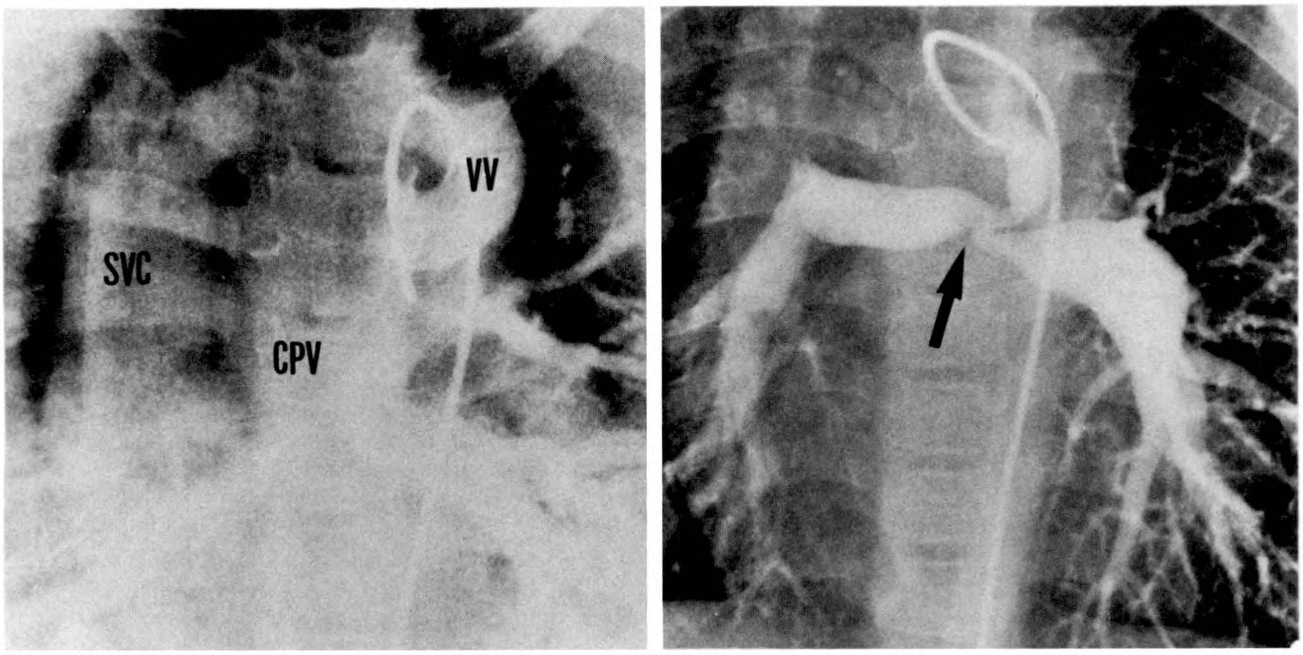

Fig 1. (Left) Levophase of the pulmonary arteriography. (CPV = *common pulmonary vein;* SVC = *superior vena cava;* VV = *vertical vein.) (Right): Ductus injection.* (Arrow = *severe bilateral juxtaductal stenosis of the pulmonary arteries.)*

Bidirectional cavopulmonary anastomosis can be defined as an operation that diverts systemic venous return from the superior vena cava to both lungs. The rationale of the operation is based on experimental work described by Carlon and associates [5] and Haller and associates [6]. The clinical utility of anastomosis of the superior vena cava to the right pulmonary artery was first demonstrated by Glenn and Patino [7]. Variations include anastomosis of bilateral venae cavae to both pulmonary arteries and the total cavopulmonary anastomosis described by Kawashima and co-workers [8] and others [4, 9]. The advantages of bidirectional cavopulmonary anastomosis over the standard Glenn shunt are as follows: continuity between the right pulmonary artery and the left pulmonary artery is preserved, systemic venous blood flow to bilateral pulmonary vascular beds is obtained, and the option for any subsequent procedure remains available [10].

We used a vertical vein in our patient with total anomalous pulmonary venous return as a conduit for bidirectional cavopulmonary anastomosis. This procedure is a prerequisite for establishing the bilateral systemic venous blood flow to both pulmonary vasculatures. Other surgical options include reconstructing central narrow parts of both the right and left pulmonary arteries with a pericardial roll, anastomosing the superior vena cava to the right pulmonary artery, and ligating the vertical vein. However, when these procedures are applied, there are some concerns about future shrinkage of the pericardial roll and growth of the reconstructed pulmonary arteries. Postoperatively, oxygen saturation increased and, as we expected [11], both the ventricular function and the regurgitation in the common atrioventricular valve improved substantially. We expect that the patient will be able to be a candidate for the Fontan repair in the near future.

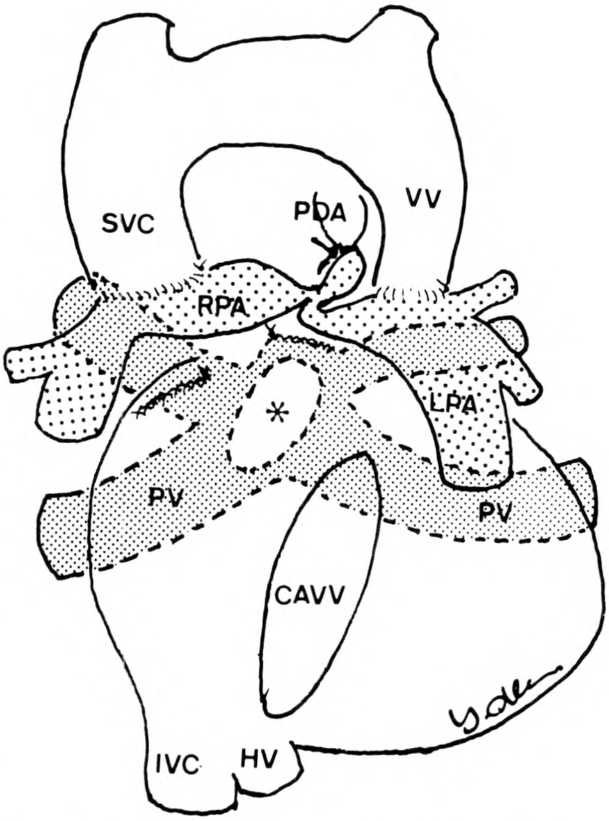

Fig 2. Operative procedures. The aorta is omitted. (CAVV = *common atrioventricular valve;* HV = *hepatic vein;* IVC = *inferior vena cava;* LPA = *left pulmonary artery;* PDA = *patent ductus arteriosus;* PV = *pulmonary vein;* RPA = *right pulmonary artery;* SVC = *superior vena cava;* VV = *vertical vein;* * = *anastomosis between the common pulmonary vein and the atrium.)*

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Large Mediastinal Mass Secondary to an Aortocoronary Saphenous Vein Bypass Graft Aneurysm

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A case of a 62-year-old man with a large mediastinal mass who had undergone aortocoronary bypass grafting 17 years earlier is presented. Computed tomography showed a 13-cm extrinsic cystic mass believed to represent a pericardial cyst or teratoma. Intraoperatively, the patient was noted to have an aneurysm of his right coronary artery bypass graft. We were able to find 4 other cases seen in this manner.

(Ann Thorac Surg 1991;52:547–8)

Aortocoronary saphenous vein bypass graft aneurysms are a rare complication of aortocoronary bypass grafting [1–8]. The following case is a report of such an aneurysm that was seen as a mediastinal mass. We found 4 other cases reported in which this type of aneurysm was seen as a mediastinal mass [1, 2].

A 62-year-old man came to our hospital complaining of difficulty breathing and dull, diffuse chest pain. The patient denied radiation of the chest pain. The patient had undergone triple aortocoronary bypass grafting 17 years earlier using saphenous vein grafts.

On admission, the physical examination was remarkable only for decreased breath sounds in both lower lung fields. Admitting chest roentgenograms showed bibasilar pulmonary edema, right pleural effusion, and a large right anterior mediastinal mass. Admitting electrocardiogram and laboratory analysis were noncontributory. The mediastinal mass was further evaluated with echocardiography and computed tomography of the thorax. Computed tomography revealed a 13-cm cystic mass with mixed densities in the right anterior and middle mediastinum. The mass was believed to be consistent with either a pericardial cyst or teratoma.

The patient underwent a right thoracotomy. A 13-cm nonpulsatile mass was noted. During dissection, a clot was noted in the mass. Subsequent bleeding established the diagnosis of an aneurysm of the right coronary artery bypass graft. The proximal and distal ends of the aneurysm were ligated with 3-0 Prolene (Ethicon, Somerville, NJ) suture. Revascularization was not performed secondary to the calcified, friable, and dilated condition of the proximal segment of the bypass graft. All clots were evacuated, and the right thoracotomy was closed. Histological evaluation of the specimen revealed multiple portions of large blood clots consistent with pseudoaneurysm.

Postoperatively, the patient sustained an acute anterior lateral wall myocardial infarction. The patient responded well to medical therapy. The patient was discharged on the eighth postoperative day in stable condition.

Comment

Aneurysms of aortocoronary saphenous vein bypass grafts are rare [1–8]. We were able to find 4 other cases reported in which such an aneurysm was seen as a mediastinal mass [1, 2]. Coronary artery venous bypass graft aneurysms can occur as early or late complications

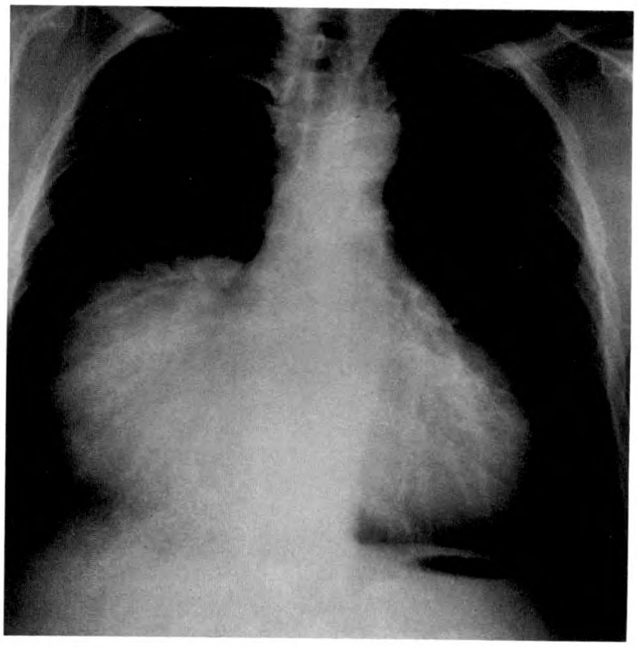

Fig 1. Posteroanterior chest roentgenogram on admission.

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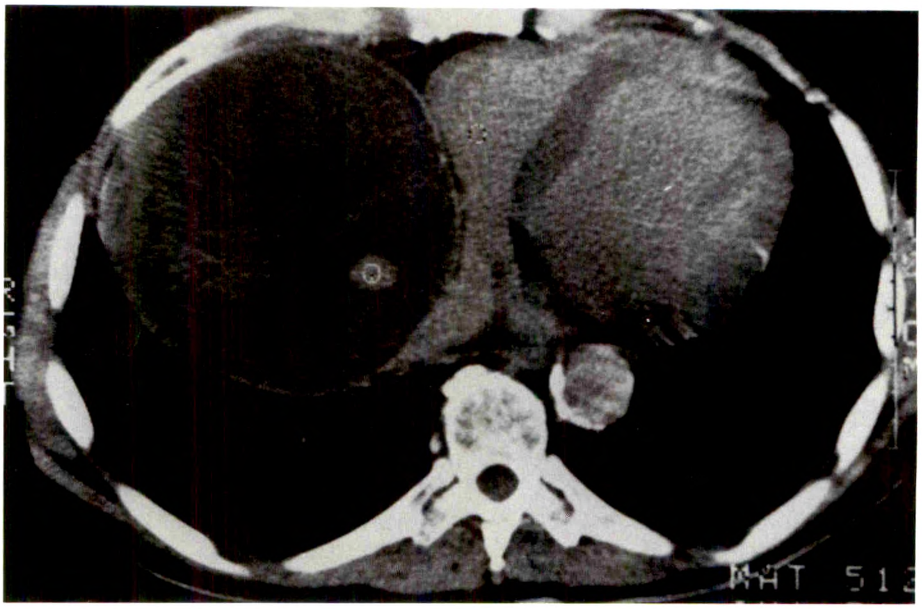

Fig 2. Computed tomogram of thorax demonstrating aneurysm (arrow).

[2, 4–6]. The majority of aneurysms associated with saphenous vein coronary artery bypass grafts occur at an anastomotic site [3, 4, 6]. Aneurysms within the body of the graft, however, do occur. The exact mechanism for the development of such aneurysms is presently unclear. Proposed mechanisms include graft necrosis, hypertension, trauma to the graft at the time of operation, weakness at a branch site, weakness in the area of valves secondary to absent circular muscle in the media, coronary steal secondary to a fistula, thrombosis, and atherosclerotic change within the graft itself [1–8]. Atherosclerotic changes within aortocoronary saphenous vein bypass grafts are well documented [1–8]. Several factors, though, may actually play a role in the development of these aneurysms.

More than one million coronary artery bypass graft operations have been performed in the United States since their inception in 1967 [5]. It is likely that an increasing number of aneurysms of aortocoronary saphenous vein bypass grafts will be encountered [1, 4, 5]. It is our recommendation that all patients with a mediastinal mass who have undergone previous coronary artery bypass grafting should have angiography performed as a part of the evaluation of the mediastinal mass.

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Successful Combined Operation for Takayasu's Arteritis

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We report a case of Takayasu's arteritis complicated with aortic regurgitation and severe stenosis of the left coronary ostium and the innominate artery. Simultaneous surgical treatment of aortic valve replacement, coronary ostium endarterectomy, and aortosubclavian artery bypass using saphenous vein were performed. We discuss the choice of simultaneous operation and the option of surgical treatment for coronary ostium lesion due to Takayasu's arteritis.

(Ann Thorac Surg 1991;52:549–51)

A 51-year-old woman was admitted to our hospital because of heart murmur, vertigo, and loss of visual acuity. In 1972, Takayasu's arteritis was diagnosed in her at another hospital. Cardiac catheterization was also performed, although the result was not known.

On admission in October 1988, the patient was well nourished and had frequent vertigo during walking. The temperature was 36.2°C. Blood pressure was 90/20 mm Hg in the right upper arm and 140/20 mm Hg in the left upper arm. Whole peripheral pulsations were well palpable. Ophthalmological examination showed no abnormalities except bilateral hyperopia. Bilateral neck bruit was heard, and there was no jugular vein engorgement. Grade 3 systolic murmur and grade 2 diastolic murmur at the upper right sternal border were audible. The abdomen was normal. No peripheral edema was found.

The hematological and biochemical studies were within normal limits except that the level of C-reactive protein was 41 mg/L. Electrocardiogram revealed mild left ventricular hypertrophy and depression of ST segment at leads I, II, aVF, and V_3 to V_6. The chest x-ray film showed mild cardiomegaly and normal vascular marking. The echocardiography showed mild left ventricular hypertrophy and fluttering of the mitral valve, and the left ventricular ejection fraction was 0.74. Aortography revealed third-degree aortic regurgitation, and coronary arteriography showed severe stenosis (95%) in the left coronary ostium (Fig 1). Systemic angiography showed severe stenosis in the innominate artery, marked dilatation of the right vertebral artery, complete occlusion in the origin of the bilateral common carotid artery, and long segmental stenosis of the right pulmonary artery. Surgical treatment was planned for aortic regurgitation, stenosis of the left coronary ostium, and stenosis of the innominate artery on November 8, 1988.

Excision of the intima of the left coronary ostium was carefully done with a knife and scissors by inserting a No. 3 Fogarty balloon catheter. Initially, only 1.0-mm probes could be inserted through the ostium, and finally a 5-mm probe could be passed after additional endarterectomy. The aortic valve was somewhat enlongated and thickened. Aortic valve replacement was performed with a 19-mm St. Jude Medical prosthesis. Proximal anastomosis of the saphenous vein graft was done during clamping of the ascending aorta, and the distal anastomosis was performed after declamping. Rewarming was done after the completion of proximal anastomosis of the saphenous vein graft. The weaning from cardiopulmonary bypass was smooth.

The patient's postoperative course was good except for poor control of inflammation due to Takayasu's arteritis

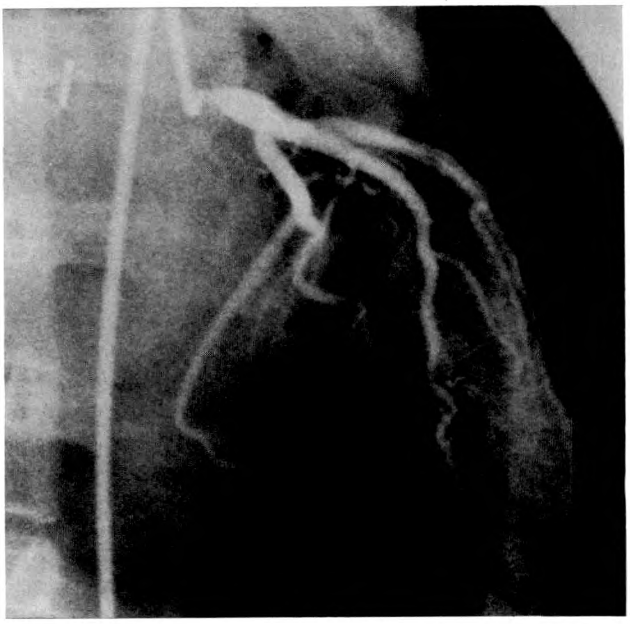

Fig 1. Preoperative coronary angiogram demonstrated 95% stenosis in the left coronary ostium.

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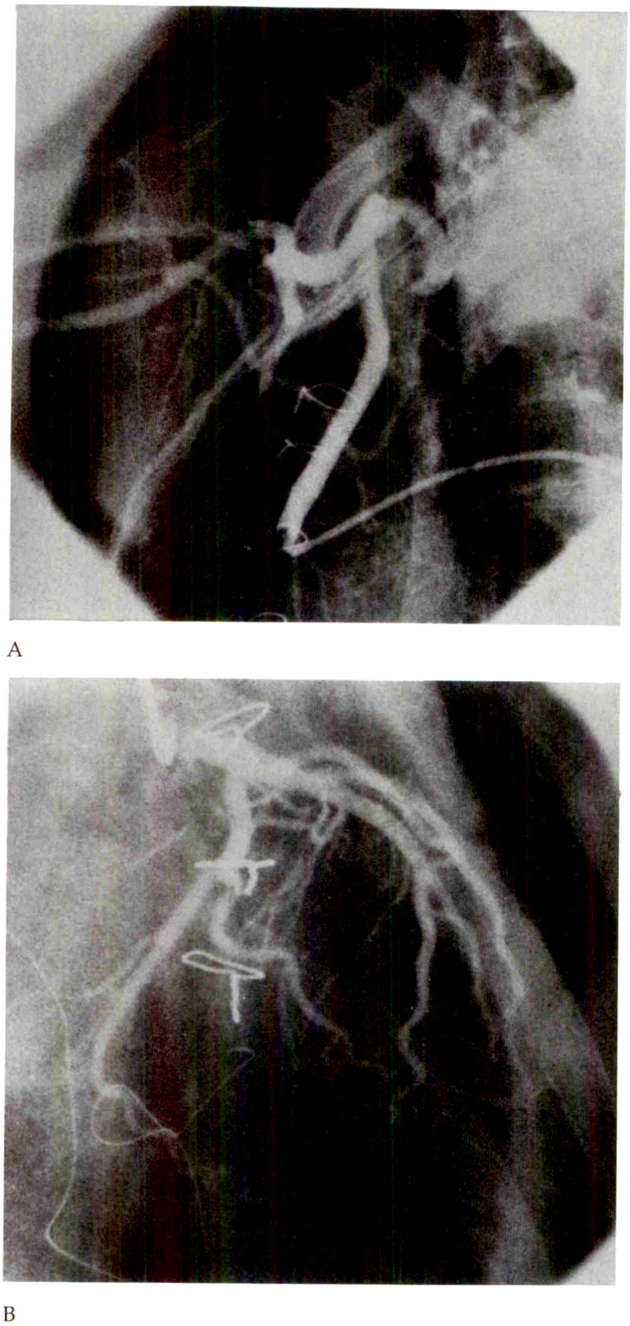

Fig 2. Postoperative angiogram shows that the aortosubclavian bypass graft is patent (A) and that severe stenosis of the left coronary ostium is improved (B).

and posttransfusional hepatitis. Although prednisolone was not prescribed preoperatively, a low dosage of prednisolone was necessary to control the inflammatory reaction after operation. Postoperative angiography demonstrated that no stenosis was observed at the left coronary ostium, and the aortosubclavian artery bypass graft was patent (Fig 2). No problems with the prosthesis were seen. The specimens from the aortic wall showed fibrous thickening at the adventitia and the media, as well as infiltration of eosinocytes and lymphocytes at the media. Three months were necessary for the control of inflammation and the recovery from hepatitis. She is now doing well 26 months postoperatively with no complaints.

Comment

Takayasu's arteritis was first reported by Takayasu, a Japanese ophthalmologist, in 1908 [1]. It had been understood as a nonspecific vasculitis, but its etiology is still not clear. The incidence of coronary arterial lesion is relatively low, about 10% [2], and 80% to 90% of the coronary lesions are observed in the coronary ostia or in the proximal vessels [3].

The surgical treatment options for coronary ostial stenosis due to Takayasu's arteritis have been reported to be coronary artery bypass grafting [4, 5] and ostium endarterectomy. Ohara and associates [6] reported 18 cases of surgical treatment for coronary ostial stenosis due to Takayasu's arteritis, and they concluded that ostium endarterectomy was superior to coronary artery bypass grafting in terms of long-term patency at 4-month to 6-year follow-up. The proximal anastomotic portion of the ascending aorta would be stenosed or occluded if inflammation extended in case of coronary artery bypass grafting [7], although the same condition at the coronary ostium could occur in case of endarterectomy. Recently, some groups have reported that arterial grafts, for example, internal mammary artery and gastroepiploic artery [8], were effective in treating the ostium lesion due to Takayasu's arteritis. But internal mammary artery and gastroepiploic artery cannot be used as grafts when the inflammation extends to the aortic arch and the descending aorta, respectively. Every kind of surgical treatment for coronary stenosis has demerits and pitfalls.

We chose ostium endarterectomy for this patient because of localized stenosis in the left coronary ostium and because no active inflammation was found preoperatively. We also performed aortosubclavian bypass in this patient because cerebral blood flow was supplied by only the right vertebral artery. Postoperatively, ocular symptoms including vertigo and loss of visual acuity were improved. Finally, the reasons that we chose the simultaneous operation are mainly summarized in the following two points: First, we estimated that the simultaneous operation in this case could be safely done in one visual field. Second, we had to prevent the occurrence of cranial nerve disturbance intraoperatively and postoperatively.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1994 may begin the recertification process in 1992. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is May 1, 1992. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Long-Term Function in University of Cape Town Prostheses in the Tricuspid Position

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A 55-year-old woman undergoing triple-valve replacement was found at operation to have a well preserved and functioning University of Cape Town prosthesis that had been inserted 18 years earlier for tricuspid stenosis. Case reporting, valve reappraisal, and factual updating on another case, the world's first documented tricuspid valve replacement for Ebstein's anomaly, are presented. These two remarkable in vivo successes speak well for this historic collar stud valve.

(Ann Thorac Surg 1991;52:552–4)

The University of Cape Town (UCT) prosthesis was first introduced and reported in *The Lancet* in 1962 by Barnard and associates [1] 2 years after Starr introduced the ball and cage valve. The early UCT prosthesis was a "ball type valve without a cage" [2], which in effect made the prosthesis one of the earliest low-profile devices. Subsequent design modifications changed the lens-shaped obturator for the mitral and tricuspid positions into a disc [2], which further optimized its mechanical function. This collar stud valve was used widely by some surgeons [2, 3] during the 1960s and early 1970s. A separate, though somewhat similar aortic prosthesis [2] was also introduced. We report a patient in whom a UCT valve functioned well in the tricuspid position for 18 years. This is the second such report of long-term in vivo success with a UCT prosthesis in this position [4].

A 55-year-old woman had had rheumatic fever when 9 years old. In 1954 she began treatment for dyspnea on exertion and by 1966, then aged 35 years, was noted to be in congestive heart failure with gross mitral regurgitation and atrial fibrillation. In 1968 right and left heart catheterization showed tricuspid stenosis, gross mitral regurgitation, and mild aortic regurgitation. During the same hospital admission, at the Royal Victoria Hospital in Belfast, the mitral and tricuspid valves were replaced under cardiopulmonary bypass. The mitral valve exposed through an atrial transeptal approach was both stenotic and regurgitant with thickened fibrosed leaflets and matted chordae. The tricuspid valve also showed rheumatic change and had a stenotic orifice measuring 2 cm × 1 cm in diameter. Mild aortic regurgitation did not warrant surgical correction. The mitral valve was excised and replaced with a No. 3 Starr-Edwards cloth-covered prosthesis, whereas the tricuspid valve was replaced with a size 4 UCT mechanical prosthesis.

Progress after operation was smooth and satisfactory, and the patient remained well with a normal lifestyle until symptoms in 1982 gradually recurred.

By 1986 New York Heart Association functional class III dyspnea and angina were present and the patient was reinvestigated. Echocardiography showed a hypertrophied left ventricle with good systolic function and a thickened and incompetent aortic valve. The right ventricle appeared normal, and the UCT tricuspid valve opened and closed well. Subsequent cardiac catheterization confirmed the echocardiographic studies and showed a wide aortic pulse pressure (153/43 mm Hg), an elevated left ventricular end-diastolic pressure (18 mm Hg), and a transvalvular aortic gradient of 39 mm Hg. The coronary arteries appeared normal on root injection, and right heart studies showed an elevated mean right atrial pressure (10.4 mm Hg), a mean diastolic gradient of 4 mm Hg across the tricuspid valve, and a low cardiac output of 1.8 L/m^2. The conclusions were mixed aortic valve disease, a probably normally functioning but small Starr-Edwards mitral prosthesis, relative obstruction at the UCT tricuspid valve, and left ventricular failure.

At reoperation the aortic valve was thickened and calcified and had peripheral commissural fusion. The UCT tricuspid valve moved well but had tissue ingrowth around the valve ring. No evidence of thrombus or obstruction, however, was present. The Starr-Edwards mitral prosthesis exposed transseptally appeared to function well but had tissue encroachment on one of the struts, some degeneration of cage cloth, and heavy calcification in the valve ring. It was also apparent that the mitral prosthesis had made a fixed impression in the left ventricular septal wall. Triple St. Jude valve replacements were carried out (aortic, 21 mm; mitral, 29 mm; tricuspid, 31 mm). The patient made steady progress after operation and was discharged on the 17th postoperative day in a satisfactory condition.

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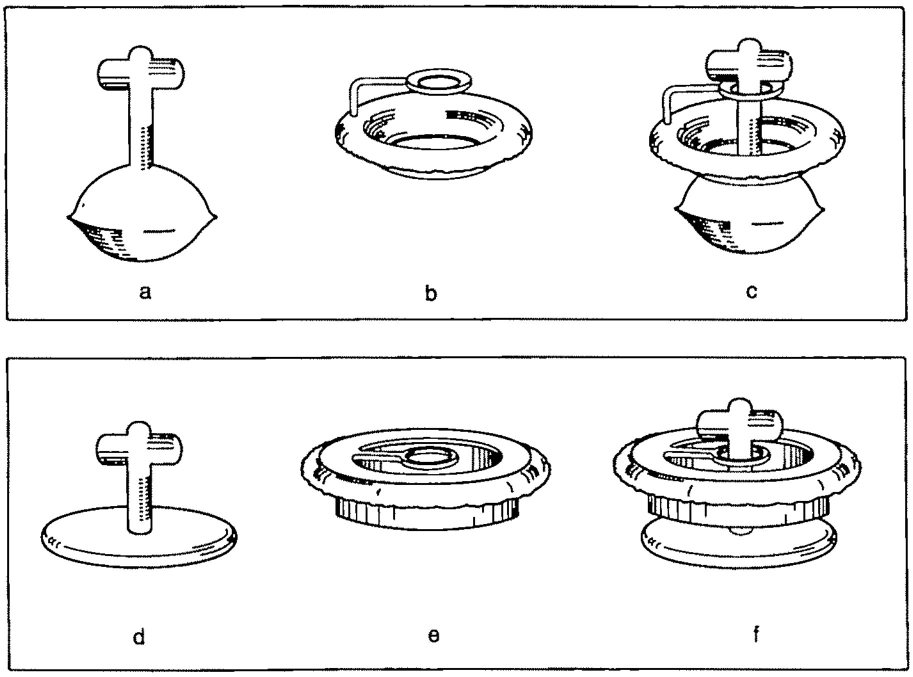

Fig 1. (a, b) Components of an early design University of Cape Town atrioventricular valve. (c) Assembled valve. (d, e) Components of a later design University of Cape Town atrioventricular valve. (f) Assembled valve.

Comment

The UCT mechanical prosthesis was introduced in 1962 by Barnard and associates [1] as one of the first low-profile valves. Its unique features included a squashed ball (lens) and no cage. These attractive design features offered an alternative and a possible advantage to the then-existing Starr-Edwards mechanical valve. Two different varieties of UCT prostheses were marketed, one being suitable for atrioventricular positions and the other for the aortic position. The current report concerns the former variety, which had two components. The fixed portion of the valve (Fig 1b) consisted of a cloth-covered valve ring and a metal suspensory apparatus situated on the atrial aspect of the prosthesis. A mobile portion (Fig 1a) made of Silastic incorporated a suspension bar, a flexible stem, and a lens-shaped obturator. This mobile poppit moved in systole and diastole through a circular ring in the suspensory apparatus (Fig 1c). Subsequent Mark 2 and 3 modifications of the early design included a suspension system horizontal with the valve ring (Fig 1e), a shorter flexible stem, and a flattened disc-shaped obturator [2] (Fig 1d). These improvements made the apparatus less bulky and more low profile (Fig 1f).

The valves were used by a number of enthusiasts in the 1960s and early 1970s, but published reports indicate that they were mainly inserted in the aortic and mitral positions. Early track record reports suggest that, in particular, the mitral prosthesis was associated with excessive thromboembolic complications [2, 3, 5]. Against this, however, it is noteworthy to state that the usage of anticoagulants with the early implants was only selectively applied.

Hemodynamic studies published by Barnard and coworkers [2, 6, 7] in 1965 and 1966 suggested that the valve in the tricuspid position was competent but did not permit flow as readily as in the aortic and mitral positions. It was further believed that the obturator may encroach on the right ventricular wall in some instances. They concluded that UCT valve insertion in the tricuspid position was its least efficient usage [2].

It is an interesting and historic fact that the first successful correction of Ebstein's anomaly by total replacement of the tricuspid valve, reported by Barnard and Schrire [8], was carried out in 1962 using an early design UCT prosthesis. This operation was performed on a 4-year-old male black child in Capetown with refractory heart failure. The same patient, reviewed and reported in the *British Heart Journal* [4] in 1981, 19 years after implantation, was alive and well with the original UCT prosthesis in situ, even though at routine cardiac catheterization in 1975 a transprosthetic gradient measured 6 mm Hg. Recent enquiries to South Africa confirmed the ongoing good health of this patient, now a 32-year-old man. After a 6-year absence he attended medical review in November 1988 for the purpose of obtaining a disability pension. He was noted to be well, rides a bicycle, and has New York Heart Association functional class I symptoms despite a 4.0-cm jugular venous pressure elevation. His electrocardiogram is unchanged, and a chest roentgenogram reveals moderate cardiomegaly with the valve ring showing clearly on the lateral view. Being unreliable in his attendance at prothrombin clinics, he has not taken anticoagulants for some years.

This case and ours are both long-term in vivo tributes to this historic collar stud valve considered many years ago by its proponents, Barnard and associates [2], to be least efficient in this, the tricuspid position.

We would like to express our appreciation and thanks to Professor P. J. Commerford, Groote Shuur Hospital, Cape Town, for generously allowing us to quote unpublished and up-to-date information about his patient, and to Marcus Cremonese for artwork.

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Schwannoma of the Brachial Plexus Mimicking an Apical Lung Tumor

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Primary tumors of the brachial plexus are unusual. We describe a patient with a large schwannoma of the lower trunk of the brachial plexus that had the radiologic appearance of an apical lung mass. Use of a posterior subscapular approach as well as intraoperative nerve action potential recording permitted resection with spared function.

(Ann Thorac Surg 1991;52:555–6)

Primary tumors of the brachial plexus are unusual. Patients commonly seek medical attention because of pain and functional loss or because of an asymptomatic neck mass [1]. We describe a patient with a schwannoma of the lower trunk of the brachial plexus that had the radiologic appearance of an apical lung mass.

A 43-year-old male smoker was found to have a right apical lung mass after evaluation for hemoptysis. He denied hoarseness, weight loss, and arm or shoulder pain. Physical examination was within normal limits. There was no ptosis, meiosis, anhidrosis, or muscle weakness in the right arm or hand. Chest roentgenogram demonstrated a right apical lung mass and a normal mediastinum (Fig 1). Chest computed tomography confirmed the presence of the mass, which appeared to be within the pulmonary parenchyma (Fig 2). A bone scan and brain computed tomographic scan were normal. Flexible fiberoptic bronchoscopy with washings, brushings, and biopsies was nondiagnostic. Serum laboratory values were normal.

A right thoracotomy was performed after intubation with a double-lumen endotracheal tube. No parenchymal lesions were palpable in the right lung. Further exploration revealed a 4 cm, well-circumscribed, extrapleural mass arising from one of the trunks of the brachial plexus. Nerve fibers were intimately involved on the superior and lateral sides of the tumor. A biopsy specimen was taken from the inferior aspect, and the diagnosis of schwannoma was established. The patient's chest was closed, and he was referred for resection using intraoperative action potential recordings. Postoperative findings included a mild and partial right Horner's syndrome and very mild reduction in hand intrinsic muscle strength. Tapping the supraclavicular nerve elicited a mild Tinel's sign along the shoulder tip.

A preoperative magnetic resonance imaging scan of the brachial plexus demonstrated intimate involvement with the inferior nerve roots (Fig 3). Definitive resection was performed through a posterior subscapular approach. Partial resection of the first rib was used to expose the tumor. A tumor 4 cm in diameter was present in the lower trunk of the plexus (Fig 4). Tumor extended to the T1 nerve root proximally, but did not involve the lower trunk division distally. Nerve action potentials were recorded before resection by stimulating the C8 and T1 nerve roots proximal to the tumor and recording from distal divisions. Dissection was accomplished by splitting fascicles away from the tumor mass. Nerve action potentials were periodically checked as the dissection proceeded. The tumor was shelled out of the lower trunk and the distal portion of T1. Though there was some reduction of velocity amplitude across the T1 nerve root to the lower trunk division, nerve action potential conduction was preserved. Tumor adherent to the pleura was resected with a small segment of apical pleura. The patient recovered without motor deficit, but did have mild paresthesias of the fourth and fifth digits. No cause for hemoptysis was identified. A subsequent head and neck examination was

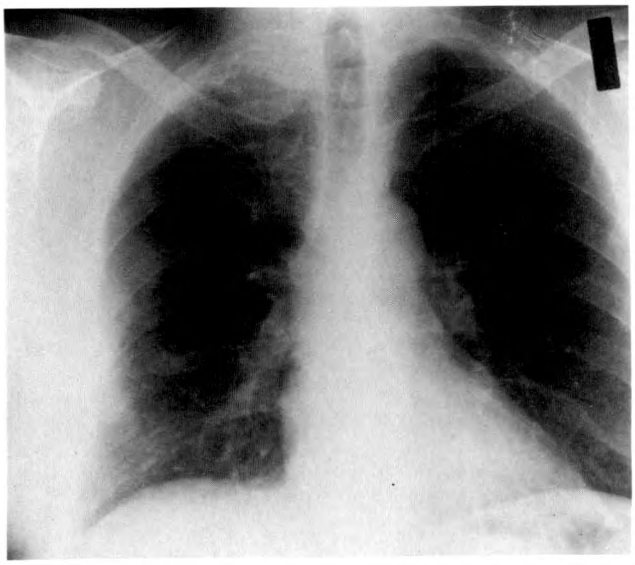

Fig 1. Posteroanterior chest roentgenogram with typical appearance of a right apical lung mass.

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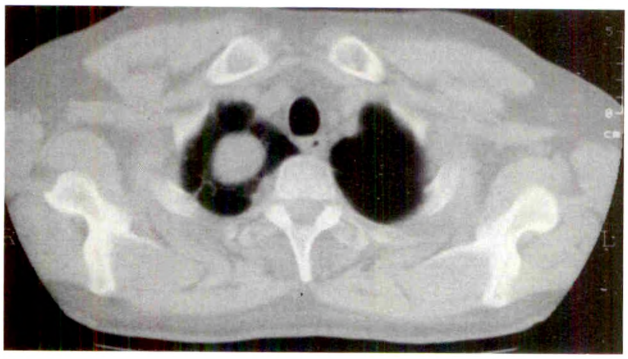

Fig 2. Computed tomographic scan demonstrating what appears to be an intraparenchymal right apical lung mass.

normal. Surveillance with roentgenograms and sputum cytology has been advised.

Comment

Lusk and associates [1] reported the largest single institutional series of brachial plexus tumors and summarized the 150 cases published during the past century. Neurofibromas were the most common (57%) of the 46 primary brachial plexus tumors treated during the 17-year study interval. A majority of these were unassociated with von Recklinghausen's disease. Schwannomas represented 26% of neoplasms. The remaining tumors were of nonneural sheath origin.

The most common physical findings associated with brachial plexus tumors are related to local growth. Motor or sensory deficits caused by axonal loss occurs more frequently with neurofibromas and malignant nerve sheath tumors. Schwannomas usually are seen as an asymptomatic neck mass [2]. Intrathoracic extension without an accompanying supraclavicular mass has been reported only once and was assumed to represent a lung cancer [3].

Surgical excision of brachial plexus tumors is indicated to prevent or minimize neural damage. A posterior subscapular (high posterior thoracoplasty incision) approach with elevation of the scapula and resection of the first rib is recommended for tumors involving the C8 and T1 nerve roots and for tumors of the lower trunk [4]. An anterior (supraclavicular) incision is used for all other tumors [5]. Neural fascicles are more intimately involved with neurofibromas than with schwannomas [1]. Fascicles draping over schwannomas can be split and teased away from the tumor mass permitting enucleation. Intraoperative measurement of nerve action potentials allows identification of functional nerve fascicles. If functional nerves must be sacrificed, then immediate repair with a graft (usually sural) can be performed.

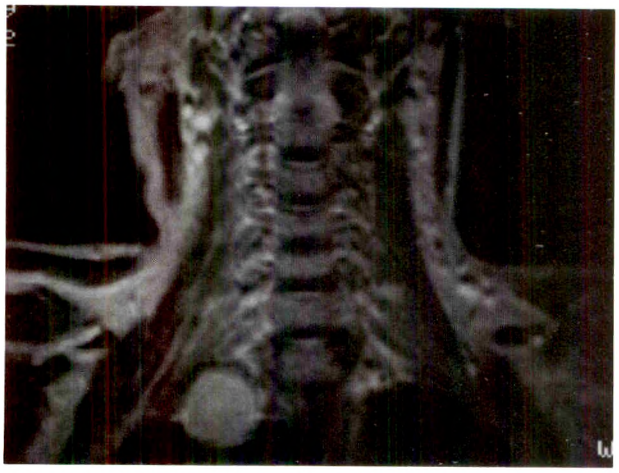

Fig 3. Magnetic resonance imaging scan demonstrating attachment of the mass to nerve roots.

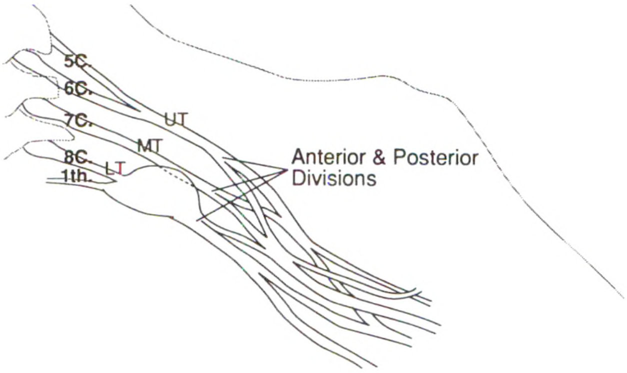

Fig 4. Intraoperative findings. Tumor extended to the T1 nerve root proximally but did not involve the lower trunk division distally. (LT = *lower trunk;* MT = *middle trunk;* UT = *upper trunk.)*

Physical examination, chest roentgenogram, and computed tomographic scan are not reliable methods of differentiating the more common apical lung neoplasm from the rare brachial plexus tumor of the lower cervical nerve roots. Magnetic resonance scanning is highly specific and would allow preoperative neurosurgical consultation but is not part of the usual evaluation of patients with presumed lung cancer. Though tumors involving thoracic roots other than T1 may be removed without functional loss, those involving T1 or cervical nerve roots cannot. If an unexpected brachial plexus tumor is encountered, resection should be deferred until appropriate neurosurgical expertise is available.

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Pericardial Mass Mimicking Constrictive Pericarditis

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Persistent pericardial hematoma due to blunt chest trauma is extremely rare. We report a case of constricted myocardium resulting from a large, partially organized hematoma in the right cardiophrenic angle. The hematoma was assumed to be caused by an occupational accident 17 years before diagnosis. Constriction could only be controlled by complete removal of the fibrous posterior capsule of the hematoma, which covered the epicardium of the right heart.

(Ann Thorac Surg 1991;52:557–9)

Constrictive pericarditis constitutes an important diagnosis because of the potential for surgical cure of the condition. Today, constrictive pericarditis is commonly of an idiopathic etiology or is a consequence of radiotherapy, cardiac surgery, or infection. Constrictive pericarditis is seen rarely in association with connective tissue disease, neoplasms, uremia, and sarcoidosis or follows chest trauma [1]. In this report, we describe an unusual case of a patient with clinical and laboratory signs of constrictive pericarditis that were associated with a pericardial mass.

A 65-year-old man was admitted for evaluation with a 4-month history of bilateral pleural effusions and increasing dyspnea and chest pain on moderate exertion. In 1974, a fractured right ankle had been repaired and omental bleeding had been controlled surgically. The latter two injuries were the consequences of an occupational accident that included a blunt abdominal trauma. During his occupational accident, the patient had fallen and also struck a wooden beam with his chest. This injury caused a fracture of the fifth rib in the right anterior axillary line, but there was no evidence of heart and lung injury at the time. In 1988, the patient had undergone several laparotomies for repair of an infrarenal aortic occlusion and subsequent adhesion ileus.

Physical examination revealed a cachectic man appearing older than his numerical age and chronically ill. There was marked jugular venous distention. Cardiac examination showed no pathological sounds or arrhythmias. Examination of the lungs revealed a bilateral basal dullness. The liver was seriously enlarged and discrete ankle edema was present. Examination of plasma and fluid from pleural effusions showed no evidence of active viral or bacterial infection, including tuberculosis. Liver function test results were slightly abnormal. Bone marrow and pleural effusion fluid contained no malignant cells. An electrocardiogram demonstrated sinus tachycardia with a descendent ST segment and preterminal inversion of the T segment in the precordial leads. Chest roentgenographic examination indicated bilateral basal pleural effusions. The heart size was normal (Fig 1). Cardiac catheterization demonstrated elevated end-diastolic pressures in the right atrium, right ventricle, and left ventricle. The intracardiac pressures were: right atrium, 14/16 mm Hg; right ventricle, 27/12 mm Hg with an early diastolic dip; pulmonary artery, 25/16 mm Hg; mean pulmonary artery wedge, 16 mm Hg; and left ventricle, 110/18 mm Hg. Left ventricular cineangiography revealed moderate coronary heart disease involving the circumflex branch of the left coronary artery and the right coronary artery. The left ventricular chamber was small and contracted well. Cardiac output (2.7 L/min) and cardiac index (1.7 L · min^{-1} · m^{-2}) were substantially reduced. A two-dimensional echocardiogram demonstrated an echolucent mass anterior to the right ventricle. This mass led to a marked compression of the right ventricle. A subsequent computed tomographic scan demonstrated a low-attenuated mass, which did not enhance with intravenous contrast material administration, and which was located in the anterior mediastinum adjacent to the right ventricle. The mass was clearly separable from the myocardium by a thickened capsule and appeared to compress the right ventricle. The capsule was regarded as the pericardium. Between the myocardium and the presumed pericardium a layer of epicardial fat was seen (Fig 2).

The typical localization of the mass in the right cardiophrenic angle and the density and morphologic appearance of the lesion were suggestive of a pericardial cyst [2]. The clinical and laboratory findings (characteristic early diastolic dip of the right ventricular pressure curve) led to the conclusion that the patient was suffering from a pericardial cyst, which caused signs of constrictive pericarditis and hence was responsible for his complaint. Accordingly, operation was recommended.

The chest was opened through a midline sternotomy.

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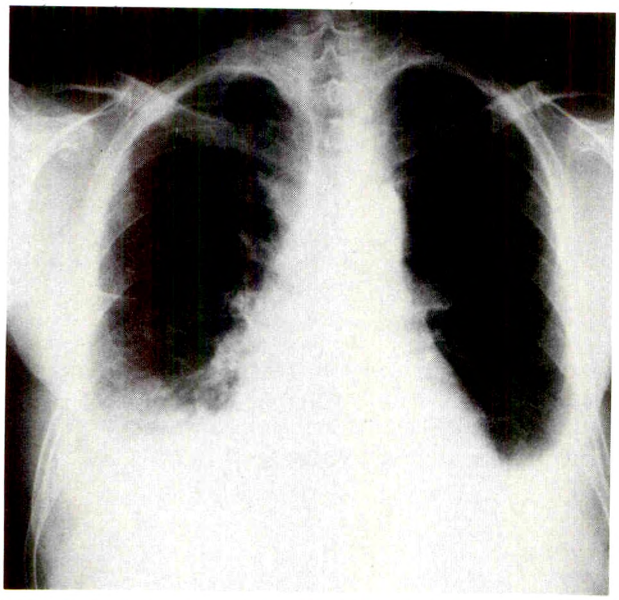

Fig 1. Frontal view of conventional radiogram showing normal heart size and bilateral pleural effusions.

The portion of the parietal pericardium that was localized over the right ventricle and atrium was found to be brownish, moderately thickened, and scarred, indicating chronic inflammation. However, it did not appear constrictive. Subsequently, the pericardium was dissected off the aorta and the area of the right atrium and ventricle. There was a large pool of thick, semifluid brownish material, presumably old blood, in the pericardial cavity. When the thrombotic and partially fibrous material was removed, the posterior wall of the hematoma became visible. This wall was a rigid, fibrous membrane, clearly not of pericardial origin, and could not be distinguished from epicardium. However, myocardial performance was not improved by complete resection of the parietal pericardium and removal of the hematoma. The right ventricle still showed signs of constriction—a rapid filling phase with an abrupt interruption and a simultaneous distention of the overlying fibrous membrane. Simultaneously, central venous pressure remained unchanged. Therefore, it was decided to dissect this membrane. When the membrane was incised longitudinally, normal epicardium appeared and the heart muscle and adjacent fat tissue herniated through the opening. The membrane was dissected off the right ventricle and the right atrium. Subsequently, central venous pressure decreased and cardiac action became much more forceful.

The postoperative course was uneventful. The patient demonstrated clear clinical improvement. Microscopic examination of the surgical specimen confirmed the intraoperative findings and showed chronic pericarditis with organized and encapsulated hematoma. Therefore, the diagnosis was changed to organized and encapsulated intrapericardiac hematoma mimicking constrictive pericarditis. Because of the preceding blunt chest trauma we assumed a traumatic origin of the hematoma and the resulting symptoms of cardiac constriction.

Comment

The etiology of constrictive pericarditis often remains unexplained in spite of aggressive clinical and laboratory examinations. It is known that the pathophysiological features of constrictive pericarditis can also be mimicked by clotted hemopericardium and epicarditis [3, 4]. In the present report we describe a case in which the fibrous capsule of an old pericardial hematoma caused symptoms of constrictive pericarditis.

The history of the patient and the anatomical findings suggest a traumatic origin of the constrictive pericardial hematoma. Thus, chest trauma is a well-established, although rare, cause of constrictive pericarditis. Furthermore, traumatic hemopericardium may stay unrecognized initially in the absence of severe clinical symptoms, and symptoms of constriction may appear as late as 20 years after the injury [5–8]. In cases of traumatic hemopericardium, a decrease in the absorptive capacity of the pericardium as well as a loss of its normal mesothelial fibrinolytic activity due to the injury is thought to lead to clot formation inside the pericardial sac. When the blood attaches to the damaged surfaces and clots, pericardial inflammatory changes occur and granulation tissue develops, initiating the process that leads to formation of adhesions and possible constriction [9].

However, persistent constrictive pericardial hematoma due to blunt chest trauma is an extremely rare finding. Of the 3 cases described in the literature [6–8], an old hematoma per se seemed to have been responsible for the constriction in 2 [6, 8], whereas in the remaining case a true constrictive pericarditis apparently had been present [7]. Our case adds the hematoma-induced formation of fibrous pseudomembranes to the etiology of posttraumatic cardiac constriction. Furthermore, our case report demonstrates the impossibility of predicting exactly the

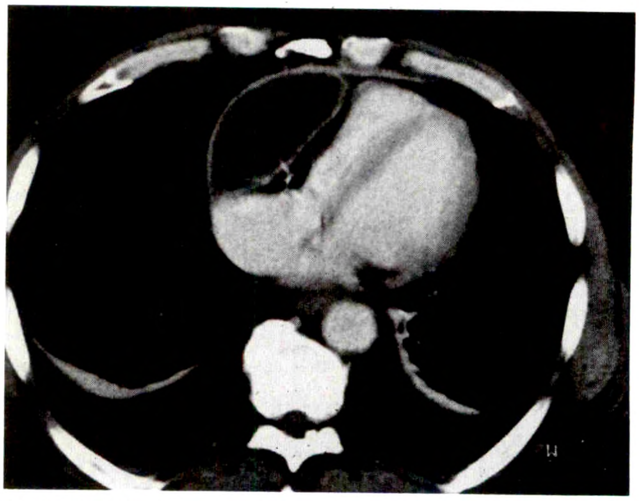

Fig 2. Computed tomographic scan during bolus contrast medium application. The scan shows a low-density, nonenhancing mass adjacent to the right ventricle and compressing it. A layer of epicardial fat is located between the myocardium and a thickened membrane, thought to be pericardium before operation. Bilateral pleural effusions can also be seen.

cause of myocardial constriction solely on the basis of preoperative diagnostic findings. Thus, close monitoring of the intraoperative responses of the heart is imperative. Only by so doing can the surgeon identify and eliminate adequately the actual cause of the myocardial constriction.

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Perfusion Without Systemic Heparinization for Rewarming in Accidental Hypothermia

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We report on a patient with accidental deep hypothermia (23.3°C) and cardiorespiratory arrest resulting from severe craniocerebral injury. Systemic anticoagulation was contraindicated, and the decision was reached to rewarm the patient with cardiopulmonary bypass without systemic heparinization using heparin-coated perfusion equipment. The patient was successfully rewarmed, was weaned from cardiopulmonary bypass, and recovered.

(Ann Thorac Surg 1991;52:560–1)

It has been recognized that in low body temperature clinical death is not synonymous with biological death as neurological examination in profound hypothermia is inconclusive regarding prognosis. Hence, the necessity of raising the core temperature in these patients is evident [1] and rewarming with cardiopulmonary bypass (CPB) appears to be most promising as previously reported [2, 3]. In the past, however, this most efficient approach could not be applied to numerous patients with major contraindications to anticoagulation, eg, in severe polytrauma or in cerebral trauma. Under these circumstances, extracorporeal blood rewarming without systemic heparinization using perfusion equipment with improved biocompatibility [4–6] appears to be indicated.

A 13-year-old boy was strangulated in a forest in the late afternoon and suffered severe craniocerebral trauma. The boy was found the next day at noon (approximately 18 hours later). At that time hypothermic cardiorespiratory arrest was diagnosed. The epitympanic temperature was 23.3°C. Neurological examination showed a deep coma (Glasgow coma scale = 3) with a large, nonreacting pupil on the left side. The right pupil could not be examined because of grotesque periorbital and frontal swelling and hematoma. After tracheal intubation and the usual resuscitation maneuvers cardiac defibrillation was attempted three times on the scene, and the patient was immediately flown to our emergency center in Zürich. Cardiac massage was continued in the helicopter. Upon his arrival we recorded ventricular fibrillation, and a core temperature of 24°C was measured rectally. At this temperature, rewarming with CPB was clearly indicated. Considering the multiple contusions, binocular hematoma, and the previously described grotesque frontal lesions suggesting skull fractures and cerebral contusions, we decided, based on our previous experiences with heparin-coated CPB equipment [4–6], to perfuse the patient without systemic heparinization.

A median sternal splitting incision was performed under cardiac massage (sternotomy was selected primarily for this pediatric patient with unknown age and body weight). Ascending aorta and right atrium were cannulated and connected to the pump oxygenator with asanguineous priming. All blood-exposed surfaces of the perfusion equipment used including cannulas, tubing set, oxygenator-heat exchanger structure, and arterial filter were heparin coated (Duraflo II; American Bentley, Irvine, CA). No heparin was given. Rewarming was started with an arterial blood temperature of 24°C. The lowest activated clotting time observed during perfusion was 172 seconds. Within 12 minutes, the core temperature had increased to 26°C and sinus rhythm occurred spontaneously. However, ventricular fibrillation reappeared after 40 minutes and electrical defibrillation proved to be unsuccessful. At this time, the serum potassium level, which had changed from an initial level of 9.5 mmol/L (creatine kinase level, 27,216 U/L; normal value in male adults, less than 270 U/L) before bypass to 6.4 mmol/L after 20 minutes of perfusion, was again 9.3 mmol/L. Standard measures such as infusion of 50% glucose with insulin were not effective, but a hemofilter connected to the pump oxygenator allowed us to reduce the serum potassium level to 7.8 mmol/L. Weaning from cardiopulmonary bypass became possible at a core temperature of 35°C. However, the potassium level increased again to 9.0 mmol/L despite peritoneal dialysis and ongoing glucose and insulin application. After 43 minutes, emergency recannulation became necessary because of recurrent ventricular fibrillation. This time, the potassium level was reduced to 5.4 mmol/L by combined hemofiltration with the pump oxygenator, venovenous hemofiltration, peritoneal dialysis, glucose-insulin therapy, and enteral polystyrolsulfouat application. The patient was weaned again from CPB. A second hemofiltration unit was connected to the contralateral femoral vein when the potassium level increased again to 8.7 mmol/L despite ongoing diuresis and the previously described measures.

At the end of perfusion, the CPB equipment was carefully rinsed and thoroughly analyzed. Some fibrin strands were detected at the level of connectors but there were no macroscopic clots. The arterial filter (Fig 1) was completely clean. The postoperative course of the patient was complicated by important rhabdomyolysis with creatine kinase levels up to 1.5 million U/L requiring bilateral

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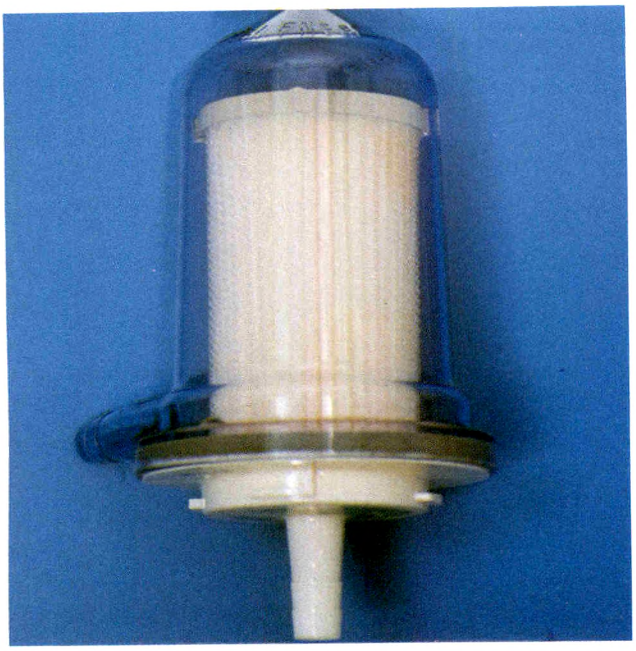

Fig 1. Heparin surface-coated arterial filter carefully rinsed after cardiopulmonary bypass without systemic heparinization: clean device. The arterial filter exposes the screen (pore size, 25 μm) on its inlet side. Red clots or fibrin strands that are generated in the perfusion circuit (venous reservoir, pump loop, heat exchanger, and oxygenator) and embolize into the arterial line would be trapped here. However, there are no macroscopic red or white deposits visible.

fasciotomies on both lower extremities, consecutive anuria requiring peritoneal dialysis for more than 3 weeks, and the bilateral cerebral contusions (computed tomographic scan demonstrated skull fractures) requiring intracranial pressure monitoring. Weaning from the respirator was possible after 3 weeks, and peritoneal dialysis was suspended after 4 weeks. Progressive somatic and neurological recovery followed and the patient now even remembers the details of the attack.

Comment

Systemic heparinization can be avoided during clinical CPB by using perfusion equipment with improved biocompatibility. We [6] have previously reported that perfusion for coronary artery revascularization can be realized with low doses of heparin (activated clotting time greater than 180 seconds) similar to the amounts given during vascular operations. A significant reduction in postoperative blood loss, which obviously resulted from the reduced heparin and protamine doses, was the main finding in this study. The approach selected in this case was further based on our animal studies [4, 7, 8] showing that high-flow and low-flow perfusion could be realized by means of heparin-coated equipment for up to 24 hours without systemic heparinization. No device failures were observed in the cited experimental and clinical studies. This also holds true for the case described: no macroscopic clots were observed in the cardiopulmonary bypass equipment and no clots were trapped in the arterial filter (see Fig 1) despite perfusion for 96 minutes and reperfusion for 43 minutes without systemic heparinization. However, a number of technical aspects have to be respected for perfusion with low or no systemic heparinization. Presently, cardiotomy suction cannot be used as no athrombogenic cardiotomy reservoir is available yet. Furthermore, blood stagnation in the perfusion system has to be avoided to prevent clotting. For this purpose, a special shunt is used in the operating field allowing recirculation between the arterial and the venous lines as well as purging of the cannulas. Hence, immediately after weaning from cardiopulmonary bypass, recirculation through the shunt is started.

We conclude from this experience that perfusion without systemic heparinization can be realized in exceptional cases with contraindications to systemic anticoagulation. However, further studies are necessary to determine the ultimate safety of such procedures.

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Peritoneopericardial Diaphragmatic Hernia Discovered at Coronary Bypass Operation

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A 68-year-old man was discovered to have a large peritoneopericardial hernia when operated on for coronary artery bypass. Such hernias are very unusual. The pathogenesis, clinical presentation, and management of this problem are discussed.

(Ann Thorac Surg 1991;52:562–3)

Diaphragmatic hernias involving the central portion of the diaphragm resulting in peritoneopericardial communication are rare. They may be congenital or traumatic in etiology [1–6]. We report a case of herniation of peritoneal contents within the pericardial sac discovered during a routine coronary artery bypass operation.

The patient is a 68-year-old man with worsening angina that was poorly controlled with appropriate medications. He had undergone percutaneous transluminal coronary angioplasty 6 months earlier with temporary relief of angina. He did not have respiratory or abdominal complaints. The past medical history is remarkable for two episodes of trauma: a motor vehicle accident 19 years earlier, causing a concussion, and a remote horse-riding accident resulting in multiple extremity fractures.

Cardiac catheterization demonstrated good left ventricular function and severe coronary artery stenoses involving the left anterior descending, circumflex, and right coronary systems. On physical examination, the breath sounds were normal, the heart rhythm was regular, and heart sounds were normal. The abdomen was flat, soft, and nontender. Peripheral pulses were full and equal. The initial preoperative chest radiograph suggested pneumopericardium, but this was not seen on a follow-up chest radiograph the same day.

At operation, the anterior mediastinum appeared normal after division of the sternum. However, the pericardial cavity was noted to be filled with abdominal contents, specifically the omentum, transverse colon, and left lobe of the liver. Further examination revealed herniation through a large defect in the central portion of the diaphragm. No hernia sac was present.

The diaphragmatic defect was repaired with a patch of expanded polytetrafluoroethylene after reduction of abdominal viscera (Fig 1). The remainder of the coronary artery bypass operation proceeded without complications, and the postoperative course was without untoward events.

Comment

Most congenital and acquired diaphragmatic hernias occur through natural diaphragmatic defects such as the foramen of Morgagni or foramen of Bochdalek, in cases of congenital hernias, or the esophageal hiatus in acquired hernias. The most common exception is traumatic diaphragmatic herniation, which usually involves the left dome of the diaphragm.

Central diaphragmatic hernias may be congenital or traumatic in etiology. Those permitting free communication between the peritoneal and pericardial cavities are rarely encountered. When symptoms occur they are likely to be nonspecific and may include dyspnea, palpitations, cyanosis, chest pain, or abdominal complaints [7]. Anginalike attacks are not uncommon. Cardiac tamponade and pericardial effusions are unusual, but have been reported [8, 9]. The chest radiograph is abnormal in the majority of cases, usually showing air within the pericardium. Pneumoperitoneum was advocated in the past as a

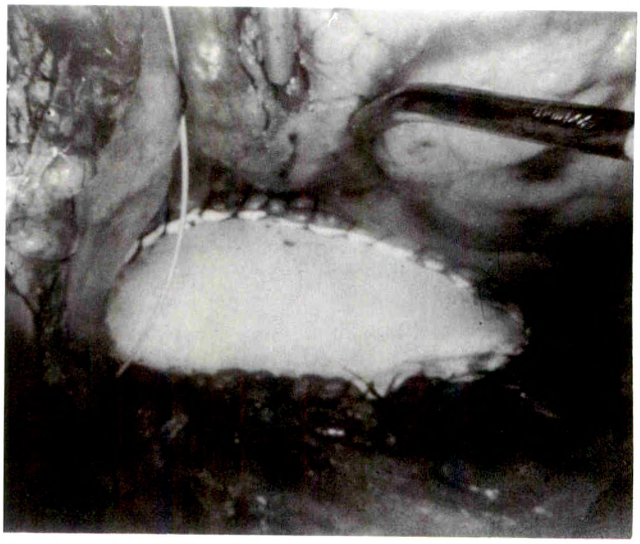

Fig 1. Completed polytetrafluoroethylene patch repair of a large peritoneopericardial hernia.

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confirmatory test, although this has been largely supplanted by computed tomography and other imaging techniques [10].

Patients with peritoneopericardial hernias should be managed surgically as they are subject to complications of strangulation, obstruction, and cardiac tamponade. The operative approach may vary, depending on the acuity and associated conditions. In the case of acute traumatic herniation, for example, in which other abdominal injuries may be present, repair from an abdominal approach is indicated. However, in chronic situations, adhesions to the pericardium and heart are likely to have developed, making exposure through the chest more desirable. A median sternotomy, as performed in this case because of concomitant coronary artery disease, provides excellent exposure and allows easy extension into the abdomen, if necessary.

Primary repair with nonabsorbable suture is satisfactory when the diaphragmatic margins are easily approximated without tension. Large defects, as in the case reported here, require patch repair, for which polytetrafluoroethylene is well suited.

Awareness of symptoms, signs, and radiographic features of peritoneopericardial herniation should alert the clinician to the possible existence of this rare condition. If herniation is confirmed by other tests, such as computed tomography, operative repair should be carried out.

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Optimal Visualization of Coronary Artery Anastomoses by Gas Jet

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A technique is described for improved visualization during the creation of coronary artery anastomoses employing a catheter-directed constant stream of oxygen to remove blood from the operative field. This technique is especially helpful when continuous blood cardioplegia is employed for myocardial protection.

(Ann Thorac Surg 1991;52:564)

Blood in the operative field poses difficulties in construction of distal anastomoses during coronary artery bypass grafting. This problem with visualization is encountered regardless of the method of myocardial protection used, whether antegrade or retrograde, intermittent or continuous infusion. Techniques that have been used in the past to deal with this problem include lowering systemic perfusion, stopping cardioplegia, snaring the coronary artery, occluding the coronary artery, and irrigating the arteriotomy with saline solution. However, these techniques are not ideal.

At the University of Toronto we have developed a new technique for improved visualization during coronary artery anastomosis employing a catheter-directed constant jet of oxygen to remove blood from the operative field.

Technique

At our institution patients undergoing coronary artery bypass grafting receive continuous warm blood cardioplegia as the method of choice for myocardial protection [1–4]. Warm blood cardioplegia is delivered continuously to the heart during construction of the distal coronary artery anastomoses. To provide an asanguineous operative field a continuous stream of oxygen is directed at the arteriotomy with a sterile, hand-held, tapered polyvinylchloride catheter (Argyle fine capacity Yankauer Suction Tube). The oxygen is supplied from the anesthetic machine at a regulated constant flow of 12 L/min (range, 10 to 15 L/min). We have found that this new method of visualization of the anastomosis is reliable, simple, and safe.

Comment

The optimal technique for visualization has not been found. Saline irrigation is a problem because of the changes in refractive index leading to parallax errors, and further, with longer cases the volume of saline solution required can lead to hemodilution of the cardiopulmonary bypass circuit. Mechanical methods such as snares or occluders may damage the coronary artery. Also, the occluders themselves can obscure the visualization of the heel and toe of the anastomosis.

We have found that this technique provides excellent visualization with all methods of myocardial protection. It is especially useful when using continuous blood cardioplegia because the anastomosis can be performed on a still, bloodless field under direct vision allowing the surgeon to perform an accurate anastomosis.

Potential adverse effects, such as coronary gas embolization, have not been a problem as long as the catheter is not placed directly into the arteriotomy. Trauma to the coronary artery is avoided by carefully regulating the gas flow. We have examined segments of saphenous veins subjected to gas flow and have not detected vascular endothelial injury. We have used oxygen from the anesthetic machine because it was readily available. However, we believe that other gases may be safely used, such as medical air or carbon dioxide.

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Dr Panos is a fellow of the Medical Research Council of Canada.

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Myocardium-Sparing Cannulation Technique for Left Ventricular Assist Device Support

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The left atrium and the left ventricular apex are the most commonly used sites of inflow cannulation for postcardiotomy left ventricular support. A new cannulation technique that requires only an ascending aortotomy is introduced. This procedure can be undertaken with equipment present in any cardiac operating room and may prove to cause fewer complications than conventional cannulation techniques.

(Ann Thorac Surg 1991;52:565–6)

Postcardiotomy shock that is too advanced for inotropic and intraaortic balloon pump therapy is frequently treated by left ventricular bypass. Several different pumps have been used for successful support of the failed ventricle, and most investigators have used either the left atrium or the left ventricular apex as sites for inflow cannulation. Complications have occurred at both of these sites. Thus, we developed a new cannulation technique for left ventricular support in which only the ascending aorta is cannulated.

Material and Methods

We used a new cannulation technique to institute left ventricular assist device support in 3 patients who could not be weaned from cardiopulmonary bypass after aortocoronary bypass grafting. None of the patients had evidence of aortic valve disease.

In each case, a Bio-Medicus centrifugal pump (Bio-Medicus, Minneapolis, MN) and standard Tygon tubing were used. A longitudinal aortotomy was performed on the right lateral aspect of the ascending aorta, proximal to the aortic cross-clamp, while cardiopulmonary bypass was continued. A 22 × 11-mm woven Dacron bifurcation graft was fashioned so that only 5 cm of the body and two limbs remained. The body was beveled so that it could be directed inferiorly. No preclotting of the graft or other preparation was necessary. A 3-0 polypropylene running suture was used to anastomose the 22-mm limb of the graft to the ascending aorta (Fig 1). A 24F wire-reinforced cannula (USCI, Billerica, MA) was then inserted through the superior limb of the graft and across the aortic valve, so that it sat in the left ventricular chamber near the apex. Umbilical tape was used to secure the graft around the cannula. The cannula and ventricle were aspirated and connected to the inflow tubing of the pump. The return cannula was placed through the remaining limb of the graft, directed distally past the anastomosis and into the aorta, and secured to the graft with umbilical tape. After the circuit was aspirated (Fig 2), left ventricular support was instituted.

The patients were successfully weaned from cardiopulmonary bypass, and the heparin was allowed to wear off. Standard chest tube drainage was performed. Both cannulas were brought out the inferior aspect of the sternotomy incision and secured to the skin. The patients were then transported to the intensive care unit for postcardiotomy care.

Results

The technique of ascending aorta cannulation to institute left ventricular assist device support was performed rapidly in all 3 patients, thereby minimizing bypass times. Excessive bleeding at the anastomosis, around the umbilical tape, or through the graft did not occur. Chest tube

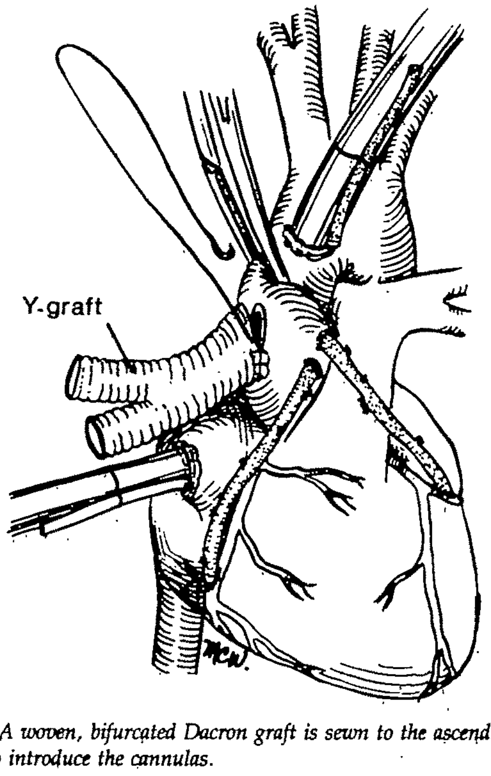

Fig 1. A woven, bifurcated Dacron graft is sewn to the ascending aorta to introduce the cannulas.

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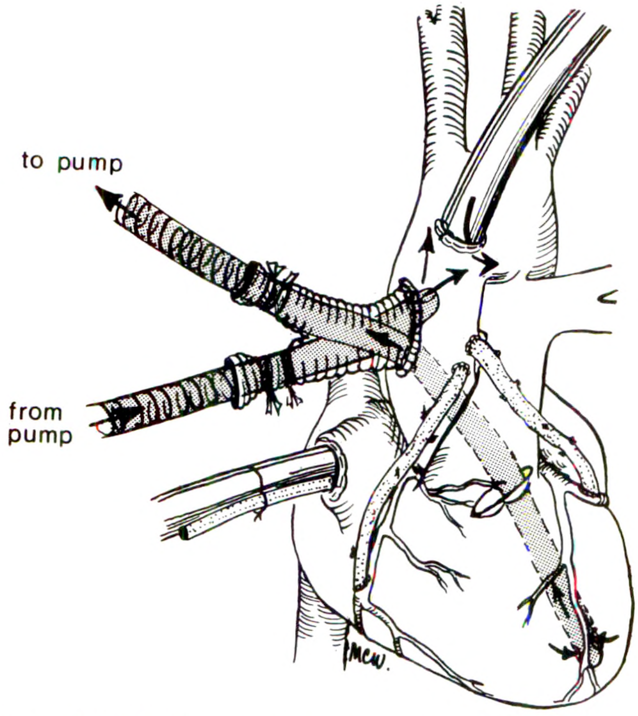

Fig 2. The reinforced cannula is placed across the aortic valve to decompress the left ventricle. Blood is returned from the centrifugal pump through a cannula inserted into the ascending aorta.

output ranged from 50 to 300 mL/h and was believed to be the result of coagulopathy, which commonly occurs in this patient population. The average pump flow was 4 to 6 L/min in all patients and was highly dependent on the patient's intravascular volume status. The ventricle appeared to be decompressed, and the pulmonary capillary wedge pressure remained low postoperatively. The cannulas did not kink or impede inflow, even when directed inferiorly below the skin at the time of closure.

The duration of support ranged from 6 hours to 3 days, and pump function was excellent. No focal neurological insults occurred. All 3 patients died of irreversible cardiac failure.

Comment

Left atrial cannulation to institute left ventricular assist device support is easy to perform, but this procedure may cause complications such as mitral valve damage, incomplete ventricular decompression, inflow cannula obstruction by the pliable walls of the atrium, and increased right-to-left shunting of blood in the presence of an unrecognized patent foramen ovale [1]. Inflow cannulation of the ventricular apex is time-consuming, and it may cause bleeding—either during the period of support or after repair following successful weaning—and regional hypokinesis in the surrounding myocardium, thereby limiting ultimate recovery [2, 3].

The technique of ascending aorta cannulation described in this report, which was inspired by Golding's work in calves using the descending thoracic aorta cannulation site [4], seems to have several advantages. It is simple and fast, and it requires only a Bio-Medicus pump, standard cannulas, and a bifurcated woven Dacron graft. In addition, the technique allows direct ventricular decompression but avoids the complications of bleeding and hypokinesis associated with direct ventriculotomy. Although more experience with this procedure is necessary before any solid conclusions can be drawn, we believe that this technique may prove satisfactory for cannulating patients requiring postcardiotomy left ventricular support.

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Preoperative Duplex Scan Assessment of the Inferior Epigastric Artery as a Coronary Bypass Conduit

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The inferior epigastric arteries are currently being evaluated as alternative autologous arterial conduits for coronary artery bypass operations. The inferior epigastric arteries are variable in diameter and length and require harvesting through separate abdominal incisions. There is a need, therefore, for a method to preoperatively assess the diameter and length of the inferior epigastric arteries to determine their suitability as a coronary artery bypass graft. We have found that the duplex scan is a valuable noninvasive preoperative imaging modality to evaluate the inferior epigastric arteries.

(Ann Thorac Surg 1991;52:567–8)

The internal mammary artery is the conduit of choice for myocardial revascularization [1]. The saphenous vein, although the most commonly used conduit, has a reduced long-term patency rate [1]. Therefore, the search for alternative autologous conduits continues. The gastroepiploic artery has been recommended as an additional arterial conduit, but its long-term patency is unknown [1]. We are currently evaluating the inferior epigastric artery (IEA) as an alternative autologous arterial conduit for coronary artery bypass grafting. Recent reports describe similarity between the IEA and internal mammary artery in terms of diameter, wall characteristics, and low incidence of atherosclerosis [2–5]. However, a substantial variability in diameter and usable length of the IEAs have been reported [3, 4, 6, 7]. Contrary to the internal mammary artery and gastroepiploic artery, which are harvested through a midline sternotomy incision, the IEAs are harvested through separate abdominal paramedian incisions. In view of the variability in vessel size and usable length and of the separate harvesting incisions, planning the operative procedure dictates a need for a preoperative method to assess the suitability of the IEAs as coronary artery bypass grafts. Selective injections of the IEAs during cardiac catheterization are difficult and would increase the quantity of angiographic contrast medium used. We have found that preoperative duplex scan can accurately define the usable length, diameter, and presence of atherosclerosis of the IEA.

Method

Use of the IEAs is currently considered for young patients, in an attempt to use more arterial grafts, in patients with absent or poor-quality veins, in reoperations for vein graft failure, and in insulin-dependent diabetics to avoid the use of bilateral internal mammary arteries. In these patients, preoperative duplex scan (ultrasonic imaging and Doppler flow-signal analysis) of the IEAs is undertaken (Fig 1).

The IEA is visualized by passing the duplex scan probe (7.5-MHz Linear Array Probe; Diasonics, Milpitas, CA) on the abdominal skin parallel to the course of the artery. The origin is detected at the medial aspect of the distal external iliac artery at the level of the inguinal ligament. The artery is then followed cephalad, medial to the lateral border of the rectus muscle, up to the level of the umbilicus. The vessel can be visualized in the areolar tissue between the rectus muscle and posterior rectus sheath. Diameter, length, flow velocity, and wall characteristics are evaluated. These findings were correlated with operative findings.

Results

Twenty-eight patients were evaluated preoperatively by duplex scan of the IEAs. Inferior epigastric arteries were harvested from 22 patients in whom the scans visualized the arteries for a length of 5 to 8 cm (6.4 ± 0.7 cm) with an internal diameter of 2.2 to 3.1 mm (2.6 ± 0.2 mm). At operation, the usable IEA vessel length was 6 to 14 cm (10.6 ± 2.1 cm), and the distal diameter was 2.0 to 3.0 mm (2.4 ± 0.3 mm). All segments were used as coronary artery bypass grafts. The discrepancy between the IEA vessel length visualized by duplex scan and the actual vessel length found at operation can be explained by difficulty in scanning the first 2 to 4 cm of the IEA at its origin from the external iliac artery, for in most patients the vessel does not follow a straight path in that segment.

In 6 patients the scans demonstrated small caliber (less than 1.9 mm) or early bifurcations of the IEA (3 to 5 cm from its origin), and in these cases the arteries were not

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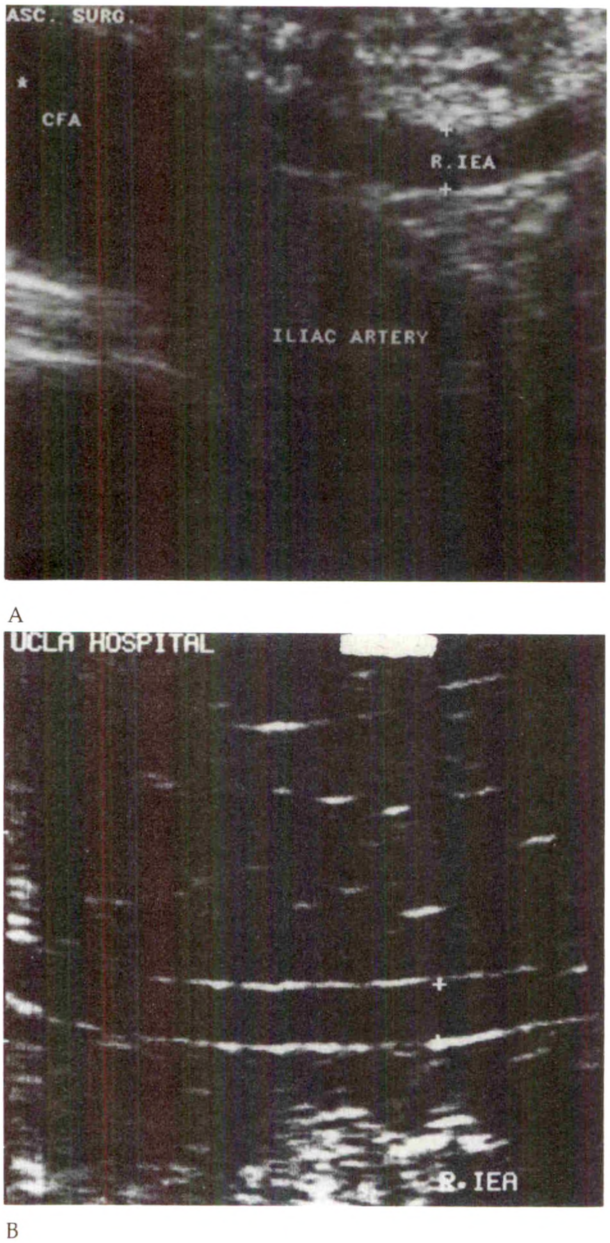

Fig 1. *Imaging of the right inferior epigastric artery* (R. IEA) *by duplex scan. (A) Internal diameter is 3 mm at its origin from the right external iliac artery. (B) Further cephalad, the artery is visualized posterior to the rectus muscle, with an internal diameter of 2.6 mm. This artery could be detected for a length of 7 cm by scan. At operation, the usable length was 11 cm, proximal diameter was 3 mm, and distal diameter was 2.5 mm.* (CFA = *common femoral artery.)*

harvested, saving the patients unnecessary incisions (Fig 2).

Comment

The IEA may become an alternative autologous arterial conduit for coronary artery bypass grafting. Duplex scan appears to be a valuable noninvasive tool to preoperatively evaluate the IEA. It may direct the surgeon as to the side with the larger IEA, and may prevent unnecessary incisions when the scan demonstrates small caliber, early branching, or atherosclerotic changes in the IEA wall. In our experience, whenever the duplex scan detected the IEA for a length greater than 5 cm and measured a diameter greater than 2 mm, the IEA was usable as a coronary artery bypass graft.

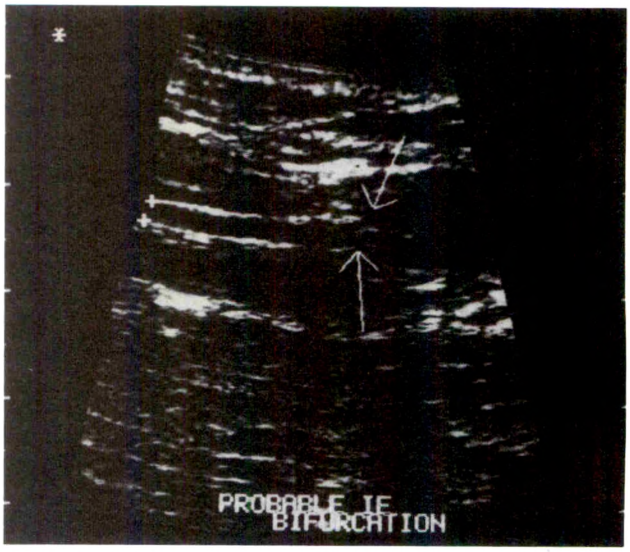

Fig 2. *Small diameter (1.8) and early bifurcation of the inferior epigastric artery* (IE) *as demonstrated by a preoperative duplex scan.*

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Resternotomy in Patients With Valved Conduits Adherent to the Sternum

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Twenty-two patients with valved conduits adherent to the sternum underwent resternotomy. Mean age was 10 ± 6 years, and mean conduit age was 4 ± 4 years. Diagnoses were D-transposition (7), truncus arteriosus (7), univentricular heart (6), Taussig-Bing anomaly (1), and corrected transposition (1). The majority of patients (68%) had reoperation for outgrown or degenerated conduits. In 17 patients, the sternum was opened with a chisel. Two of these patients sustained conduit neointimal collapse from manipulation, and 3 had conduit tear requiring immediate cardiopulmonary bypass through the femoral vessels. In the last 5 patients, the sternum was opened above and below the conduit, and the inner table was chiseled and left attached to the conduit avoiding injury and undue conduit manipulation. Cardiopulmonary bypass and operation were carried out uneventfully. We believe that the recent technique described provides a safe alternative approach to valved conduits adherent to the sternum.

(Ann Thorac Surg 1991;52:569–71)

Valved conduits adherent to the sternum pose a special problem on reoperation. Serious morbidity, such as conduit tear and neointimal collapse, can occur during the initial dissection that may necessitate immediate institution of cardiopulmonary bypass through the femoral vessels [1, 2]. This is a report of our experience in patients with valved conduits adherent to the sternum requiring reoperation.

Material and Methods

Patients

Over a 9-year period ending in 1989, 22 patients with valved conduits adherent to the sternum underwent reoperation. Ages ranged from 2 to 19 years (mean age, 10 ± 6 years). The diagnoses were D-transposition of the great arteries (7), truncus arteriosus (7), univentricular heart (6), Taussig-Bing anomaly (1), and corrected transposition (1). The age of the conduit or length of time the conduit had been implanted at the time of reoperation ranged from 2 months to 11 years (mean, 4 ± 4 years).

Indications for operation included outgrown or degenerated conduit (15), aortic insufficiency after a Damus-Stansel-Kaye procedure (2), occluded conduit (2), conduit infection (1), tricuspid patch disruption (1), and thrombosed right atrium to right ventricle prosthetic valve (1), all after a Fontan procedure.

Surgical Technique

Both sternal and groin areas were prepared. The incision was made and carried down to the sternum. With the edges of the linea alba being separated by a self-retaining retractor, the outer table of the sternum was opened from below using the chisel and mallet technique (Fig 1A, B). The inner table was also opened from below onto the area of the conduit. In the early experience, when injury to the conduit would occur or the conduit would be quite adherent to the sternum, cardiopulmonary bypass would be instituted through the femoral vessels before completely splitting the inner table. In the last 3 years, we adopted a technique of dividing the inner table from below onto the conduit as described above. Should the conduit be found quite adherent to the sternum, the inner table superior to the conduit would then be divided from above using the chisel, leaving the segment over the conduit intact. With self-retaining retractors the sternal edges above and below the conduit were separated. The conduit was then separated from the sternum using the chisel with a thin layer of inner table attached to it (Fig 1C–E). Once the conduit was freed, the sternal edges could be fully retracted, dissection continued, and the planned procedure performed.

Results

There was no intraoperative death. There was one hospital death (4.5%) in a 9-year-old boy with truncus arteriosus who was doing well and suddenly had cardiac arrest, probably from arrhythmia, on the fifth postoperative day. Three patients sustained bleeding from conduit tears, which were temporarily controlled while cardiopulmo-

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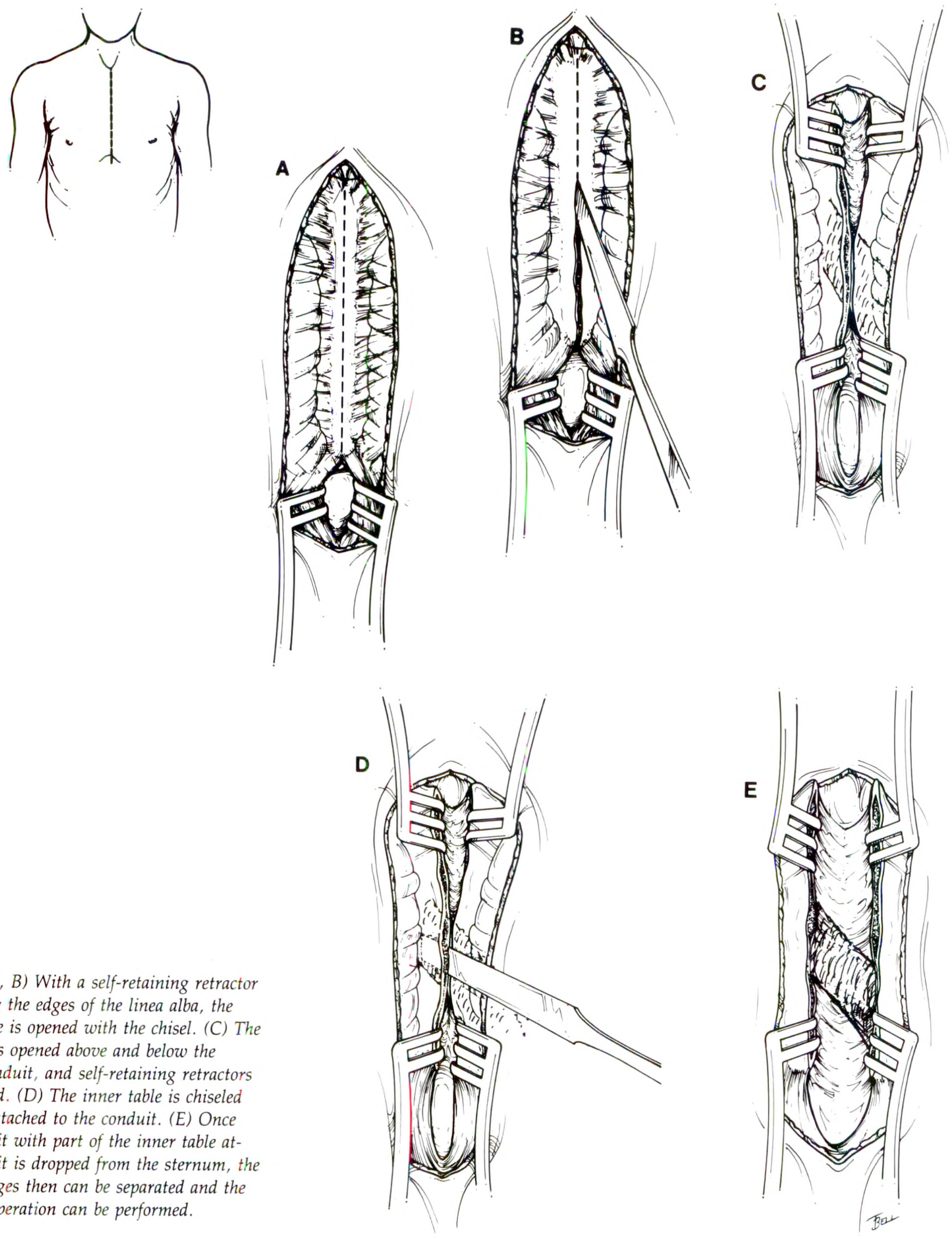

Fig 1. (A, B) With a self-retaining retractor separating the edges of the linea alba, the outer table is opened with the chisel. (C) The sternum is opened above and below the valved conduit, and self-retaining retractors are applied. (D) The inner table is chiseled and left attached to the conduit. (E) Once the conduit with part of the inner table attached to it is dropped from the sternum, the sternal edges then can be separated and the planned operation can be performed.

nary bypass was being instituted through the femoral vessels. Conduit obstruction from neointimal collapse developed in 2 patients with a Glenn shunt and Fontan operation undergoing repair of a disrupted tricuspid patch. In one of these 2 patients the problem was detected at the time of operation, prompting clearance of the conduit of neointima. In the other patient, replacement of an obstructed conduit was performed 6 months later. Five patients whose conduits were quite adherent to the sternum had uneventful operation using the recent technique.

Comment

The use of valved conduits remains an important tool in the surgical management of complex congenital heart defects. Because there is a trend toward early repair and limited longevity of the valved conduit, replacement will be required for outgrown and degenerated conduits [3–6]. Kirklin and colleagues [7] reported that 40% of xenografts or irradiated allografts would require replacement within 10 years of implantation.

The location of the valved conduit in relation to the sternum and the age of the conduit will determine the safety of resternotomy. The valved conduit in severe forms of tetralogy of Fallot, such as pseudotruncus, is usually located to the left side of the sternum, thus making resternotomy relatively safe. The valved conduit in transposition of the great arteries, truncus arteriosus, and Taussig-Bing anomalies often passes under the sternum and will pose some difficulties on reoperation. Valved conduits are now rarely used for the atrioventricular or pulmonary connection in patients with univentricular heart undergoing the Fontan operation [8].

Reoperation on patients with valved conduits carries significant morbidity and mortality rates. Schaff and colleagues [1] reported an 18% (18/100) incidence of hemorrhage as a result of laceration of the myocardium or injury to the conduit in patients undergoing replacement of obstructed pulmonary ventricle to pulmonary artery conduit. Institution of cardiopulmonary bypass was accomplished through the femoral artery in 68% of patients. They reported a 7% mortality. None of the deaths were directly attributed to intraoperative conduit injury, although when stratified by diagnosis the mortality rate was 25% among patients with truncus arteriosus, where there is a high possibility of the conduit passing under the sternum. Boyce and colleagues [3] reported an 18% (5/28) mortality rate in patients undergoing replacement of 12-mm porcine valved conduit from the right ventricle to the pulmonary artery. Although the direct cause of death was not clearly reported, 78% of their patients had truncus arteriosus.

Several surgical approaches are available to patients with valved conduits adherent to the sternum. Cardiopulmonary bypass can be accomplished through the femoral vessels followed by resternotomy, or resternotomy can be initially performed with cardiopulmonary bypass instituted through the femoral vessels in the event of difficulties. The technique we describe provides an alternative approach to patients with valved conduits adherent to the sternum that might minimize conduit injury and neointimal collapse.

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INVITED COMMENTARY

This report recommends a technique that has been effective in five operations. Reoperations of this type are hazardous, and the authors' overall results in 22 patients are excellent with and without the sternal sculpturing herein described. The dangers of this technique are obvious from the illustrations, and its "safety" is certainly unproved at this time. It does, however, seem reasonable to present the technique as an expansion of the surgeon's armamentarium in dealing with this difficult situation.

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Surgical Cure of Cardiac Arrhythmia

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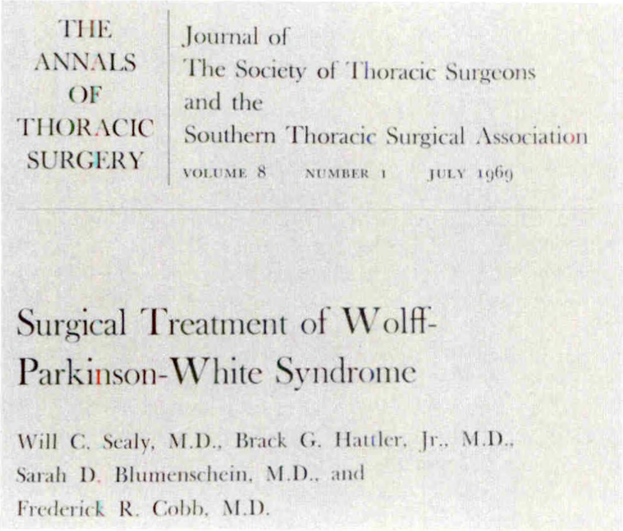
The first surgical procedure undertaken for the intended purpose of curing a patient with recurrent tachycardia was performed by Sealy in 1968, and the report of that operation was published in *The Annals of Thoracic Surgery* in 1969. The first attempted accessory bundle ablation was successful, marked the beginning of cardiac arrhythmia surgery, and has set the standard against which other methods of arrhythmia control must be measured.

(Ann Thorac Surg 1991;52:572–5)



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Surgical Treatment of Wolff-Parkinson-White Syndrome

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Few cardiac surgical procedures offer the opportunity for a complete cure of the patient. Almost all heart operations, whether for congenital or acquired disease, are only palliative in nature and do not result in normal cardiac function and a medication-free and restriction-free normal lifespan for the patient. One of the few exceptions is the surgical ablation of accessory (Kent) bundles in patients with Wolff-Parkinson-White (WPW) syndrome. Sealy and associates' account [1] of the first such procedure, actually the description of the birth of arrhythmia surgery, was published in *The Annals of Thoracic Surgery* in 1969.

The WPW syndrome was first described by Drs Wolff, Parkinson, and White [2] in 1930, but the mechanism of dysfunction in this syndrome of paroxysmal tachycardia in healthy young people with short PR intervals was not understood. In 1914, Kent reported the existence of multiple conduction pathways between the mammalian atrium and ventricle [3]. Although he incorrectly considered such anomalous pathways to be normal, his description led to the eponym for the Kent bundle. In 1933, Wolferth and Wood [4] proposed that the cause of abnormal electrocardiographic tracings and paroxysmal tachycardia in patients with otherwise normal hearts was due to "an accessory pathway of [atrioventricular] conduction (such as that described by Kent, between the right auricle and the right ventricle)." Ten years later, Woods and colleagues [5] reported the detailed autopsy dissection of the heart of a young man who had been followed up for several years with the WPW syndrome before his sudden death. Their painstaking histologic studies confirmed the presence of a right lateral free wall muscle bundle connecting the atrium to the ventricle and thus provided the anatomic-physiologic-electrocardiologic link to explain the WPW syndrome.

In 1967, the pattern of normal and abnormal epicardial excitation was investigated by Durrer and Roos [6], and later that year Burchell and associates [7] described the intraoperative temporary abolishment of tachycardia in a patient with the WPW syndrome by the injection of procaine into the right ventricular muscle at the site of earliest ventricular activation. Thus, the technique of intraoperative electrophysiologic epicardial mapping was initiated and used to locate the accessory bundle and allow for temporary obtundation by drug injection. The next logical step was an attempt at permanent relief from arrhythmia by surgical ablation of the anatomical abnormality.

In February 1968, a 32-year-old fisherman was evaluated at Duke University for type B WPW syndrome with heart failure secondary to uncontrolled supraventricular tachycardia. Medical therapy was unsuccessful. Surface electrocardiographic studies indicated that the site of early ventricular excitation was at the right lateral free wall. In May 1968, Sealy performed the first direct surgical procedure to cure a cardiac arrhythmia. Through a median sternotomy, the patient's heart was exposed and photographed for epicardial mapping landmarks. Sinoatrial node to right ventricular conduction times were recorded

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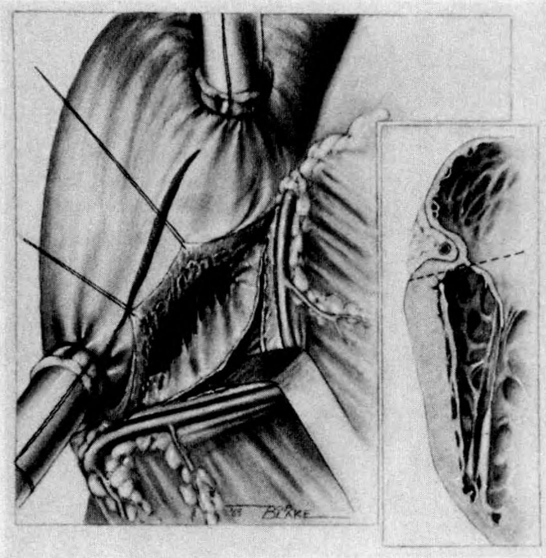

FIG. 4. With the patient on cardiopulmonary bypass, the right auricle is opened. An incision is then made through the ventricle (insert). The tricuspid valve is separated from the ring. A margin of 1 cm. is then made on each side of the 1.5-cm. band demonstrated by epicardial mapping to be the bundle of Kent.

Fig 1. Figure from Sealy's 1969 paper illustrating the approach used for the first Kent bundle ablation. (Reprinted with permission from [1].)

along the atrioventricular groove, and the area of earliest ventricular activation was confined to a region only 1.5 cm in width. The right coronary artery was mobilized, and the patient was placed on cardiopulmonary bypass. Through a right atriotomy, a 3.5-cm endocardial incision was made through the ventricular musculature, 3 mm below the atrioventricular groove (Fig 1). No anatomical abnormalities or aberrant tissue that might be identified as the anomalous bundle could be identified. The decision on where to separate the atrium and ventricle was made entirely on the basis of the epicardial mapping procedure, and normal sinus rhythm resumed as soon as the incision was made. The patient remained in sinus rhythm, free of tachycardia, during the 9 months of reported follow-up, and his heart failure subsided [1].

The fundamentals established in this landmark surgical approach to cardiac arrhythmias are still important to the practice of arrhythmia surgery today. (1) Careful patient selection: Although the electrocardiographic findings of short PR interval and delta waves may occur in 1 of 1,500 young patients, operation is reserved for selected symptomatic patients. (2) Appropriate preoperative evaluation: Available techniques are used for electrophysiologic evaluation to identify the site of genesis of the rhythm disturbance. In 1968, this involved isopotential body surface mapping; today, catheter electrophysiologic testing is performed. (3) Intraoperative mapping before and after ablation, when possible. This is performed to confirm the site of interest and the effectiveness of the ablative procedure. (4) Teamwork: Close cooperation is essential between the operating surgeon and the participating electrophysiologists.

The technique of accessory bundle ablation has evolved from Sealy's original ventricular incision to the supraannular endocardial dissection and epicardial approaches. Sealy's descriptions of the surgical anatomy, based on electrophysiological mapping, anatomical dissections, and surgical exploration, led to the extension of surgery to the ablation of right and left free wall and posterior and anterior septal pathways [8, 9].

Cure of the WPW syndrome is achieved in more than 95% of the patients who undergo accessory bundle ablation, and operative mortality is less than 0.5%. Successful ablation results in a patient with normal cardiac function who requires no medications and has no restrictions, and whose prognosis for a normal lifespan is excellent. Thousands of patients have been cured of this congenital abnormality that can threaten both the quality and duration of life. Furthermore, the principles of successful arrhythmia surgery have been applied subsequently to the ablative surgical treatment of other arrhythmogenic anatomical abnormalities (atrioventricular nodal reentry, automatic atrial tachycardias, and ventricular arrhythmias), device therapy for ventricular arrhythmias, and surgery for atrial fibrillation.

The future of ablative surgery for the WPW syndrome remains uncertain at this time. Radiofrequency catheter ablation, a successful technique with a low complication rate, is replacing surgery at many institutions. This less invasive catheter procedure, however, developed as an extension of the surgical techniques and anatomical studies pioneered by Dr Sealy [3] as a result of his experience with the "fisherman with the fast pulse."

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INVITED COMMENTARY

The fisherman with WPW syndrome and uncontrollable tachycardia arrived at the Duke Hospital at just the right time to be cured and to be the first patient to have division of an accessory pathway of atrioventricular conduction. I would like to explain this favorable result as having occurred because of months of careful planning. The fact of the matter is that success was due to a combination of luck and serendipity, the latter defined according to Hugh Walpole, who introduced the word to our language. His definition was acting with sagacity after finding unsought-for agreeable things.

Shortly before the patient's admission, Andrew Wallace and John Boineau introduced clinical electrophysiologic studies to Duke. John had just returned from a year with Dirk Durer in Amsterdam. With surgical fellows, including James Cox, John was regularly performing epicardial activation sequences during open heart operations for a variety of reasons, including right bundle-branch block phenomenon. Thus, mapping equipment was ready to go in the operating room. Andrew Wallace, on returning to the senior staff at Duke from the National Institutes of Health, was given space in my laboratory. In return he helped me and a surgical fellow, James Holsinger, do studies on the atrial conduction system. This introduced me to electrophysiology.

The patient was known to have WPW syndrome from electrocardiographic tracings made on admission to the hospital in years past because of a benign brain tumor. A cardiology fellow, Frederick Cobb, recognized the patient's problem and realized the operation was the only solution. He was no doubt influenced by Durer's and Roos' epicardial activation studies on patients with WPW syndrome, the temporary interruption by procaine injection of an accessory pathway by Burchell and Magoon, and the correction by Robert Ellison of a reentry tachycardia in a patient with WPW syndrome by His bundle interruption with the electrocautery.

Although some of the next events may be described as serendipitous, pure luck explained most of them. The patient was admitted just at a time that preoperative electrophysiologic studies were available, thus allowing us to confirm the diagnosis. The equipment and experience of mapping the activation sequences of the epicardial surface of the ventricles were in place in the operating room, and thus the pathway, which is invisible, could be found. Fortunately it was in the approximate location described by the microscopic studies of Francis Wood and erroneously thought to be the location by Albert Francis Stanley Kent for normal conduction pathways, that is, on the right free wall. The pathway was then interrupted by dividing the entrance of the pathway to the ventricle just below the annulus fibrosus. Later on it became evident to me, as well as to Takashi Iwa, that an atrial incision for division of the pathway was more prudent than a ventricular one. Gerard Guiraudon subsequently advocated an epicardial plus cryoablation approach.

What would we have done had the pathway been anywhere else other than on the right free wall? Actually we were prepared to go ahead with His bundle interruption, although my experience in achieving this in the laboratory had not been good. This was before cryoablation was introduced.

Expanding this lesson to pathways in other locations was difficult. Continued progress occurred when John Gallagher returned to Duke from 2 years spent on Damato's Service at the Staten Island Marine Hospital. Electrophysiologic studies were expanded in both the clinic and operating room in patients with WPW syndrome as well as other arrhythmias. He introduced, from his laboratory, the use of cryothermia as an ablation method in arrhythmia surgery. Finally, with continued work on details of what are in effect exercises in normal anatomy, approaches to the accessory pathways anywhere around the atrioventricular junction were worked out. This resulted from careful analysis of electrophysiologic studies, preoperatively and postoperatively, and correlating these with the various surgical maneuvers used. This effort led to what is the accepted classification of pathways based on anatomical considerations rather than vector forces on the electrocardiogram. Progress in direct surgery for other supraventricular arrhythmias followed. Knowledge gained from these experiences has led to the development of other operations for supraventricular tachycardias by another group of surgeons, the so-called nonpharmacologic therapeutic cardiologists, who accomplish their operations with catheters.

The conclusion I can draw with certainty from this work is that both the fisherman and I were at the right place at the right time.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 16, 1992. The closing date for registration is August 1, 1991.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

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Cystic and Bullous Lung Disease

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One of the most difficult problems facing clinicians is the evaluation and management of patients with dyspnea whose chest roentgenogram shows single or multiple large cystic spaces. This is made more difficult when underlying lung disease is present. The dilemma focuses on whether the obliteration or removal of the cystic areas will benefit or cause further deterioration of the patient's condition. The pathophysiology of the problem is not completely understood, but accumulated clinical experience has shown that surgical therapy can be beneficial but requires proper patient selection.

(Ann Thorac Surg 1991;52:576–80)

Most physicians refer to cystic air spaces seen on chest roentgenograms as "bullae" and conclude that the patient has "bullous emphysema." This implies that all such lesions are part of a generalized destructive process, such as panlobular emphysema. Not commonly appreciated is that approximately 20% of patients with such roentgenographic findings do not have an underlying parenchymal abnormality and can benefit from surgical removal of the cyst [1, 2]. Important in the management of such patients is whether the cystic space is an isolated entity or part of a more generalized destructive process. The approach to therapy will differ on how this question is answered.

Definition and Classification

Historically, the terms *bulla, cyst,* and *bleb* have been used interchangeably, and this has led to confusion. More precisely, *cyst* is used to describe a congenital bronchial or bronchogenic cyst or an acquired cyst. The first two refer to a developmental abnormality resulting from abnormal budding and branching of the tracheobronchial tree. Such a cyst is located in the lung or mediastinum and is lined by ciliated cuboidal respiratory epithelium. An acquired cyst is a thin-walled space that remains as a residuum of an injury to the lung. It results from a check-valve obstruction of a small bronchiole that distends the distal lung to form a coalescent space or from inflammatory necrosis of the bronchial wall resulting in compression of adjacent pulmonary parenchyma to form a pneumatocele (Fig 1). Histologically, this parenchymal air space lacks an epithelial lining.

The term *bleb* usually connotes a subpleural collection of air within the layers of visceral pleura caused by a ruptured alveolus. The air dissects through the interstitial tissue into the thin, fibrous layer of visceral pleura where it accumulates to form a bleb. Rupture of a bleb is often associated with the development of a spontaneous pneumothorax.

The term *bulla* is used to describe an air-filled space within the lung parenchyma resulting from deterioration of the alveolar tissue. These lesions have a fibrous wall and are trabeculated by the remnants of alveolar septa. They can develop in a lung that is otherwise normal or in a lung in which the architecture has been destroyed by chronic obstructive disease. These are the lesions to which the term *bullous emphysema* is more appropriately applied.

Consequently, acquired cyst or bulla describes an air-space lesion that possesses a smooth wall of minimal thickness ranging in volume size from a cubic centimeter to one that fills the entire hemithorax. If the wall is thicker than 3 mm, it is often called a cavity. Although this term implies a different etiology, it may not be so, as both cysts and bullae can become infected and develop thick walls (Fig 2).

Patients with bullae or acquired cysts can be divided into two groups: those with structurally abnormal lung parenchyma in addition to the bulla or bullae and those with normal pulmonary parenchyma. The second group accounts for 20% of patients, but this figure can be misleading because of problems of semantics, which result in the inappropriate inclusion of patients with congenital cysts, blebs, and hyperlucent lungs. The last is due to agenesis or atretic pulmonary vessels or bronchi or to complete or partial bronchial obstruction, which can lead to bullous changes through the process of collateral ventilation and air trapping.

Controversies in the Pathophysiology of Bullous Lung Disease

The rationale for operation for bullous disease is to allow a normal, compressed lung to reexpand. This is based on the conventional view of the pathophysiology of bullous disease, which defines a bulla as a localized area of parenchymal destruction supplied by airways that contain a valvular obstruction allowing gas to enter but not leave the cystic space. Continued alveolar destruction and the inflating effect of gas entering the space expand it, causing compression and collapse of the surrounding lung. This has been conceptually referred to as an "intrapulmonary pneumothorax."

This concept has rarely been challenged despite evidence that a bulla is unlikely to grow in such a manner. It

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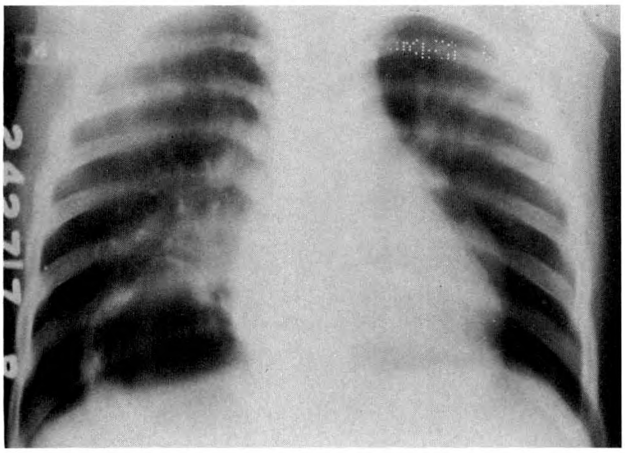

Fig 1. Tomogram of the chest shows a postinflammatory (staphylococcal) pneumatocele of the right lung.

has been observed during operation and on examination of resected specimens that in the floor of a bulla, there are bronchial openings that are widely patent with no evidence of a valvular mechanism [3]. It has been postulated that these openings could be floppy and close prematurely at low airway pressures and act as a trap valve. Such a valvular mechanism implies that either extreme negative pleural pressures result in preferential inflation of the bulla or the bulla is filled in expiration or during coughing. The latter is unlikely, as expiratory positive pleural pressure will be applied equally to the bulla and the lung, and the gas should follow the pressure gradient to the mouth. It is conceivable that positive pressures transmitted to the walls of bullae during coughing could enlarge the space by further rupture of the lung parenchyma, but this pressure would not be maintained during normal respiration [3].

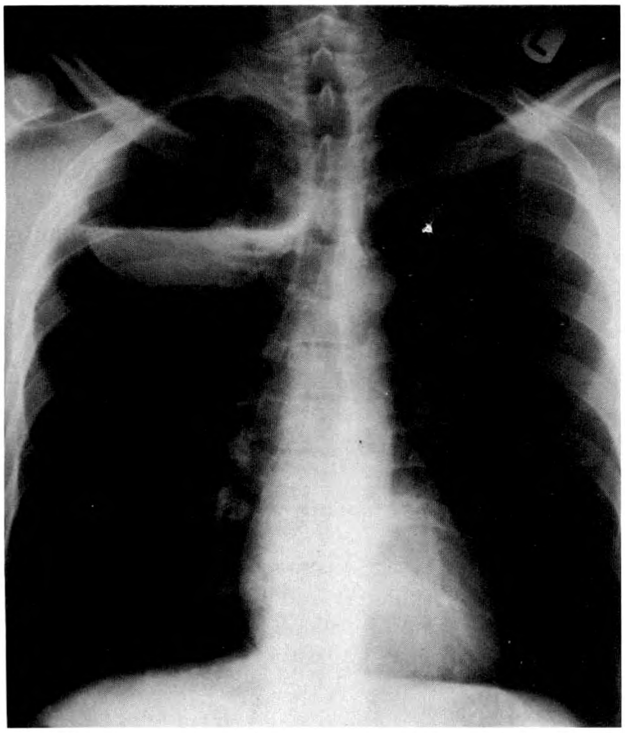

Fig 2. Chest roentgenogram demonstrates an infected cystic cavity of the right upper lobe with thickening of the walls and an air-fluid level.

Direct observation of the volume change and ventilation of bullae by computed tomography also contradicts the conventional pathophysiological view of bullous disease [4]. A bulla observed on computed tomography during inspiration and expiration does not change to any appreciable degree in size, and the change that does occur is always in phase with the rest of the lung during respiration, ie, growth during inspiration and reduction during expiration. This observation is further substantiated by recent studies [3] that show bullae contain gas that is identical to that in the alveoli during normal respiration. Overall gas flow into and out of bullae appears to be small but unimpeded [3, 5].

The evidence that a bulla compresses surrounding lung is based on the observation that adjacent lung appears collapsed on computed tomograms or at operation. Although it is agreed that collapsed lung may adjoin a bulla, the lung need not be compressed for this to occur. In fact, it is more likely for computed tomography not to show evidence of adjacent collapsed lung, and the density of the peribullous lung is not increased by maneuvers that compress the lung. Morgan and colleagues [3] measured pressures within a bulla and related them to simultaneous measurements of airway and pleural pressures during the induction of anesthesia. Pressures in the bulla were never positive in inspiration and were never more positive than pleural pressure at end-expiration, and the pressure swings were always in phase. This suggests that at least during spontaneous respiration, bullae are spaces that are subject to the same forces as the surrounding lung and respond in a similar manner.

If a bulla is not expanded by positive pressure, then how does it inflate preferentially and assume a spherical shape? Ting and co-workers [6] explained the preferential filling of a bulla by examining the pressure–volume characteristics of isolated bullae. They found that bullae behave like paper bags, which are extremely compliant until they are full, when they become tense. The lung surrounding the bulla is less compliant, and the pressure required to inflate it exceeds the pressure necessary to inflate the bulla. This means that when bulla and lung are exposed to the same negative intrapleural pressure, the bulla will fill preferentially and always completely before the remainder of the lung is filled. Consequently, once a parenchymal weakness in the lung exceeds a certain size, it will result in a space within the lung that fills preferentially. The force of elastic recoil will produce retraction of the surrounding lung away from the hole or space and enlarge it. Even though the bulla remains in free communication with the airway, it will not participate in ventilation to any important extent because of its large volume, or in gas exchange because of its relatively small and avascular internal surface area [3].

The degree to which bullae contribute to dyspnea depends more on the amount of lung they replace and the extent of underlying destructive lung disease present than on the amount of lung they compress [7–9]. Consequently, the decision regarding surgical intervention is difficult, as it benefits only the latter. With this understanding of the pathophysiology of bullous disease, it is easier to understand that the expected clinical improvement after surgical obliteration is highly variable.

Patient Presentation and Selection of the Operative Candidate

The current rationale for surgical intervention for bullous disease is to allow normal but compressed lung to reexpand. The manner of presentation of patients with bullae varies enormously, ranging from incidental findings of small bullae in patients with normal lung function to the "vanishing lung syndrome" where all tissue appears to have been replaced by multiple bullae, associated with severe dyspnea, respiratory failure, and cor pulmonale.

Patients with normal lung function rarely require operation, and if in that situation the bullae enlarge and result in dyspnea, good postoperative improvement is the rule. Most patients referred for surgical consideration have bullae associated with chronic obstructive pulmonary disease and show little difference clinically or functionally from patients with chronic obstructive pulmonary disease without bullae. It is difficult to know to what degree a lung with an emphysematous process will improve. If the process of emphysema is paraseptal or periacinal (nonobstructive) instead of panlobular (obstructive), then substantial improvement can be expected. However, no method can diagnose the type of emphysema during life, so selection of patients suitable for operation must be based on clinical, physiological, and radiographic considerations.

Clinical Evaluation

The history and physical examination should be aimed at distinguishing between patients who have primarily emphysema (pink puffers) from those with chronic bronchitis (blue bloaters). Clinical experience [10] has shown that it is in this latter group with productive cough, frequent respiratory infections, carbon dioxide retention, bronchospasm, and heart failure that surgical outcome has been less successful. Continued smoking also adds to the morbidity [7].

Physiological Evaluation

Severe reduction in forced expiratory volume in 1 second and severe dyspnea in the presence of a single bulla or small bullae occupying less than one third of the lung suggest that symptoms are related to diffuse disease and that operative intervention will not ameliorate them [4, 9–11]. With large bullae, two physiological signs relate the cause of the dyspnea to the bullae: a reduction in forced vital capacity almost equal to the reduction in forced expiratory volume in 1 second, a finding suggesting a restrictive pattern, and a discrepancy between functional residual capacity measured by body plethysmography and gas dilution technique [10]. This discrepancy will often be between 2 to 4 L and represents the amount of trapped gas.

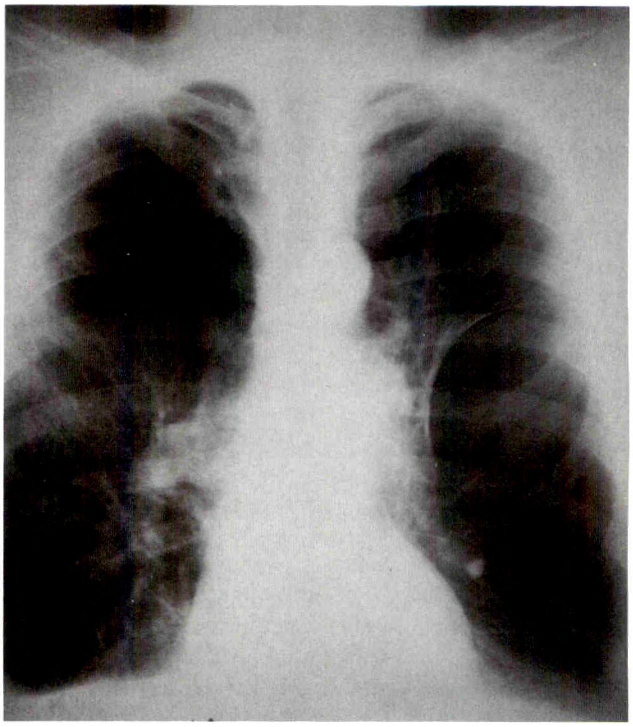

Fig 3. Chest roentgenogram demonstrates a large solitary bulla of the left chest.

Although attempts have been made to correlate operability and survival with pulmonary function variables, they have been unsuccessful. Long-term survival with improved functional status has been shown in patients with forced expiratory volume in 1 second of less than 1 L [4, 9, 11]. Lack of specificity has also been shown with single-breath diffusing capacity and bronchospirometry [10].

Radiographic Evaluation

Most observers [4, 9–11] have agreed that the volume occupied by the bulla is the most important variable in determining clinical and physiological outcome. Bullous lesions that occupy more than 50% of a hemithorax with normal underlying parenchyma have the best results (Fig 3), whereas lesions occupying less than one third of the lung may not show any substantial improvement. This is also valid for patients with underlying emphysema.

Determining the size and number of bullous lesions from a plain chest roentgenogram is not always reliable. Computed tomography is more sensitive and provides better localization of bullae and consequently is a greater aid to surgical planning [4, 10]. It is also an excellent noninvasive method of evaluating lung architecture and assessing the extent of underlying lung disease [12]. Ventilation/perfusion scanning, previously used to localized bullae, has been replaced by the computed tomographic scan.

Angiography can provide useful information about

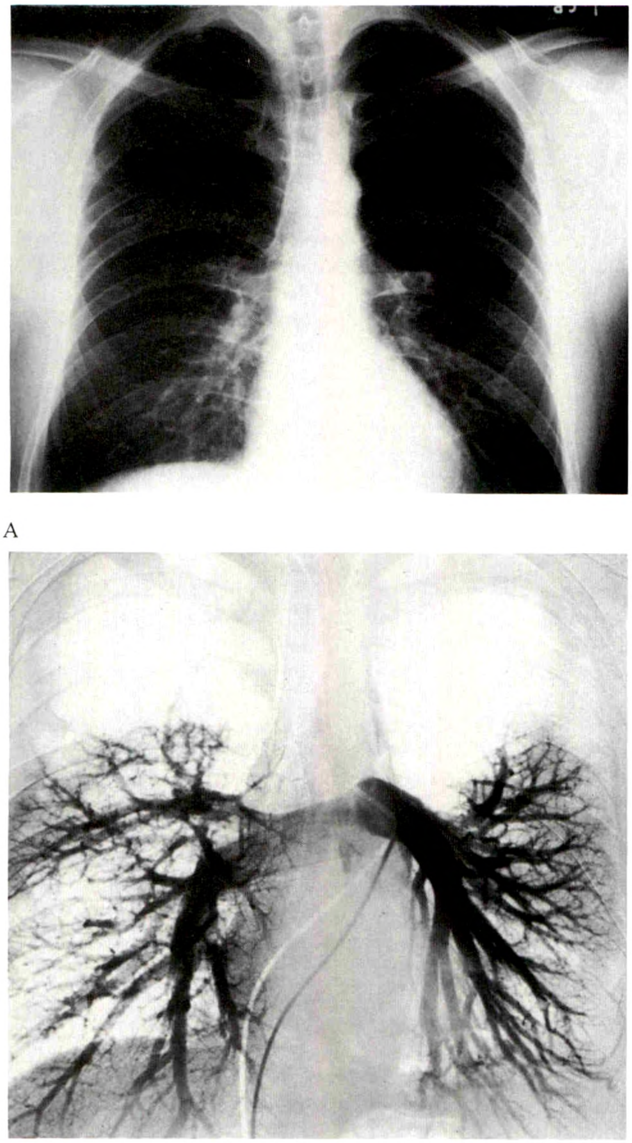

Fig 4. (A) Chest roentgenogram demonstrates large biapical bullae. (B) Pulmonary angiogram demonstrates intact but crowded vasculature.

unaffected areas of the lung but is not always necessary. If the vasculature surrounding the bulla is intact but crowded (Fig 4), there is a high probability of good expansion of functioning lung tissue after bullectomy, whereas lack of crowding with thinning and disruption of the vasculature suggests a poor result from resection.

Operative Procedure

A number of operative procedures have been described for the surgical therapy of bullous lung disease or acquired cysts. They include local excision, plication, wedge resection, lobectomy, bilobectomy, segmental resection, Monaldi's tube drainage, and marsupialization. Improved understanding of the pathophysiology of bullous lung disease has resulted in the discarding of most of these procedures. A disease characterized by loss of lung parenchyma cannot be optimally treated by removal of further lung tissue. Lobectomy results in far greater reductions in forced expiratory volume in 1 second in both the early and late postoperative periods than do procedures that conserve lung tissue [13]. Consequently, lobectomy is not only undesirable but rarely necessary. Indeed, observations have shown intact residual lung tissue in lobectomy specimens obtained because the impression at operation was that no functional lung tissue remained in the lobe [8].

The operative procedure should aim at conservation of lung tissue. Narrow-necked bullae are excised after ligation of their pedicled stalk, whereas smaller bullae can be obliterated by coagulation. Broad-based bullae are oversewn or plicated. Giant bullae with wide bases are removed by first opening the cavity and excising the transparent wall along the line where it merges with normal lung. Any trabeculae are divided, and a careful search is carried out for air leaks. The cavity is closed airtight by approximating the pleural edges of the lung with a running mattress suture. The correct placement of at least two chest tubes is very important to properly evacuate postoperative air leaks [8, 9, 14, 15].

If the patient has clinical respiratory failure and is receiving continuous oxygen therapy, intracavitary suction drainage as described by Monaldi [16] is the preferred procedure. The Monaldi tube suction procedure was initially performed in two stages: the first was a rib resection and iodoform gauze packing to adhere the visceral pleura to the parietal pleura, and the second was the insertion of a tube into the bulla. Recently it has been modified to a single-stage procedure. A small piece of rib is resected, and a pursestring suture of absorbable material is placed so that it encompasses both pleura and cyst wall. A large Foley catheter is inserted within the pursestring, and 5- to 10-mm Hg suction is applied for 36 to 48 hours, followed by underwater seal for 21 days [17]. Small bronchiolar leaks close and the bulla contracts during this period as a result of the inevitable secondary infection with resultant scar contracture of the bulla [8].

Simultaneous operation on both lungs through a median sternotomy has been recommended for patients with bilateral disease [18, 19]. Advocates of this approach emphasized that median sternotomy produces the least degree of morbidity and allows simultaneous correction of bilateral disease with a single operation.

Large bullae can be confused with a pneumothorax, particularly in patients complaining of sudden chest pain and dyspnea. The knee-jerk reaction of placement of a chest tube in such a patient often causes troublesome air leaks that may require thoracotomy for management.

Conclusion

The proper management of cystic and bullous lung disease requires careful evaluation. Because there is no effective medical management, these patients should be considered for surgical therapy.

Single, isolated bullae in asymptomatic patients should be observed. In individuals who are symptomatic, determination of whether the process is isolated and possibly expanding or is part of a diffuse process with major underlying lung disease should be determined. The size of the lesion is important. If it occupies greater than 50% of the hemithorax, further evaluation is necessary, for these patients may have the most dramatic benefits from operation. Pulmonary function tests are mandatory, but computed tomography is the most useful method of assessing the extent of underlying lung disease. If the underlying lung architecture is diffusely cystic, then any surgical option is of a palliative nature. Nevertheless, these patients should not be precluded from surgical consideration.

It is essential that fundamental surgical principles be observed. Potentially viable parenchyma must not be sacrificed. Limited resections that preserve all functioning lung tissue ensure maximal improvement. The high incidence of postoperative complications demands careful attention to tracheobronchial toilet, daily chest roentgenograms, and immediate treatment of mechanical complications.

The indications for resection of large bullae in the face of hypercapnia and diffuse emphysema require careful individual study because in these patients, a small increment in function may be of great clinical benefit.

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CORRESPONDENCE

Balloon Angioplasty of Aortic Coarctation

To the Editor:

I read with interest the recent letter by van Son and associates [1] on the optimal age for surgical repair of aortic coarctation. However, neither van Son and associates [1], nor Heikkinen and Ala-Kulju [2], on whose article van Son and associates commented in their letter, mentioned percutaneous balloon angioplasty as an alternative to surgical treatment of either native coarctation or aortic recoarctation. The results of the Valvuloplasty and Angioplasty of Congenital Anomalies Registry that were published recently [3, 4] showed that balloon angioplasty can be equally effective in both situations. It is safe and equally applicable to newborns and young infants as it is to older patients, where it has already gained considerable support [3].

Percutaneous balloon angioplasty offers the substantial advantage over operation of avoiding intubation, general anesthesia, and repeat thoracotomy. It also minimizes blood loss and shortens the duration of hospitalization considerably. The potential savings in patient discomfort and monetary expenditures are very substantial.

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4. Hellenbrand WE, Allen HD, Golinko RJ, Hagler DJ, Lutin W, Kan J. Balloon angioplasty for aortic recoarctation: results of Valvuloplasty and Angioplasty of Congenital Anomalies Registry. Am J Cardiol 1990;65:793–7.

Reply

To the Editor:

We appreciate the letter of Cheng in which he reports the advantages of percutaneous balloon angioplasty over operation in the management of both native and operated coarctation of the aorta [1, 2]. Although we recognize that the technique of percutaneous balloon angioplasty appears to be an attractive alternative to surgical repair of coarctation restenosis in neonates and children, we consider this technique investigational in native coarctation of the aorta until further follow-up on patients is available, especially with regard to the long-term development of the dilated area [3–5]. Because balloon angioplasty works by producing a controlled injury, in which the intima and part (or all) of the media are disrupted, there are chances of complete vascular disruption (although small) and of aneurysm formation at the dilation site [4]. In addition, in the long term there may be an increased risk of infective aortitis and aortic dissection at the site of dilation. Also, vascular complications at the introduction site of the catheter have limited the use of this technique, especially in neonates [6].

Balloon angioplasty may play a role in the palliation of symptomatic neonates with coarctation of the aorta. Several studies have shown a short-term beneficial effect of balloon dilation for the treatment of these critically ill patients who may successfully undergo surgical repair at an older age when surgical results are more favorable [1, 5]. However, we think that, if the condition of the neonate or child allows it, optimal treatment of coarctation of the aorta consists of resection of all ductal tissue, followed by construction of a wide end-to-end anastomosis of the aorta [7, 8]. Thus the coarctogenetic potential of ductal tissue is eliminated, whereas ductal tissue is left in situ in balloon angioplasty.

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References

1. Tynan M, Finley JP, Fontes V, Hess J, Kan J. Balloon angioplasty for the treatment of native coarctation: results of Valvuloplasty and Angioplasty of Congenital Anomalies Registry. Am J Cardiol 1990;65:790–2.
2. Hellenbrand WE, Allen HD, Golinko RJ, Hagler DJ, Lutin W, Kan J. Balloon angioplasty for aortic recoarctation: results of Valvuloplasty and Angioplasty of Congenital Anomalies Registry. Am J Cardiol 1990;65:793–7.
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Unusual Complication of J Wire Used for Central Venous Cannulation

To the Editor:

Instrumentation of patients undergoing heart operations is now routine, and includes insertion of peripheral venous and arterial lines and a central venous or pulmonary artery (Swan-Ganz)

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Ann Thorac Surg 1991;52:581–5 • 0003-4975/91/$3.50

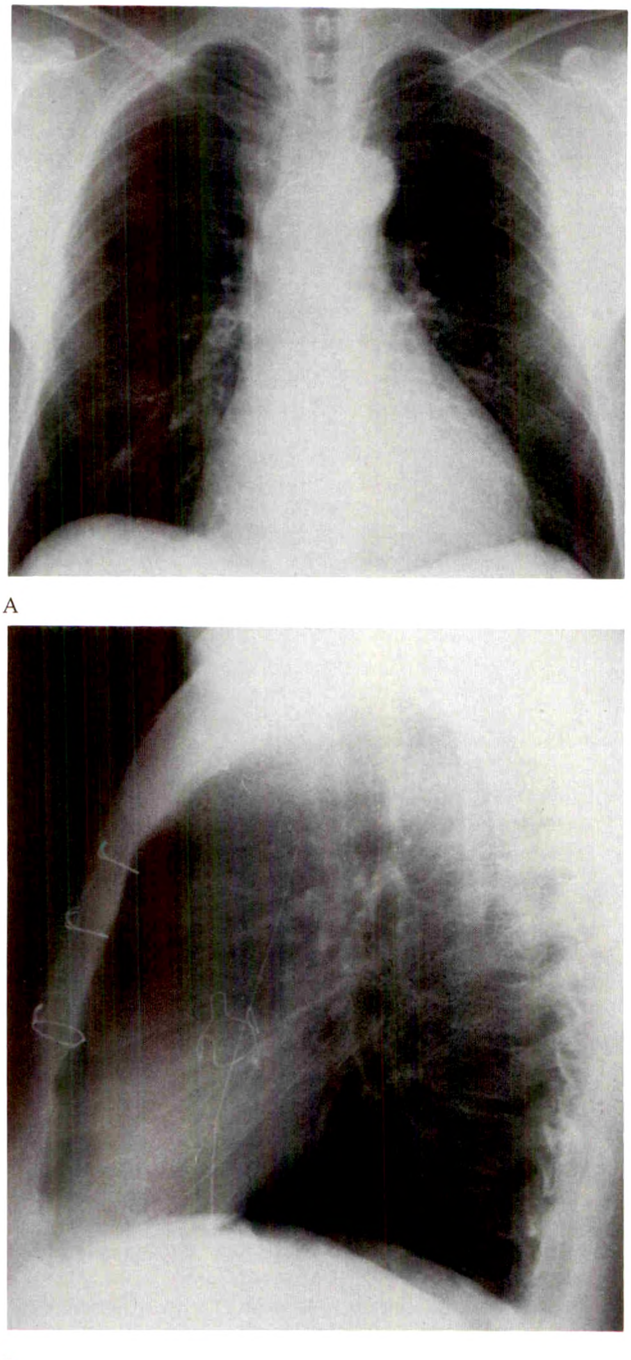

Fig 1. Chest roentgenograms showing retained J wire.

catheter. We recently had a patient with a retained J wire in the superior vena cava. The patient was a 55-year-old man who underwent elective aortic valve replacement for previous endocarditis and aortic insufficiency. At the time of operation he was instrumented with peripheral venous and arterial lines and a Swan-Ganz catheter. His operation and postoperative course were uneventful, and he was discharged from hospital on the tenth postoperative day.

He returned to the emergency room 6 weeks later with a febrile illness feeling unwell. Blood cultures done in another hospital revealed coagulase-negative staphylococci. On admission he was febrile (oral temperature, 39.5°C). The valve sounds were normal and the chest was clear to auscultation. The remainder of the physical examination was normal. A chest roentgenogram, to the surprise of everyone, revealed the presence of a foreign body in the superior vena cava extending through the heart and into the inferior vena cava, compatible with the J wire used for insertion of central lines (Fig 1). On reviewing the chart, there was no mention of any problems with the insertion of the central line.

Postoperative chest roentgenogram reports did not document the presence of the foreign body, although it was present in retrospect. The patient was immediately transferred to the radiology department where, under local anesthesia and using a snare, the J wire was removed through the percutaneous route. He made an uneventful recovery after 6 weeks of appropriate intravenous antibiotic therapy.

The literature on retained foreign bodies in the venous system and heart is large and consists of a variety of materials including needles, pacemaker leads, Kirschner wires, bullets, and others [1–5]. We recently became aware of another patient who had a J wire retained for 6 years after elective coronary artery bypass grafting without untoward physical effects. However, in our particular patient, bacteremia became a problem due to the presence of an aortic valve prosthesis. Removal of the wire allowed for rapid control of the infection.

On reviewing the medical records, the operating room count was correct. However, until recently, it was not routine to count the contents of the disposable set that is used to insert the Swan-Ganz catheter. The roentgenogram reports did not reveal the presence of the foreign body, although it was present in retrospect. Therefore, it is recommended that the contents of the disposable set used for Swan-Ganz catheter insertion be included in the count in the operating room. Further, it is imperative that the postoperative roentgenograms be reviewed by the surgical staff. Removal of the retained wire, as in our case, is possible through the percutaneous route without serious complications.

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Hydrostatic Dilation of Arterial Conduits

To the Editor:

In their interesting article, Mills and Everson [1] describe in detail the technique of harvesting of the inferior epigastric artery as an

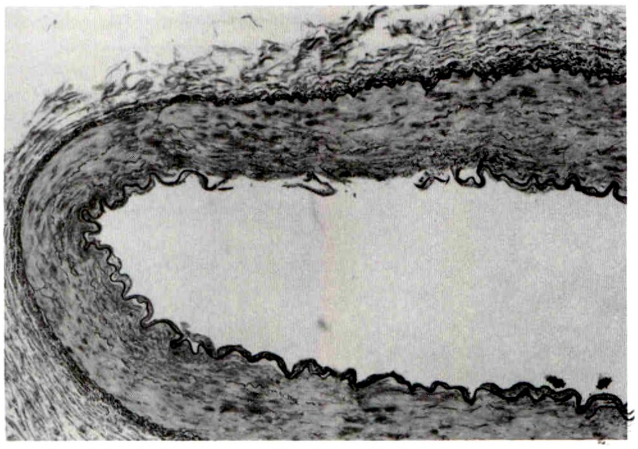

Fig 1. Example of an elastomuscular internal mammary artery (section obtained at the level of the epigastric bifurcation) after hydrostatic dilation with diluted papaverine solution. Note the stretching of the normally meander-shaped (at least in the in vitro situation) intima and internal elastic lamina. In the superior aspect the intima and internal elastic lamina have been disrupted. (Elastin-trichrome after Masson stain; ×80 before 31% reduction.)

alternative conduit in myocardial revascularization [2]. Their technique is a modification of the technique that our group has previously published [3] and that was first reported in 1988 [4]. I have some concern, however, about one aspect of the harvesting technique of the inferior epigastric artery as described by Mills and Everson, and that is hydrostatic dilation of the conduit by diluted (1:40) papaverine solution. In a recent study [5], my colleagues and I have demonstrated the potential detrimental effect of hydrostatic dilation on the intima and internal elastic lamina of the internal mammary artery (IMA) and the musculophrenic and superior epigastric arteries. In this study, which strictly adhered to the technique of IMA dilation as described by Mills and Bringaze [6], overdistention of distal IMA specimens (all obtained at operation and all extending beyond the epigastric bifurcation) was avoided, at least subjectively. Histological examination showed no significant difference in the number of fenestrations in the internal elastic lamina in the elastic and elastomuscular segments of the IMA and musculophrenic and superior epigastric arteries between dilated and nondilated groups of patients, whereas in the muscular segments of the musculophrenic and superior epigastric arteries the number of fenestrations was significantly greater in dilated specimens than in nondilated specimens. In addition, the percentage of disruptions of the intima and internal elastic lamina in the examined sections of the elastic, elastomuscular (Figs 1, 2), and muscular segments of the IMA and the musculophrenic and superior epigastric arteries was 20, 17, and 35, respectively. In analogy to the almost purely muscular musculophrenic and superior epigastric arteries (van Son JAM, Smedts F, de Wilde PCM, et al; unpublished results), I fear that hydrostatic dilation of the inferior epigastric artery may also have potential detrimental effects on the intima and the internal elastic lamina that may lead to impaired endothelium-dependent vasodilation and enhanced intimal thickening and, ultimately, accelerated atherosclerosis of this conduit [7].

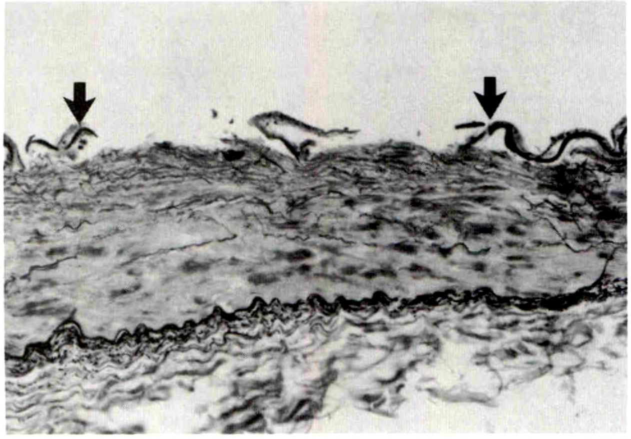

Fig 2. Detail of disruption of intima and internal elastic lamina of elastomuscular internal mammary artery after hydrostatic dilation with diluted papaverine solution. The arrows *indicate both ends of the disrupted intima and internal elastic lamina. (Elastin-trichrome after Masson stain; ×400 before 31% reduction.)*

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Aortic Vent Efficiency

To the Editor:

Van der Linden and Casimir-Ahn's elegant study [1] using transcranial Doppler to document cerebral air emboli in 100% of 10 patients undergoing open heart valve operations despite "standard deairing procedures" is remarkable and important. However I write to call attention to procedures number 4 and 6 in their deairing technique, namely a freely bleeding large-bore needle inserted in the aortic root. The authors were apparently unaware of a report I published in February 1971 [2] with Drs Eugene Wallsh and Frank C. Spencer of New York University in which we computed the air venting efficiency of various aortic vents in an vitro model. Among the worst vents were freely bleeding needles and tacks (efficiency, 20% to 36%). When connected to suction the efficiency of a 17-gauge needle vent increased from 21% to 91%. The best vent we tested was a freely bleeding stab wound with a nonconstricted, multifenestrated 10F plastic catheter connected to suction, which had a 97% air venting efficiency. I write to endorse the authors' appeal for greater attention to air venting protocols in contemporary open heart operations and to call attention to our research, now 20 years old, which still can contribute to enhanced patient safety in the 1990s.

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1. Van der Linden J, Casimir-Ahn H. When do cerebral emboli appear during open heart operations? A transcranial Doppler study. Ann Thorac Surg 1991;51:237–41.
2. Brenner WI, Wallsh E, Spencer FC. Aortic vent efficiency. A quantitative evaluation. J Thorac Cardiovasc Surg 1971;61: 258–64.

Reply

To the Editor:

We appreciate Dr Brenner's interest in our report [1], and we agree with his appeal for greater attention to air venting protocols. The elegant experimental study of Dr Brenner and his colleagues [2] does clearly emphasize the efficacy of continuous suction on the venting aortic needle (preferably fenestrated). Indeed, at the time of the study most of our surgeons, but not all, were using suction on the aortic vent. However, the simple on-line ability of the transcranial Doppler to detect when cerebral embolization occurs during cardiac operations has made all our surgeons more aware of this problem. Furthermore, it has stimulated our surgeons' interest in trying to optimize their techniques, including deairing, to minimize the incidence of cerebral emboli during cardiac operations.

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References

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2. Brenner WI, Wallsh E, Spencer FC. Aortic vent efficiency. A quantitative evaluation. J Thorac Cardiovasc Surg 1971;61: 258–64.

Role of Thoracoplasty in the Treatment of Chronic Empyema

To the Editor:

The treatment of the infected pleural space has always been a source of controversy. Accordingly, a report on the role and technique of thoracoplasty in its management [1], a discussion of this subject [2], and a list of key references [3] are always timely.

The cautionary editorial comments by Pairolero and Trastek [2] concerning the importance of limiting the use of thoracoplasty cannot be overemphasized. They stated that when thoracoplasty is employed to obliterate an infected pleural space, the procedure "should be limited, confining resection to only a portion of a few ribs, and then combined with other space reduction techniques, primarily intrathoracic transposition of extrathoracic skeletal muscle."

I believe one other point should be emphasized. When the classic ten-rib Alexander thoracoplasty [4] was performed in the treatment of pulmonary tuberculosis, removal of the first rib was necessary to obtain adequate collapse therapy. However, in performing a limited thoracoplasty to assist in the elimination of an infected pleural space, or a limited tailored thoracoplasty in association with a pulmonary resection when a space problem is anticipated, the first rib *should not be removed* as noted by Dr Kamal A. Mansour in his discussion of the article by Horrigan and Snow [1]. This point was repeatedly emphasized by my mentor Dr Robert H. Wylie, Emeritus Professor of Surgery, Columbia-Presbyterian Medical Center, New York, NY.

Removal of the first rib is not necessary in the management of the infected pleural space, almost invariably produces a deforming scoliosis with a loss of pulmonary function, and therefore should not be performed.

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References

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Reply

To the Editor:

We agree with Dr Jaretzki that resection of the first rib is not advisable when a limited thoracoplasty is performed to control an infected apical pleural space. Although often successful, the accompanying deterioration in pulmonary function and cosmetic deformity from subsequent scoliosis may at times be serious and increases with the number of additional ribs resected.

If an apical space still exists after limited thoracoplasty without first rib resection, it can be managed by other space reduction techniques, such as intrathoracic muscle transposition of extrathoracic skeletal muscles. Both the serratus anterior and pectoralis major muscles have an arc and axis of rotation that easily reach the apex of the chest, and rarely is either injured during prior thoracotomy.

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Reply
To the Editor:

I would like to compliment Dr Jaretzki for his elaborate discussion of my discussion. I appreciate his expanding on the two main points regarding thoracoplasty for the infected pleural space, ie, limited extent of rib resection and unwarranted resection of the first rib, thanks to the use of muscle flaps. It is also good to include a list of key references.

However, I have two further points to bring up. First, I am glad to know that northern and southern surgeons are in agreement on this subject, and second, from a historical viewpoint, the work on myocutaneous flaps was developed in the South. Although muscle flaps were randomly used in the past, it was in 1972 and under the guidance of Dr Maurice Jurkiewicz, Chief of Plastic and Reconstructive Surgery, Emory University School of Medicine, that serious animal and cadaver studies of myocutaneous flaps were undertaken. For several years, Drs McCraw, Mathes, Arnold (then residents), and Vasconez (faculty) described the anatomy, the blood supply, and clinical applications of myocutaneous flaps. It is this work that resulted in the publication of *Clinical Atlas of Muscle and Musculocutaneous Flaps* by Mathes and Nahai in 1979 (C.V. Mosby Co, St. Louis, MO), *Clinical Applications for Muscle and Musculocutaneous Flaps* by Mathes and Nahai in 1982 (C.V. Mosby Co), and *McCraw and Arnold's Atlas of Muscle and Musculocutaneous Flaps* in 1990 (Raven Press, New York, NY). I believe these references would be of help to those who are interested.

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Diuresis in Hemodynamically Compromised Patients
To the Editor:

We read with interest the article by Magovern and Magovern entitled "Diuresis in hemodynamically compromised patients: continuous furosemide infusion" [1]. They describe use of this technique in "stable but hemodynamically compromised" patients with good renal function. We fail to understand the advantages of a continuous furosemide infusion over the employment of small bolus (5 or 10 mg) doses of furosemide as and when necessary. We would like to introduce to you our approach to the problem of maintaining diuresis in such patients.

Of late we have successfully employed a combination of dopamine and furosemide in 6 oliguric patients in the postoperative period. The dosage of furosemide that we employ is 10 $mg \cdot kg^{-1} \cdot day^{-1}$, of which half is given as a bolus and the remaining is combined with a dopamine infusion at a dose of 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

We have found this combination of furosemide and dopamine to be extremely effective in maintaining good diuresis without adverse hemodynamic effects. As pointed out by Magovern and Magovern, this infusion too can be regulated and tapered off. After the patient has maintained a steady urine output of more than 2 $mL \cdot kg^{-1} \cdot h^{-1}$ for more than 24 hours we observe the response to oral diuretics and wean the patient off the dopamine-furosemide infusion.

As in all cases, this obviously requires close monitoring of the hemodynamic variables and electrolyte levels. The advantage of the additional low-dose dopamine infusion in combination is to provide uniform central and peripheral venous dilatation and thus allow optimum tissue perfusion in these patients while providing a substantial urine output.

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Reference

1. Magovern JA, Magovern JG Jr. Diuresis in hemodynamically compromised patients: continuous furosemide infusion. Ann Thorac Surg 1990;50:482–4.

Reply
To the Editor:

Honnekeri and Tendolkar have asked about the advantage of continuous rather than bolus administration of furosemide. This question was addressed in our paper. The continuous infusion provides a steady and continuous urine output without changing cardiac preload. Intermittent boluses of furosemide result in periods of vigorous diuresis alternating with periods of relative oliguria.

The technique of combining a continuous infusion of furosemide with dopamine seems like a sound concept. The authors give 10 $mg \cdot kg^{-1} \cdot day^{-1}$ of furosemide. In a 70-kg patient, this would be 700 mg of furosemide per day—350 mg as a bolus and 350 mg over a 24-hour period. This is a much larger dose than we have described in our report, in which the average furosemide dose was less than 150 mg/day.

Certainly, patients can be diuresed with intermittent furosemide. This technique has been used for many years with excellent results. Continuous infusion of furosemide is also a simple and reliable technique that has certain advantages in select patients.

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THE SOUTHERN THORACIC SURGICAL ASSOCIATION

Thirty-eighth Annual Meeting
November 7–9, 1991
The Hyatt Regency Grand Cypress, Orlando, Florida

The Thirty-eighth Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Grand Cypress Resort on November 7–9, 1991, with the Postgraduate Program on Thursday, November 7, from 7:45 AM to noon. The meeting is open to all duly qualified physicians. Both members and nonmembers are urged to register in advance.

Registration and Fees

Advance registration forms, hotel reservation cards, and details regarding transportation arrangements will be mailed to Association members. Nonmembers should write to the Secretary-Treasurer, Hendrick B. Barner, MD, Southern Thoracic Surgical Association, 401 North Michigan Ave, Chicago, IL 60611-4267.

Members may register for the Scientific Program for $50. Residents and fellows may register without payment of a registration fee upon presentation of a letter from their chief of service. The fee for other physicians is $300, except guest speakers, authors, and co-authors. Nurses and paramedical personnel may register upon payment of a $300 fee and presentation of a letter of introduction from a member.

Registration for the Postgraduate Program is $50 for all physicians, residents, and fellows.

Presentation and Publication

Papers presented at the Scientific Session must be submitted in triplicate to the Secretary-Treasurer, Hendrick B. Barner, MD, at the time of presentation, or to the Editor of *The Annals of Thoracic Surgery* prior to presentation.

Speakers for the Regular Program are limited to 10 minutes; those in the Forum are limited to 5 minutes. Speakers are asked to use the preview room prior to the opening of their session to prepare their slides for presentation.

Accreditation

The Southern Thoracic Surgical Association is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. One credit hour in category I of the Physician's Recognition Award of the American Medical Association may be claimed for each hour of participation by the individual physician. A maximum of 16 hours is available for credit at this meeting.

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PROGRAM
THIRTY-EIGHTH ANNUAL MEETING
SOUTHERN THORACIC SURGICAL ASSOCIATION

November 7, 1991 (Thursday)
Postgraduate Program

7:45 Introduction to the 1991 Postgraduate Course
Course Director: William A. Baumgartner, Baltimore, MD

Acquired Cardiac Disease

7:50 Reoperations for Coronary Artery Disease: Techniques and Conduits
Noel L. Mills, New Orleans, LA

8:20 Advances in Intraoperative Echocardiography
Duke E. Cameron, Baltimore, MD

8:50 Current Applications, Techniques, and Complications of the AICD
John W. Hammon, Jr, Nashville, TN

Congenital Heart Disease

9:20 Congenital Heart Disease in the Adult
Harvey W. Bender, Jr, Nashville, TN

9:50 Break

Thoracic Disease

10:20 Newer Radiographic Techniques in the Evaluation of Thoracic Disease
Kerry M. Link, Winston-Salem, NC

11:00 Modern Day Management of Empyema Thoracis
William J. Anderson, Nashville, TN

11:30 Treatment Options for Complications of Emphysema
Frederick L. Grover, San Antonio, TX

12:00 Adjourn

November 7, 1991 (Thursday)
First Scientific Session

1:30

1. Operative Treatment of Obstructive Hypertrophic Cardiomyopathy in the Pediatric Population: A 26-Year Experience
   Richard E. Clark, Christopher D. Stone, Charles L. McIntosh, and Hani A. Hennein, Bethesda, MD
2. Risk Factors Predictive of Operative Mortality Vary Among Subsets of CABG Patients
   Frederick L. Grover, Randall Johnson, and Karl E. Hammermeister, VA Cardiac Surgeons, San Antonio, TX
3. Congenital Vascular Rings: A 22-Year Experience
   J. Alex Haller, Jr, Karen Chun, Paul M. Colombani, L. R. Scherer, and David L. Dudgeon, Baltimore, MD
4. Heparin Antibody in Open Heart Surgical Patients: Sequelae of Late Recognition
   Joseph T. Walls, Jack J. Curtis, Donald Silver, Theresa M. Boley, Richard A. Schmaltz, and Weerachai Nawarawong, Columbia, MO

2:30 Guest Lecturer
Role of Ventricular Mechanics in the Selection of Patients for Valvular Procedures
Blase Carabello, Charleston, SC

3:00–3:45 Break

3:45 5. Esophageal Ultrasound Assessment of Preoperative Therapy of Esophageal Carcinoma
Thomas W. Rice, Gregory A. Boyce, Michael V. Sivak, David J. Adelstein, and Thomas J. Kirby, Cleveland, OH

6. Transhiatal Esophagectomy: A Safe Alternative
Thomas M. Daniel, Kirk J. Fleischer, Terry L. Flanagan, Curtis G. Tribble, and Irving L. Kron, Charlottesville, VA

7. Cricopharyngeal Dysfunction Associated with Incompetent Lower Esophageal Sphincter
Kamal A. Mansour and Hakob G. Davtyan, Atlanta, GA

8. Video-Thoracoscopy: Improved Technique and Expanded Indications
William H. Coltharp, William C. Alford, Jr, George R. Burrus, David M. Glassford, Jr, John W. Lea IV, Michael R. Petracek, William S. Stoney, Clarence S. Thomas, Jr, John H. Arnold, and Robert N. Sadler, Nashville, TN

9. Thallium Imaging in Preoperative Evaluation of the Pulmonary Resection Candidate
Joseph I. Miller, Atlanta, GA

10. Randomized Trial of Neoadjuvant Therapy for Lung Cancer: Interim Analysis
Harvey I. Pass, Helen Pogrebniak, Seth Steinberg, James Mulshine, and John Minna, Bethesda, MD

11. Colorectal Lung Metastases: Results of Surgical Excision
Mark S. Allen, Molly K. McAfee, Victor F. Trastek, Duane M. Ilstrup, and Peter C. Pairolero, Rochester, MN

November 8, 1991 (Friday)
Second Scientific Session

8:00 12. Isolated Lung Transplantation for End-Stage Lung Disease: A Viable Therapy
Thomas M. Egan, Jan H. Westerman, C. Jake Lambert, Jr, Frank Detterbeck, Jeanette T. Thompson, Michael R. Mill, Blair A. Keagy, and Benson R. Wilcox, Chapel Hill, NC

13. Pulmonary Embolism in the Cardiac Surgical Patient
A. Marc Gillinov, Elizabeth A. Davis, Anthony J. Alberg, Mary Rykiel, Timothy J. Gardner, and Duke E. Cameron, Baltimore, MD

14. Emergent Applications of Cardiopulmonary Support: A Multiinstitutional Experience
Jonathan G. Hill, Pam Bruhn, Sheldon Cohen, Michael Gallagher, Frank Manart, Charles Moore, Paul Seifert, Paul Askari, and Candace Banchieri, Portland, OR

15. Improvement in Multiorgan Dysfunction With Prolonged Univentricular Support
Michael S. Sweeney, Clay M. Burnett, O. H. Frazier, J. Michael Duncan, J. David Vega, and Branislav Radovancevic, Houston, TX

9:15–10:00 Break

10:00 16. Early Repair of VSD in the Small Infant with Congestive Heart Failure
Thomas L. Spray, Alan D. Muskett, and Charles E. Canter, St. Louis, MO

17. The "Learning Curve" for Surgeons for Evaluating Repair of Congenital Heart Defects Using Intraoperative Echocardiography
Ross Ungerleider, William Greeley, Ronald Kanter, and Joseph Kisslo, Durham, NC

18. Atrioventricular Pacing in Congenital Heart Disease
John M. Kratz, Paul C. Gillette, Robert M. Sade, and Fred A. Crawford, Charleston, SC

10:45–11:30 Presidential Address
Robert M. Sade, Charleston, SC

11:30–12:15 Business Meeting

November 9, 1991 (Saturday)
Third Scientific Session, Surgical Forum (Two Simultaneous Sessions)
Cardiovascular Session

8:00–9:30 19. Noninvasive Detection of Acute Rejection in a New Experimental Model of Heterotopic Heart Transplantation
James R. Stewart, Steven J. Hoff, William H. Frist, James Atkinson, Marvin Kronenberg, and Martin Sandler, Nashville, TN

20. Preservation of Chordal Mechanism with Mitral Valve Replacement Does Not Enhance Left Ventricular Function
Panagiotis Iakovidis, David J. Cohen, Gregory Freeman, Irma Montemayor, Michael Clem, Miguel Zabalgoitia, Robert O'Rourke, and Frederick L. Grover, San Antonio, TX

21. The Influence of Age and Gender on Human Internal Mammary Artery Size and Reactivity: Relevance to Coronary Artery Bypass Grafting
Andrew S. Wechsler, Rebecca J. Dignan, Thomas Yeh, Jr, Cornelius M. Dyke, and Harry A. Lutz III, Richmond, VA

22. A Staged Expanding Pulmonary Artery Band
E. Taliaferro Warren, Bobby J. Heath, and Woodrow W. Brand III, Jackson, MS

23. Early Diagnosis of Cardiac Tamponade Using Right Ventricular Indices
James W. Jones, N. N. Izzat, M. N. Rashad, T. R. McLean, J. I. Thornby, and A. C. Beall, Jr, Houston, TX

24. Implantation of Cardioverter Defibrillators in the Poststernotomy Patient
Ralph J. Damiano, Andrew H. Foster, Kenneth A. Ellenbogen, William J. Welch, and Andrew S. Wechsler, Richmond, VA

25. Improved Right Ventricular Performance With Retrograde Cardioplegia
John M. Kratz, E. Charles Douville, Francis G. Spinale, Fred A. Crawford, Calvert C. Alpert, and Andrew Pearce, Charleston, SC

26. Spinal Cord Oxygenation, Intraoperative Blood Supply Localization, Cooling, and Function with Aortic Clamping
Lars G. Svensson, Vasishta Patel, and E. Stanley Crawford, Houston, TX

Simultaneous Pulmonary Forum Section

8:00–9:30 27. Adjuvant Treatment Using Transfer Factor for Bronchogenic Carcinoma: 5- to 10-Year Follow-up
Marvin M. Kirsh, Richard I. Whyte, Herbert Sloan, and Mark B. Orringer, Ann Arbor, MI

28. Radiolabeled Monoclonal Antibody Imaging and Lung Cancer: Initial Clinical Results
James R. Stewart, John A. Carey, Walter H. Merrill, David H. Johnson, and Martin P. Sandler, Nashville, TN

29. Effect of Pulmonary Resection on Right Ventricular Function
Carolyn E. Reed and Francis G. Spinale, Charleston, SC

30. Bronchial Carcinoid Tumors
David H. Harpole, Scott Buchanan, W. Glenn Young, and Walter G. Wolfe, Durham, NC

31. Long-Term Survival Following Resection of Pulmonary Metastases From Carcinoma of the Breast
Joe B. Putnam, Jr, Louis A. Lanza, Giri Natarajan, and Jack A. Roth, Houston, TX

32. Renal Cell Carcinoma: Resection of Solitary and Multiple Metastases
Harvey I. Pass, Helen Pogrebniak, Gabe Haas, Marston Linehan, and Steven A. Rosenberg, Bethesda, MD

33. Comparison of Modified Euro-Collins and UW Solutions for Pulmonary Preservation Using Rat Orthotopic Single Lung Isotransplant Model
Bartley P. Griffith, Ryo Aeba, Robert J. Keenan, and Samuel A. Yousem, Pittsburgh, PA

34. Long-Term Function of Reduced-Size Porcine Lobar Pulmonary Transplants: Studies of Vascular Compliance
John A. Kern, Barry B. K. Chan, Terry L. Flanagan, Curtis G. Tribble, and Irving L. Kron, Charlottesville, VA

9:30–10:15 Break

Third Scientific Session, Continued

10:15 35. Primary Repair of Aortic Transections
Lawrence R. McBride, Keith S. Naunheim, Hendrick B. Barner, Vallee L. Willman, George C. Kaiser, and Andrew C. Fiore, St. Louis, MO

36. Predictors of Surgical Salvage After Failed Angioplasty
A. Michael Borkon, Trent L. Failing, Marcia L. Hoskins, Jeffrey M. Piehler, Duncan A. Killen, and William A. Reed, Kansas City, MO

37. Coronary Sinus Cardioplegia: A Clinical Trial Without "Antegrade-Retrograde" Approach
Kit V. Arom and Robert W. Emery, Minneapolis, MN

38. Time to First Pulse After AICD Implantation
Jack J. Curtis, Joseph T. Walls, Theresa M. Boley, Hugh E. Stephenson, Richard A. Schmaltz, Weerachai Nawarawong, and Greg C. Flaker, Columbia, MO

39. Hypothermic Circulatory Arrest as a Surgical Adjunct: A 5-Year Experience With 60 Adult Patients
Bruce A. Reitz, Duke E. Cameron, Elizabeth Davis, and A. Marc Gillinov, Baltimore, MD

40. Ultrasound Decalcification of Aortic Valve Stenosis With Intraoperative Color Doppler Echocardiography
Oscar R. Baeza, Naweed K. Majid, Daniel P. Conroy, and James S. Donahoo, Newark, NJ

41. MRI Evaluation of Recipient for Cryopreserved Aortic Allograft
Neal D. Kon, Kerry M. Link, William P. Buchanan, Abdel-Mohsen Nomeir, Thomas R. Downes, and A. Robert Cordell, Winston-Salem, NC

42. Thoracic Deceleration Injuries Exclusive of Aortic Disruption
Joe B. Putnam, Jr, R. B. Lee, P. Miller-Crotchett, William E. Walker, O. H. Frazier, and Michael S. Sweeney, Houston, TX

43. Mitral Valve Replacement: Randomized Trial of St. Jude and Medtronic-Hall Prostheses
Andrew C. Fiore, Keith S. Naunheim, Stephen D'Orazio, George C. Kaiser, Lawerence R. McBride, D. Glenn Pennington, Pamela S. Peigh, Vallee L. Willman, and Hendrick B. Barner, St. Louis, MO

12:30 Adjourn

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Carpentier-Edwards* Model 2625 and Model 6625 Bioprostheses
For information, call or write Baxter Healthcare Corporation, Edwards CVS Division, 17221 Redhill Avenue, Irvine, CA 92714-5686, (800) 424-3278. Refer to following for a brief summary of prescribing information.

For single use only

Design

As compared to other types of prostheses, preserved porcine bioprostheses exhibit certain potential advantages: the need for long-term anticoagulation is minimized; and although the durability of porcine bioprostheses is a limiting factor in long-term evaluation, tissue degradation is usually progressive and symptomatic, thereby permitting detection with proper annual follow-up in person with a physician, and elective intervention when necessary.

Models 2625 (aortic) and 6625 (mitral) Carpentier-Edwards* Bioprostheses are comprised of porcine aortic valves that have been preserved in buffered glutaraldehyde and then mounted on flexible frames. Glutaraldehyde is shown to both reduce the antigenicity of porcine xenograft valves and increase tissue stability. It has not been shown to affect or reduce the calcification rate of the valve.

The frame is designed to be flexible at the orifice as well as at the commissures. The compliance of the stent's commissure supports is intended to reduce the loading shock at the valve commissures and free margin of the leaflets. The flexibility of the orifice is intended to reduce the loading shock at the base of the leaflet. The flexible orifice concept is based on the physiology and mechanics of natural heart valves and reported experience with implantation of unstented homografts.

The lightweight frame is made of Elgiloy,† a corrosion-resistant alloy chosen because of its superior spring efficiency and fatigue-resistant characteristics.

The metal frame is covered with a porous, knitted polytetrafluoroethylene cloth to facilitate tissue ingrowth and encapsulation. The inflow orifice is contoured to support the right coronary cusp of the porcine valve. The commissure supports are not equidistant to conform to the anatomical configuration of the porcine aortic valve. The suture ring has a soft, silicone-rubber insert that is covered with a porous, seamless polytetrafluoroethylene cloth. The aortic sewing ring has been scalloped to conform to the natural aortic root. The compliant nature of the suture ring facilitates coaptation between the valve and an often irregular or calcific tissue bed.

The porcine aortic valves are fixed in buffered glutaraldehyde, and then mounted on cloth-covered frames. After mounting, the valves are tested *in vitro* to ascertain their competency and flow characteristics. After final inspection, the valves are sterilized, rinsed, and then transferred into fresh glutaraldehyde and packaged under aseptic conditions.

Indications

Bioprostheses are indicated for use in patients suffering from valvular heart disease. Aortic and mitral valvular heart disease are conditions involving any of the following: obstructions of the heart valves, or stenosis; leakage of the valves, known as regurgitation or insufficiency; and combinations of the two, sometimes referred to as mixed disease or combined lesions.

Valvular heart disease may be caused by any number of factors, including congenital abnormalities, infection by various microorganisms, degenerative calcification, rheumatic heart disease and rupture or dysfunction of the mitral subvalvular mechanism secondary to myocardial ischemia.

Bioprostheses are used particularly in those patients for whom long-term anticoagulation is contraindicated or who may be difficult to maintain on anticoagulation therapy.

The Carpentier-Edwards Bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of th [illegible] al valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis.

Warnings

For Single Patient Use Only

In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, and in patients who are on maintenance hemodialysis.

The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient and consideration of alternative methods of treatment. Overall durability, especially long-term, has not been established for bioprostheses.

Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery.

Note: Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis (see Clinical Experience).

Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed.

Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection.

Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombus. The appropriate anticoagulation therapy must be determined by the physician on an individual basis.

Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc., should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result.

Carpentier-Edwards Bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.

Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards Bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided.

Both the valve and glutaraldehyde storage solution are sterile. The outside of the jar is not sterile and must not be placed in the sterile field. Furthermore, the valve and container should never be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation.

Storage between 10°C and 25°C (50-77°F) is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue.

Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with the eyes, seek medical attention.

Surgical Precautions

1. When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result (Ref. 50). The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient.
2. Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts.
3. Left ventricular perforation or ulceration has been reported (Ref. 16). Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage.
4. The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15 mm, produced a pursestring effect, causing compression of the valve orifice.
5. When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures.
6. Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue.
7. As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs, which would interfere with proper valvular function.
8. The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence.
   Note: The contrasting suture material should not be removed from the prosthesis.
9. In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia.
10. A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar and implantation data card. If any differences are noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal.
11. Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation.

Complications

As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days), necessitating reoperation and replacement of the prosthetic device.

Complications associated with the use of Carpentier-Edwards Bioprostheses compiled from the literature and from reports received through the complaint handling system in accordance with the United States (Federal) regulations establishing Good Manufacturing Practices, 21 CFR Section 820.198, include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, degeneration, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct.

Note: Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies (Ref. 10) show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes a potential relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the post-operative period, and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards Bioprostheses to calcification as compared to other commercially available porcine valves.

Clinical Experience

Clinical data, available on 288 patients requiring isolated aortic valve replacement (AVR) and on 241 patients requiring isolated mitral valve replacement (MVR) with Models 2625 and 6625 Carpentier-Edwards Bioprostheses, respectively (mean follow-up of 2.6 and 2.2 years, respectively), indicate operative (< 30 days) and postoperative (> 30 days) survival rates of 97.2% and 96.0% per patient year, respectively, for the isolated AVR population, and 90.9% and 93.2% per patient year, respectively, for the isolated MVR population.

In the isolated AVR population there were a total of 208 (72.2%) males and 80 (27.8%) females with a mean age (± standard deviation) of 66.2 (± 10.1) years and a range of 24-88 years. The indications for valve replacement were stenosis (62.4%), regurgitation (13.5%), mixed disease (18.4%), and previous prosthetic mitral valve dysfunction (2.1%). For 13.5% of the patients, the diagnosis for replacement is unknown.

In the isolated MVR population there were a total of 79 (32.8%) males and 162 (67.2%) females with a mean age of 63.3 (± 10.5) years and a range of 30-84 years. The indications for valve replacement were stenosis (27.0%), regurgitation (36.1%), mixed disease (19.9%), and previous prosthetic aortic valve dysfunction (7.5%). For 9.5% of the patients, the diagnosis for replacement is unknown.

The follow-up methods used at each clinic included hospital visits, office visits, and contact by telephone or letter with either the patient himself, his family, or his local doctor.

These data were compiled from a multicenter clinical trial conducted by Edwards CVS Division.

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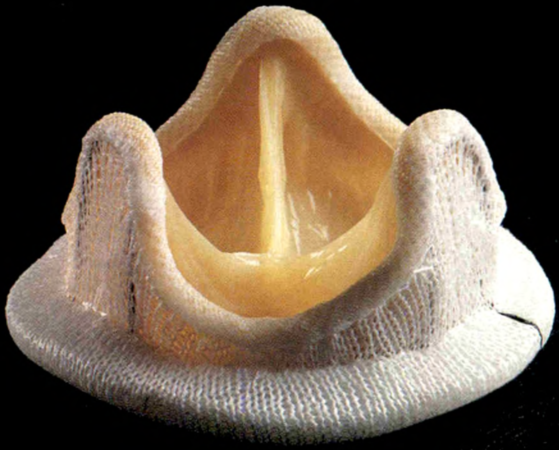

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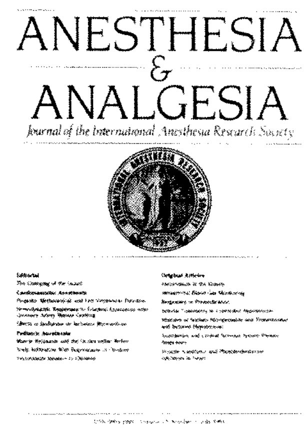

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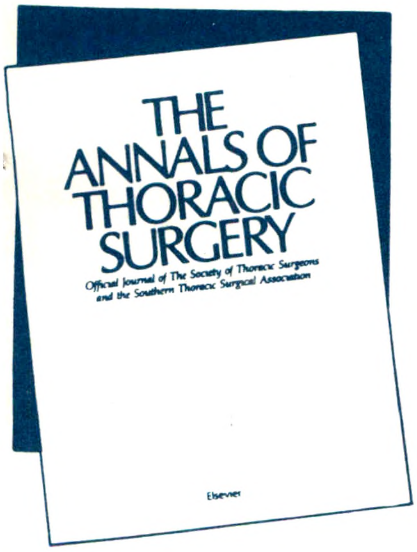
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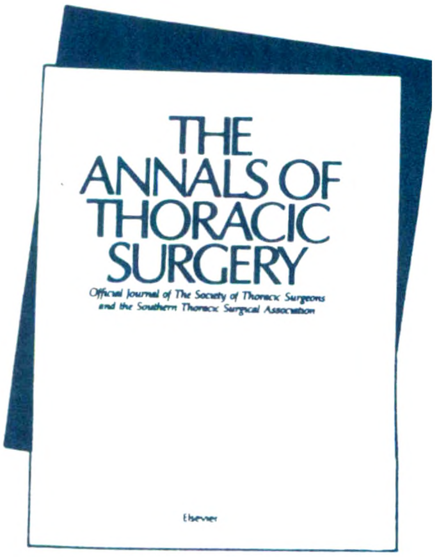
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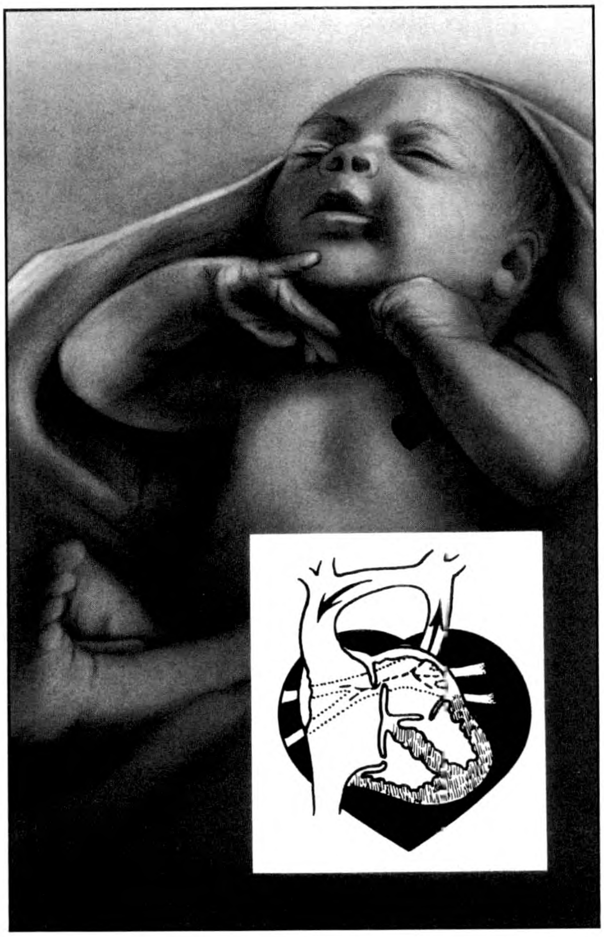
Current Controversies and Techniques in Congenital Heart Surgery

September 8–9, 1989
Baltimore, Maryland



Guest Editor
Anthony L. Moulton, MD

Introduction

Anthony L. Moulton, MD

University of Nebraska, Omaha, Nebraska

Major obstructive lesions to left ventricular outflow tract (LVOTO) comprise a wide spectrum of lesions, from "simple" coarctation, which was one of the first cardiovascular defects to be surgically corrected, to hypoplastic left heart, which only a decade ago was considered a universally fatal, surgically uncorrectable lesion. Although controversy in the rapidly changing approach to the newborn with hypoplastic left heart is understandable, presentations at major meetings about "newer" and "improved" methods for approaching coarctation sometimes stimulate even more discussion and debate. Hopefully this supplement will help delineate the role of various surgical procedures in the treatment of this complex and difficult group of patients. The topics of many of the articles in this supplement were presented at the symposium "Current Controversies and Techniques in Congenital Heart Surgery" in Baltimore, MD, September 8 and 9, 1989, but all of these have been expanded and updated within the past few months. Others are contributions solicited from authors who have innovative or extensive experience in the treatment of LVOTO to "round out" the discussion. All of the authors were urged to stress technical detail, pointing out indications, contraindications, and potential pitfalls. They were also asked to emphasize any particular innovations or "tricks" that they had found particularly useful during the operations described, rather than concentrating on results.

Much of the controversy surrounding the management of the newborn with coarctation has been rooted in the belief that modifying the surgical technique of the repair of the coarctation alone is somehow the major determinant of the immediate and ultimate outcome. Using "standard" techniques of repair, the experience of our own group and that of several others indicates that the answer to the rhetorical question posed in the title of Messmer and associates' article, "Does surgical technique influence the result?" would be a resounding "No." In our series of 51 infants undergoing subclavian flap repair of coarctation with an early mortality of 8%, the incidence of recoarctation was 6% in follow-up ranging from 2 to 8 years (mean, 4 years). This rate correlates with those (16% to 21%) reported by other groups using subclavian repair. It is also equivalent to that seen after resection with end-to-end anastomosis by other groups (Table 1).

Residual ductal tissue is often implicated as the culprit in recoarctation. Resection may prevent the development of the ductal "shelf" so often cited as the cause of recoarctation; but it does not prevent recoarctation itself, as so eloquently discussed in Jonas's article. It is interesting that the use of absorbable sutures likewise does not prevent recoarctation. The increasingly aggressive role of the interventional cardiologist is outlined in the approach advocated by Dr Rao and Dr Chopra.

One contribution to the problem of recoarctation may be limitation of flow caused by persistence of isthmic hypoplasia proximal to the site of coarctation repair. Resection or enlargement of the hypoplastic isthmic segment, as advocated by Lacour-Gayet and Planche, may relieve this component of flow limitation. However, Siewers and associates, with data from a group of well-studied patients, advance a powerful argument that this may not be necessary in the majority of patients in whom this segment will increase in size after "simple" coarctation repair. The truth probably lies somewhere between, with the selective application of both points of view. Even if the more "radical" repair of coarctation with isthmic enlargement is performed, the persistence of more proximal or intracardiac LVOTO may potentiate the development of recoarctation.

Similar concerns highlight the controversy between primary versus staged repair of interrupted aortic arch. Concerns about malalignment of the infundibular septum and possible worsening of LVOTO by the ventricular hypertrophy induced by pulmonary artery banding have always made us advocates of one-stage repair for interrupted aortic arch. This approach is supported by the study cited by Jonas, but Irwin and associates and Lamberti demonstrate spectacular results with a staged protocol, which may force reevaluation of this issue.

The management of the child with critical aortic stenosis and severe hypoplasia of the outflow tract has under-

Table 1. Coarctation of the Aorta: Repair in Infants Less than 2 Months of Age

Repair	No. of Patients	Mortality (%)	Follow-up (mo)	Recoarctation (%)	Recoarctation Rate (%/mo)
Subclavian flap repair					
Our series	42[a]	11	47	6	0.1
10 published series[b]	239	19	30	16	0.4
Resection with end-to-end anastomosis (7 published series)[b]	157	32	47	18	0.4

[a] 37 of the 51 infants were neonates, but because other published series discussed patients less than 2 months of age, this range was included to allow comparisons. [b] References available on request.

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gone progressively more radical approaches. In his inimitable style, Robert Anderson corrects many of the classic misconceptions about the morphology of this anatomical area. Standard surgical valvotomy techniques are discussed by Turley, Mayer, and Williams. Unfortunately, as an important counterpoint, an anticipated discussion of the role of balloon valvuloplasty in the management of aortic stenosis never materialized.

For the older child with complex aortic stenosis, Vouhé and Neveux present a well-organized approach, with indications for various techniques. Fleming gives some helpful hints for reoperating on the patient with a previous Konno aortoventriculoplasty who has outgrown or had degeneration of his or her other valvular prosthesis. Ilbawi and colleagues present an innovative technique for conserving the native valve, developed from a revised understanding of the morphology of aortic stenosis, as outlined in the article by Anderson and associates.

Clarke presents his large experience with aggressive homograft aortic root replacement for severe hypoplasia of the outflow tract, with commentary and cautions by Donald Ross. McKay and Ross then summarize their updated experience with pulmonary autografts, which attempts to maximize the benefits of autogenous tissue when the outflow tract is otherwise nonreconstructable.

Bypass of complex left-sided stenosis, with the incorporation of normal right ventricular outflow tract as proposed by Damus, has been used extensively by Lamberti's group. They summarize some of the technical modifications that they have developed, and they update their overall experience. DeLeon and associates emphasize the indications and variations of the Chicago surgical approach.

The last decade has witnessed the transition from total despair to a cautiously hopeful outlook for the newborns with hypoplastic left heart. William Norwood summarizes his pioneering efforts with this lesion and recounts the reasons for progressive modifications in "his" procedure. As described, he now uses a three-stage approach: A Norwood procedure, then a hemi-Fontan, followed by a modified Fontan. Jonas presents a lucid and extremely useful discussion of the management of patients with problems that need to be corrected before the first stage survivor can become a candidate for the "second-stage" Fontan. Both articles cite changes that have resulted in even further reduction in mortality to ranges that are lower than those associated with lesions generally assumed to be less lethal.

Behrendt, Bove, and Mavroudis have extensive experience with this hypoplastic left heart syndrome, and they present helpful hints for the role of transplantation as therapy for this lesion. However, this supplement suffers from a lack of a summary of the uniquely successful Loma Linda program with neonatal transplantation. This now includes almost 150 patients with extremely low operative mortality and 5-year actuarial survival of 84%.

In the era of fetal echocardiography and prostaglandins, previously fatal intracardiac lesions have become identifiable and immediately survivable, allowing attempted surgical palliation. Many of the most severe of these are easily recognizable variants of hypoplastic left heart, but many others have a combination of lesions that, if taken individually, are at least potentially correctable. I am concerned that our surgical therapy to date has frequently attempted to address each of these serially, occasionally with spectacular success but more often with disappointing and sometimes dismal failure.

A case in point is our own experience with neonatal coarctation. Despite a low initial mortality for "repair" directed at the coarctation alone, and a relatively low incidence of recoarctation, 42% of the patients with "complex" intracardiac lesions were dead on follow-up. Almost all of these deaths were associated with some form of severe LVOTO and varying degrees of left ventricular hypoplasia, and most were related to subsequent attempts to address the intracardiac anomaly.

I believe the time has come to broaden our horizons. We must look at coarctation and the other lesions addressed in this supplement as a spectrum of LVOTO comprised of multiple levels and forms. Immediate therapy should encompass an overall long-term plan for treating *all* of these levels of obstruction. With the improved results with more radical forms of therapy, one could argue that the results with many of these complex anomalies may be enhanced by an aggressive surgical attack on all levels in the neonatal period, rather than being wooed by our confidence to treat each of the components of the LVOTO in a serial manner.

On the other hand, there is another seemingly contradictory message in this supplement. For the older child, the applicability of cardiac transplantation is continually being expanded by innovative approaches such as those described by Chartrand and Menkis and associates and discussed by Michler and Rose. Their success might argue that relatively lower risk palliative procedures, which may eventually lead to transplantation, may be justified for selected patients. The problem, of course, is limited donor availability, so this cannot be a widely used approach.

Hopefully, this supplement will address many of these controversies and help clarify the role of the various procedures described. I wish to thank all of those who submitted their contributions and willingly modified them in response to editorial comments to help achieve the overall goal, and to thank Dr Thomas Ferguson and Carol Blasberg at *The Annals of Thoracic Surgery* and Mr Roger Morrison at Elsevier Science Publishing for their perseverance, help, and patience. I also thank Ms Linda Washington for her hard work with all of the manuscripts.

Addendum

The intensity of the debate about coarctation is revealed in the recent heated exchanges in *The Journal of Thoracic and Cardiovascular Surgery* (1990;100:817–9, 1991;101:165–6, and 1991;102: 153–5).

Surgical Correction of Coarctation in Early Infancy: Does Surgical Technique Influence the Result?

Bruno J. Messmer, MD, Carmine Minale, MD, Eberhard Mühler, MD, and
Götz v. Bernuth, MD

Departments of Thoracic and Cardiovascular Surgery and Pediatric Cardiology, University Hospital, Aachen, Germany

Between 1979 and 1988, a total of 53 infants less than 1 year of age underwent repair of coarctation. Thirty-seven patients (70%) were younger than 3 months. Median age was 0.9 month. Four different surgical techniques were used: resection with end-to-end anastomosis, patch enlargement, subclavian flap aortoplasty, and subclavian displacement aortoplasty (Meier-Mendonca technique). Hospital mortality was 7.5% and was limited to patients with additional complex intracardiac defects. Neither age nor surgical technique had an influence on the operative risk. Follow-up averaged 15 to 43 months for the four different groups. Restenosis developed in 9 (19%) of 47 patients regularly followed up, 5 (11%) of whom have had reoperation. Age at operation was not a predictor for restenosis, which occurred in 17.4% of patients less than 1 month and 20.8% of those greater than 1 month of age at operation. Patch enlargement and the subclavian displacement technique demonstrated the highest restenosis rates (42% and 43%, respectively). However, patients who underwent patch enlargement had less favorable pathological conditions. It is concluded that results of coarctation repair in early infancy do not depend as much on the operative method itself as on the specific pathological aspect, which largely determines the method of treatment. Some reservation must be made in regard to the subclavian displacement technique.

(Ann Thorac Surg 1991;52:594–603)

Coarctation can be responsible for severe congestive heart failure in neonates and infants less than 1 year of age especially when associated with intracardiac defects or a wide open poststenotic ductus perfusing the lower part of the body. For repair, various surgical techniques are available, each one with specific advantages and disadvantages as well as proponents and opponents among pediatric cardiologists and surgeons. The pathology of coarctation in this age group is so variable, however, that one single technique can hardly meet the specific requirements in each instance. Besides the classic differentiation between a preductal and postductal type, the stenosis can be membranous and short or tubular and of various lengths. Hypoplasia of the distal aortic arch, incorporation of the left subclavian origin into the stenosis, or the presence of a retroesophageal right subclavian artery taking off within or distal to the stenosis can not only complicate the picture but also determine the surgical technique.

This report presents the experience with and results of four different techniques for treating coarctation, namely, resection and end-to-end anastomosis, patch enlargement, subclavian flap aortoplasty (often called the Waldhausen technique) [1], and subclavian displacement aortoplasty (here referred to as the Meier-Mendonca technique) [2].

Material and Methods

Surgical Techniques

RESECTION AND END-TO-END ANASTOMOSIS. This technique (Fig 1) represents the original method for treatment of coarctation introduced clinically by Crafoord in 1944 and reported by him and Nylin [3] in the English-language literature in 1945. It is the only method that can completely remove the pathological structures within the aortic wall that are thought to be the primary cause of restenosis.

To achieve a large anastomosis without traction, mobilization of the left subclavian artery, the distal aortic arch, the left carotid artery, and the descending thoracic aorta all the way down to the diaphragm, must be done. This extensive preparation involves the risk of damaging important structures such as the vagal nerve and especially the recurrent laryngeal nerve or the lymphatic duct. It may also be necessary to interrupt the first pair, but rarely the second pair, of intercostal arteries.

Even with careful preparation, the anastomosis can be technically difficult, and it may be advantageous to place the suture in the posterior wall using a continuous polypropylene suture that can be pulled tight when turning to the anterior wall. This is not possible with PDS, the

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Presented at the Current Controversies and Techniques in Congenital Heart Surgery Meeting, Baltimore, MD, Sep 8–9, 1989.

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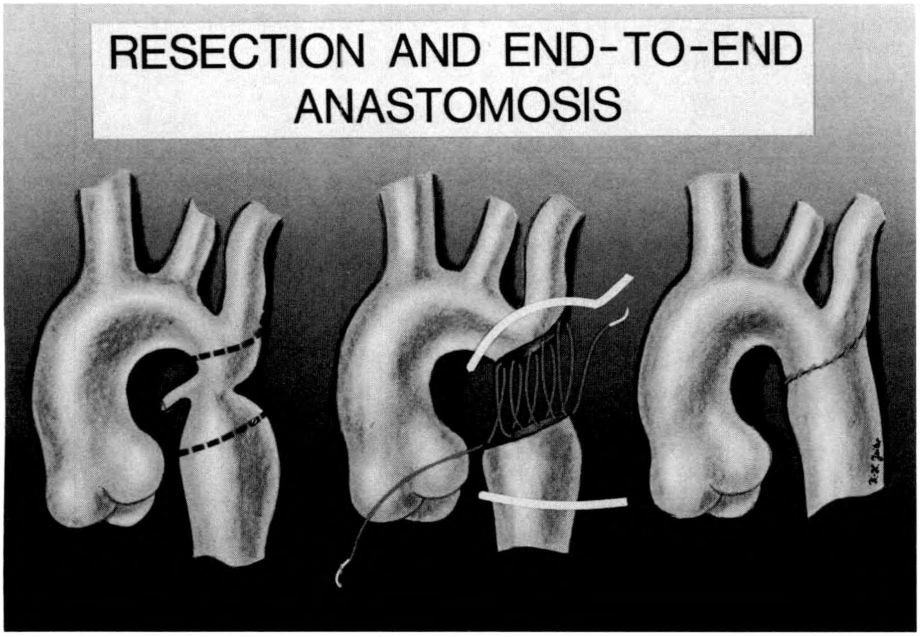

Fig 1. Resection and end-to-end anastomosis. The advantage of this method is total resection of pathological structures. The disadvantages are extensive preparation, possible damage to recurrent laryngeal nerve or lymphatic duct, eventual interruption of intercostal arteries, circumferential anastomosis, and possibility of a technically difficult anastomosis.

absorbable polydioxanone suture, because unlike Prolene, PDS gets rather sticky once it has been in touch with tissue. Nevertheless, PDS was the preferred suture material in the present series because it creates minimal scarring and allows normal growth of the anastomosis.

To adapt the proximal lumen to the generally wider opening of the distal segment, a longitudinal incision into the lateral aspect of the subclavian artery is often necessary. A similar incision on the medial side is not advisable because the often more friable tissue can give origin to tears and bleeding, difficult to control in the short axis of the aortic arch.

PATCH ENLARGEMENT. This technique (Fig 2) was introduced by Vosschulte [4] in 1961. Simple longitudinal splitting of the stenosis requires a minimum of preparation. All intercostal arteries remain intact, and the length of the stenosis is of virtually no importance. When necessary, the patch can be expanded into the left subclavian artery and the distal aortic arch. Whether the posterior shelf should be resected or not is still a matter of controversy. Hehrlein and colleagues [5] do not recommend it because of the danger of wall weakening and secondary aneurysm formation. Even with resection, pathological wall structures such as juxtaductal tissue generally remain

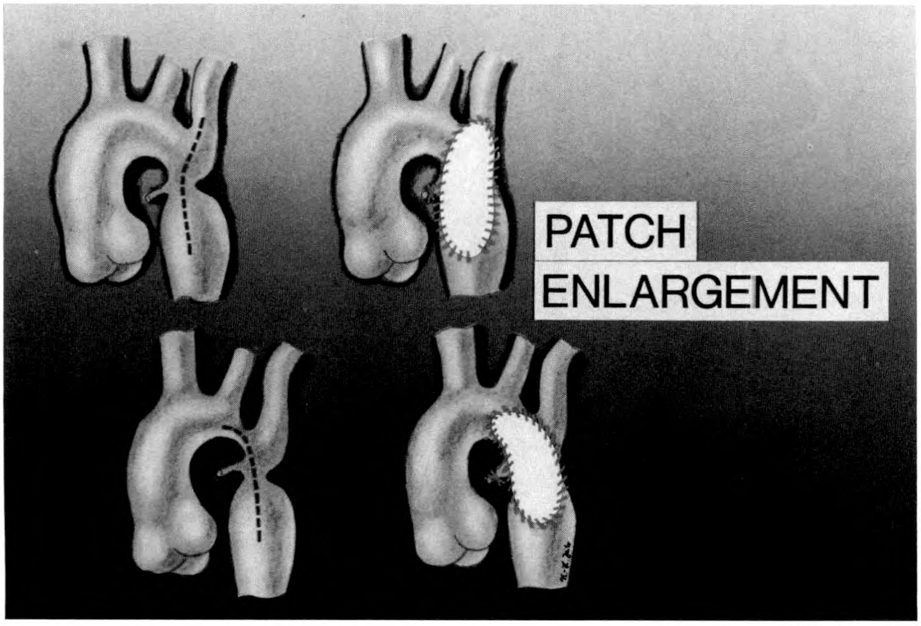

Fig 2. Patch enlargement. The advantages of this method are little mobilization of aorta, preservation of intercostal arteries, and applicability in long tubular stenoses. The disadvantages include pathological structures remaining in situ, implantation of foreign material without the potential to grow, and possible aneurysm formation.

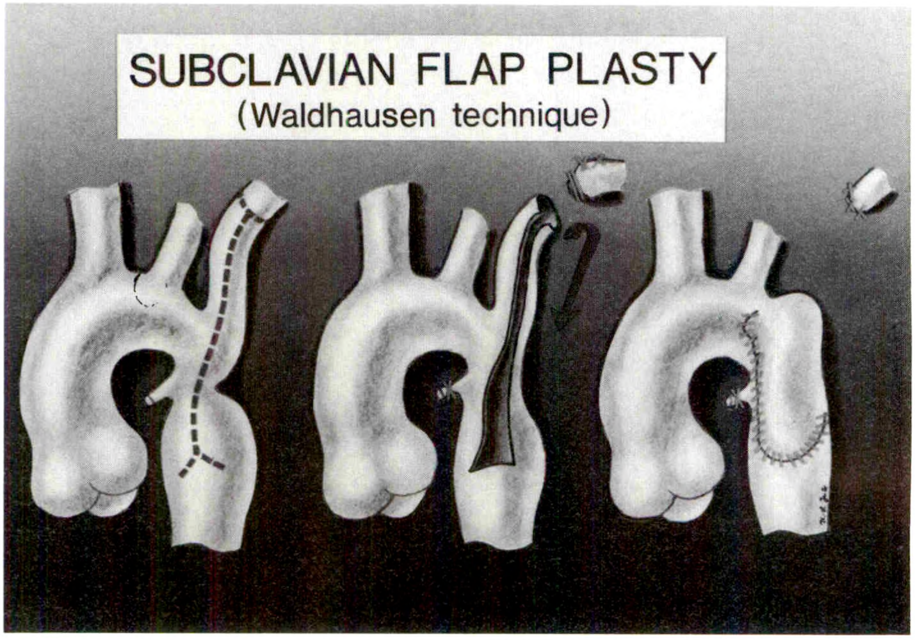

Fig 3. Subclavian flap aortoplasty (Waldhausen technique). The advantages of this method are little mobilization of aorta, preservation of intercostal arteries, and use of a patch of autologous tissue with the potential to grow. The disadvantages are sacrifice of left subclavian artery, pathological structures remaining in situ, and inapplicability in long tubular stenoses.

and can give origin to restenosis. For these reasons, the posterior shelf was not resected in the present series except in a few patients who had a membrane that was rather thin and easy to resect. A patent ductus as well as the ligament must be divided in all patients to eliminate the traction from this side, as it contributes to kinking and stenosis.

As patch material, either glutaraldehyde-preserved heterologous pericardium or, more frequently, a Gore-Tex patch was used. Both of these materials have the disadvantage of not growing. To minimize blood loss, a 7-0 or 8-0 Gore-Tex suture is strongly recommended when a Gore-Tex patch is used. Unlike Prolene, this suture does not leave bleeding suture holes. They are difficult to control even with fibrin glue, which scarcely adheres to Gore-Tex material.

SUBCLAVIAN FLAP AORTOPLASTY (WALDHAUSEN TECHNIQUE). In 1964, Waldhausen and Nahrwold [1] presented this method (Fig 3), which represents an almost optimal repair technique for infant coarctation. Besides requiring little dissection and preserving intercostal arteries, the technique results in a patch of autologous tissue with the potential to grow. The most severe disadvantage of this method is the sacrifice of the left subclavian artery.

When the left subclavian artery is prepared, the distal

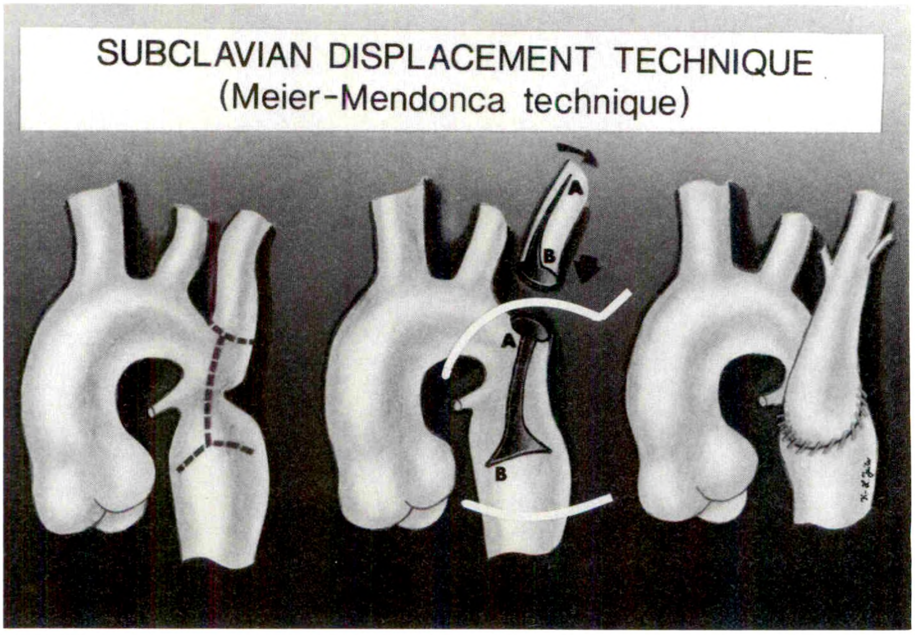

Fig 4. Subclavian displacement aortoplasty (Meier-Mendonca technique). The advantages of this method include little mobilization of aorta, preservation of intercostal arteries, use of a patch of autologous tissue with the potential to grow, and preservation of left subclavian artery. The disadvantages are pathological structures remaining in situ and inapplicability in long tubular stenoses.

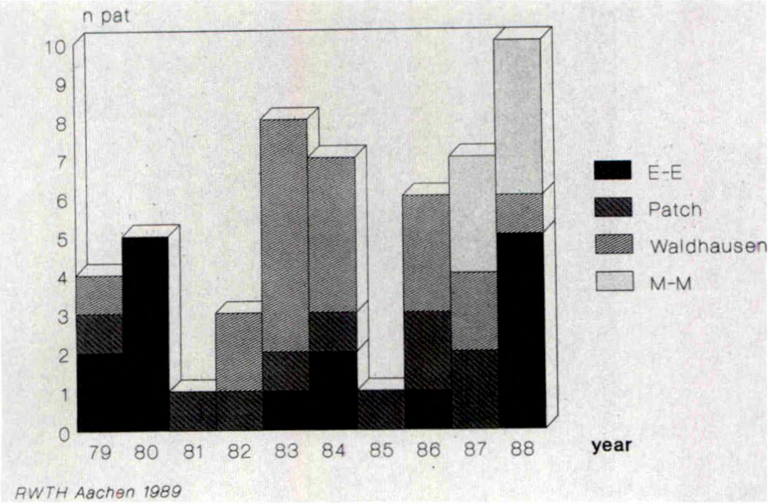

Fig 5. Number of patients treated per year and frequency of use of the surgical methods of repair. (E-E *= end-to-end anastomosis;* M-M = *Meier-Mendonca technique.)*

branches and especially the vertebral artery should be ligated to prevent a subclavian steal mechanism later on. Leaving the internal mammary artery and the thyrocervical trunk intact may improve collateral flow to the arm but shortens the length of the patch. To guarantee sufficient enlargement, the patch must have a minimum width of 12 mm over the maximum narrowing after suturing. This requires a subclavian artery diameter of 4 mm in its midpart and distal part. In long tubular stenosis, the method is rarely suitable. Inadequate length of the incision into the poststenotic aorta or too much traction at the lowest insertion point because of too short a patch may increase the possibility of restenosis.

SUBCLAVIAN ARTERY DISPLACEMENT (MEIER-MENDONCA TECHNIQUE). This technique (Fig 4), elaborated by de Mendonca and associates [6] and clinically introduced by Meier and co-workers [2], tries to incorporate the advantages of the subclavian flap aortoplasty while eliminating the disadvantage of subclavian artery sacrifice. Even though the method is intriguing, its realization is not always easy. It can be difficult to gain enough tension-free length without interrupting the vertebral and cervical arteries as well as the internal mammary artery. The technique is best suited to older infants and children with a proximally situated short stenosis, but even in such patients, it can be difficult to reach a tension-free reimplantation with sufficient enlargement of the stenotic area.

Patient Population

Between 1979 and 1988, a total of 53 infants less than 1 year of age underwent surgical intervention for coarctation with critical hemodynamics. The median age of the group was 0.9 month with 26 patients less than 1 month old and 27 patients more than 1 month old.

Excepting the relatively new Meier-Mendonca technique, no true prevalence of a specific method was observed over the years (Fig 5). The choice of method depended on the individual pathology. Resection with end-to-end anastomosis was used in 11 patients with a short stenosis and a good proximal aortic diameter, and patch enlargement was done in 14 patients with a long tubular stenosis often combined with distal arch hypoplasia or an aberrant right subclavian artery. In 20 patients, a Waldhausen aortoplasty was performed, and during the years 1987 and 1988, the Meier-Mendonca technique was used in 7 patients. A Clagett anastomosis was done in 1 patient who is not included further.

Median age was significantly lower in patients who had either end-to-end anastomosis or patch enlargement (0.7 month) than in patients who underwent the Waldhausen technique (1.8 months) and the Meier-Mendonca tech-

Table 1. Age and Surgical Technique

Technique	No. of Patients	Median Age (mo)
End-to-end anastomosis	11	0.7
Patch enlargement	14	0.7
Subclavian flap aortoplasty (Walhausen technique)	20	1.8
Subclavian displacement (Meier-Mendonca technique)	7	2.3

Table 2. Age Distribution for the Four Surgical Techniques[a]

	Months of Age			
Technique	<1	2–3	3–6	6–12
End-to-end anastomosis	7 (64)	1	2	1
Patch enlargement	9 (64)	3	1	1
Subclavian flap aortoplasty (Waldhausen technique)	8 (40)	4	5	3
Subclavian displacement (Meier-Mendonca technique)	2 (29)	3	1	1

[a] Numbers in parentheses are percentages.

nique (2.3 months) (Table 1). The percentage of patients less than 1 month of age was 64% each in the groups having end-to-end anastomosis or patch enlargement, 40% in the subclavian flap series, and only 29% in the subclavian displacement group (Table 2).

Associated severe intracardiac defects were present in half of the patients. This was even more pronounced in the neonatal group, in which two thirds of the patients had concomitant cardiac defects and only one third required operation for isolated coarctation. In patients older than 1 month, the proportion was exactly opposite (Table 3).

The series and the different techniques were evaluated with regard to early mortality, restenosis, and factors relevant to restenosis. If not linked to restenosis, late mortality was of minor interest, as it was generally due to the concomitant and predominant intracardiac lesion.

Follow-up ranges between 3 months and 10 years. It averages 36 months in patients with resection and end-to-end anastomosis, 43 months in the group with patch enlargement, 42 months in the Waldhausen aortoplasty group, and 15 months in patients with the Meier-Mendonca technique.

Restenosis was defined on the basis of the need for reoperation or the systolic arm-to-leg pressure gradient measured by ultrasound technique. Direct measurement with Doppler ultrasonography could not be obtained in all instances. Therefore, a systolic arm-to-leg pressure gradient of 20 mm Hg or more was taken as proof of restenosis. In some patients, late angiographic control data were obtained, and more recently, restudy by magnetic resonance technique has been started (Fig 6).

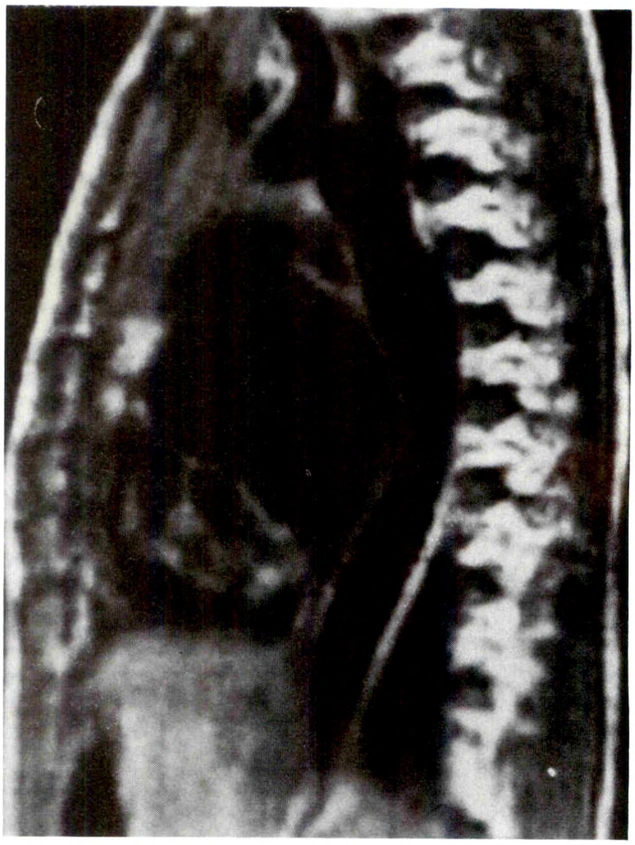

Fig 6. Nuclear magnetic resonance study 4 years after coarctation repair at 6 months of age with subclavian flap aortoplasty (Waldhausen technique).

Results

Hospital mortality was 7.5% for the series of 53 patients, and the 4 patients who died all had additional complex intracardiac malformations (Table 4). To date, we are aware of two late deaths, one due to progressive congestive heart failure resulting from a complex intracardiac malformation and the other, a result of reoperation for mycotic aneurysm after patch enlargement of the coarctation.

Restenosis has been observed in 9 (19%) of 47 survivors regularly followed up. Five of these patients have undergone reoperation, and another patient has had successful balloon dilation. The rate of restenosis was 0% with the Waldhausen technique, 9% after resection and end-to-end anastomosis, 42% for patch enlargement, and 43% for the Meier-Mendonca technique. In the patch enlargement group, 1 patient each needed reoperation for patch rejection and a mycotic aneurysm (Table 5).

Table 3. Associated Defects and Age Distribution[a]

Defect	Patients <1 Month Old	Patients >1 Month Old
Simple coarctation	9 (33)	17 (65)
Isolated	5	14
+PDA	4	3
Concomitant coarctation	18 (67)	9 (35)
+VSD	2	3
+Complex defects	13	4
+Hypoplastic LHS	3	2

[a] Numbers in parentheses are percentages.

LHS = left heart syndrome; PDA = patent ductus arteriosus; VSD = ventricular septal defect.

Table 4. Hospital Mortality[a]

Technique	No. of Patients	No. of Hospital Deaths
End-to-end anastomosis	11	0 (0)
Patch enlargement	14	2 (14)
Subclavian flap aortoplasty (Waldhausen technique)	20	2 (10)
Subclavian displacement (Meier-Mendonca technique)	7	0 (0)
Clagett anastomosis	1	0 (0)
Total	53	4 (7.5)

[a] Numbers in parentheses are percentages.

Table 5. Surgical Technique and Restenosis

Technique	No. of Patients	Follow-up[a] (mo)	Restenosis	Reoperation
End-to-end anastomosis	11	36 (3–116)	1 (9%)	1 (9%)
Patch enlargement	12	43 (6–120)	5 (42%)[b]	3 (25%)[b]
Subclavian flap aortoplasty (Waldhausen technique)	17	42 (3–78)	0	0
Subclavian displacement (Meier-Mendonca technique)	7	15 (6–22)	3 (43%)	1 (14%)

[a] Data are shown as the mean with the range in parentheses. [b] Restenosis was caused by a mycotic aneurysm in 1 patient and patch rejection in another, and both underwent reoperation.

No correlation was found between age at operation and incidence of restenosis (Table 6). The rate of restenosis was similar for patients younger than and older than 1 month of age, 17.4% and 20.8%, respectively. There was also no difference in regard to the respective technique in the two age groups.

In the majority of patients, 6-0 and 7-0 Prolene and PDS were used as suture material. Restenosis seems to be more dependent on the operative technique than on the suture material, even though PDS showed a lower incidence (3/35) than nonabsorbable materials including silk (6/16) (Table 7).

Comment

Surgical treatment of coarctation in early infancy is still controversial, even among experts. In spite of the fact that hospital mortality has steadily decreased, early surgical intervention should be restricted to patients with congestive heart failure resistant to medical treatment. In most instances, an additional intracardiac defect or a large, open poststenotic ductus are the determinant factors for early relief of the stenosis.

One of the major pitfalls after coarctation repair in this age group is restenosis, which is thought to be dependent on age at the initial operation as well as on the operative technique [7–9]. Resection and direct end-to-end anastomosis has often been considered responsible for a high percentage of restenosis, and the size of the aorta has been thought to influence late results [7]. Körfer and colleagues [10], however, had a restenosis rate of only 9% with this method and believed faulty technique rather than pathological anatomical conditions to be responsible.

To avoid extensive preparation, resection, and a circumferential anastomosis, other techniques such as patch enlargement, subclavian flap aortoplasty, and subclavian displacement aortoplasty became valuable alternatives. Data on restenosis rates with these techniques, however, are either scarce or as different as they are with primary end-to-end anastomosis. For the Waldhausen technique, the rates range between 0% and 22% in more recent series [11–13]. Little is known about the recurrence rate after patch enlargement because this method was more attractive for studies on aneurysm formation, and late results with the relatively new Meier-Mendonca technique are completely lacking.

Our experience confirms the good results that can be obtained with end-to-end anastomosis as well as with the subclavian flap technique and that are independent of age at operation. Loss of direct circulation to the left arm after subclavian flap repair is the only disadvantage of this method, but major complications are rare [14, 15]. Suture material and suture technique may have an important influence. The fact that restenosis did not occur in our series with end-to-end anastomosis or subclavian flap

Table 6. Age at Operation and Restenosis[a]

	Age <1 Month		Age >1 Month	
Technique	No. of Patients	Restenosis	No. of Patients	Restenosis
End-to-end anastomosis	7	0	4	1 (25)
Patch enlargement	8	4 (50)[b]	4	1 (25)[c]
Subclavian flap aortoplasty (Waldhausen technique)	6	0	11	0
Subclavian displacement (Meier-Mendonca technique)	2	0	5	3 (60)
Total	23	4 (17.4)	24	5 (20.8)

[a] Numbers in parentheses are percentages. [b] One patient had a mycotic aneurysm. [c] This patient had patch rejection.

Table 7. Suture Material and Restenosis[a]

Technique	Prolene	PDS[b]	Gore-Tex	Silk
End-to-end anastomosis	1 (0)	9 (0)	0	1 (1)
Patch enlargement	11 (3)	0	3 (2)	0
Subclavian flap aortoplasty (Waldhausen technique)	1 (0)	19 (0)	0	0
Subclavian displacement (Meier-Mendonca technique)	0	7 (3)	0	0

[a] Numbers in parentheses represent patients with restenosis. [b] Polydioxanone.

repair when the absorbable PDS suture was used demonstrates the superiority of this type of suture material.

Less favorable is our experience with patch enlargement. However, it must be taken into consideration that this technique was used only when a long tubular stenosis, often including the distal arch and the origin of the left subclavian artery, was present or when an aberrant right subclavian artery was present. Therefore, this group represents a rather negative selection in view of the anatomical pathological circumstances. On the other hand, the posterior shelf was not resected in all of these patients. This can hardly be the only triggering mechanism for restenosis, however, because in the group with subclavian flap aortoplasty, resection of the shelf was not routinely done either. Other mechanisms such as increased turbulence not absorbed by the stiff patch material must be considered. Resection of the shelf is often advocated [1, 16, 17], but this can give origin to aneurysm formation by weakening the aortic wall at a place of increased wall tension caused by residual turbulence.

Disappointing, at least in our hands, are the results with the Meier-Mendonca technique. Massive concentric thickening of the residual aortic wall and the displaced subclavian artery wall has been found at reoperation. To date, no other long-term results have been published, and it is difficult to draw conclusions about the method from our experience only. We speculate that too much longitudinal traction on the stretched subclavian artery could induce such wall changes.

In the early 1980s, balloon dilation was introduced for relief of coarctation [18, 19]. Even though this method may represent a valuable alternative in skillful hands, especially for patients with additional complex intracardiac defects, high restenosis rates and a considerable risk of aneurysm formation mandate caution [20].

In conclusion, based on our experience, results of coarctation repair in neonates and young infants are not dependent as much on the operative method itself as on the specific pathological aspect and the selected method of its treatment. For circumscribed short stenosis, both resection with end-to-end anastomosis and subclavian flap repair give excellent results. Long tubular stenoses or additional vascular malformations require the less advantageous patch enlargement technique.

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COMMENTARY: Is There a Role for Prosthetic Patch Aortoplasty in the Repair of Coarctation?

The improved techniques and results for resection and primary end-to-end anastomosis in the repair of aortic coarctation have created a wave of enthusiasm for this approach. The ease with which primary aortic repair can be performed, even through a median sternotomy, makes one-stage correction of interrupted aortic arch an operation of choice in many surgical centers (including ours). The descending aorta can be mobilized to such an extent that it usually can be brought up to a position underneath the aortic arch with minimal tension and allow for a reasonable anastomosis. Furthermore, the success of balloon angioplasty techniques in treating restenosis often transforms this outcome into a minor inconvenience when compared with the advantages of one-stage total correction. Considering the aggressiveness with which problems of the aortic arch are now approached, the role of prosthetic patch aortoplasty in repairing a discrete aortic coarctation might seem quite limited. But before it is relegated to the category of historical interest only, it is relevant to review the several advantages of this technique in the modern era.

Is Patch Aortoplasty Really Bad?

Before discussing the highlights of this technique, it pays to get the worries about it out in the open. Opponents of patch aortoplasty are usually quick to point to the potential for long-term aneurysm formation opposite the patch. Furthermore, some are concerned about the use of prosthetic material, which not only will never grow, but which might become infected. As the use of this technique does not require the removal of the diseased area, and in fact, requires that it be left in place and serve as the posterior wall of the reconstructed aorta, the possibility of early recurrence or of late degeneration of the aorta is often raised.

In our experience, these objections have remained theoretical, and none of these problems have occurred. Since January 1987, 54 patients with aortic coarctation (30 less than 1 year of age) have received repair at the Duke Heart Center through prosthetic patch aortoplasty using a fairly consistent technique. Mortality has been 0% (even in 15 infants less than 1 week of age with critical coarctation), and recurrence rate has been 5% (2/54). (Both recurrences occurred in patients in whom a technical reason can be postulated—1 patient had too small a patch placed and 1 had a long area of tubular hypoplasia that might have lent itself better to excision and primary repair). The methods (and results) parallel those reported by Yee and co-workers in 1986 [1].

Aneurysm formation has not been seen in any of our patients. Although the follow-up period is short with respect to this complication, there are several reasons why we do not expect it to occur. (1) We do *not* resect or incise the intima along the coarctation ridge posteriorly. There is experimental evidence [2] that this may lead to the formation of aneurysms in otherwise normal aortas, presumably by creating a weakness in the intima. By leaving the intimal ridge intact, there is no disruption in the integrity of the aorta posteriorly to lead to aneurysm formation. (2) We prefer the use of expanded polytetrafluoroethylene (eg, Gore-Tex) material to Dacron, homograft, or pericardium. This material is exceptionally strong and does not fray, even if sutures are placed close to its edge. Furthermore, unlike homograft or pericardium, we do not expect it to deteriorate over time. (3) The repair is performed with a large piece of prosthetic material that extends well above and below the narrowed area of aorta. We emphasize the importance of making this patch generous in size, usually one and one half times the diameter of the descending aorta. This usually results in the elimination of any gradient, and we suspect that a good hemodynamic result has less likelihood of leading to aneurysm formation regardless of the technique used. Although it is suggested that the formation of aneurysms may take 12 to 15 years or more, this only supports our belief that the aneurysms observed after this repair occur most often if the technical considerations mentioned above are violated, as they most commonly were with the early methods of this technique and with the types of material then available. Furthermore, the risk of aneurysm formation after balloon angioplasty of recurrent stenosis at the site of an end-to-end anastomosis may produce a considerable problem. It is important that we not abandon a valuable operative approach if its failings were due to problems that can be improved upon using better understanding and technology.

The use of prosthetic material has not been a major concern as this is not commonly a foundation for decision making in pediatric heart surgery. Prosthetic material is frequently used to patch defects or to enlarge reconstructed pulmonary outflow tracts and should be considered whenever it can help enhance a repair. This leads us to consider the advantages of this type of repair.

What Are the Advantages of Prosthetic Patch Aortoplasty?

This is a simple, quick, and effective technique to relieve the hemodynamic problems imposed by aortic coarctation. The aorta does not need to be extensively mobilized, which minimizes the amount of dissection, especially in operations for restenosis after an end-to-end repair. We have had no postoperative chylothoraces in our series. Aortic clamp time is short (especially in neonates), and the average clamp time in our patients is 12 minutes (range, 7 to 18 minutes). The incidence of postoperative paraplegia in this series is 0%. Bleeding, when it occurs, is usually anterior and easy to control. There is no posterior suture line that can bleed and lead to a site for residual gradients after attempts at control. This type of repair places no tension on the repair site—a problem that can result in late restenosis after an end-to-end reconstruction and that can be possibly a source of suture line disruption leading to the formation of false aneurysms. Unlike the use of the subclavian flap procedure, the use of prosthetic patch repairs allows for the instant elimination of any serious hemodynamic obstruction by providing an abundance of patch material that can easily extend proximally and distally well beyond the region of disease.

When is Patch Aortoplasty a Uniquely Valuable Option?

Although there are surgeons who prefer to avoid patch aortoplasty, the technique should be considered in certain circumstances.

When the Patient Has a Significant Intracardiac Defect That Makes Combined Repair Through a Median Sternotomy Incision Desirable [3]

We have operated on 6 patients in our series who had cardiac defects (4 ventricular septal defect, 1 double-outlet right ventricle, 1 aortic valvar stenosis) amenable to correction on cardiopulmonary bypass that we believed would be best treated by concomitant repair of the aortic coarctation. In each patient, the coarctation site was repaired with a patch of prosthetic material. In infants undergoing repair through this approach, we now prefer the use of homograft material in much the same manner that it is used for patients undergoing aortic arch reconstruction as part of the palliative reconstruction for hypoplastic left heart syndrome. In patients with aortic arch interruption, we commonly employ end-to-end reconstruction of the aorta, although the use of patch material for the anterior portion of the anastomosis can help eliminate tension. An alternative approach to these patients is to repair the coarctation separately through a left thoracotomy and then reposition the patient and repair the cardiac defect through a sternotomy during the same anesthetic period. We have done this for 2 patients and found that the patch aortoplasty did not produce any complications with the immediately subsequent run on cardiopulmonary bypass. The decision of which option to use depends on the location of the coarctation, the nature of the aortic arch, and the type of cardiac repair that is necessary.

In Infants With Critical Coarctation and "Borderline-Sized" Left Ventricles (20–25 mL/m^2)

Use of patch aortoplasty in these patients allows the ductus arteriosus to be preserved and to be left open after completion of the aortic repair. We have had 2 such patients (left ventricular volumes of 20 and 23 mL/m^2) in our series treated in this manner. In both patients, the ductal patency was maintained with prostaglandin E_1. This enabled the patients to use their ductus to "pop-off" their high pulmonary artery pressures until the left ventricle demonstrated its adequacy as a systemic pump, as evidenced by reversal of flow through the ductus with the shunt becoming left-to-right from an initial postoperative right-to-left (documented by daily color-flow Doppler echocardiography and umbilical artery blood gases). This physiological shift occurred in our 2 patients on day 5 and 2 after coarctation repair, respectively. Administration of prostaglandins is then stopped to allow the ductus to close spontaneously. Both patients had successful convalescences after coarctation repair despite their high preoperative risk.

In Patients With Recurrent Coarctation After End-to-End Repair That Does Not Respond Well to Balloon Angioplasty

We have been referred 5 such patients. One of these patients had partial paraplegia with substantial weakness of his lower extremities, and the operative report from the previous procedure noted a fairly long aortic clamp time with difficulty encountered in controlling the bleeding at the anastomosis site. This patient's cardiac defect was easily repaired through a median sternotomy and with cardiopulmonary bypass using a short (14-minute) period of total circulatory arrest at 18°C. The result was excellent. The use of a prosthetic patch for recurrent stenosis simplifies a potentially difficult procedure by eliminating the need for mobilization of a scarred aorta, and the procedure can be performed through a thoracotomy in a standard fashion or through a sternotomy using cardiopulmonary bypass and total circulatory arrest as described.

When Should Patch Aortoplasty Be Avoided?

Infants with a long area of tubular hypoplasia present a difficult problem for any technique, but may be best treated by resection of the abnormal area and end-to-end reconstruction. Using a prosthetic patch for these patients has several disadvantages including the stiffness of the material compared with the friability of the posterior aortic wall. Furthermore, the patch becomes the majority of the aortic wall over a long segment, and the long-term result with respect to restenosis is likely to be less satisfactory.

True hypoplasia of the aortic arch in infants can be repaired through a left thoracotomy using end-to-end reconstruction after extensive mobilization of the aorta [4] or through a median sternotomy using a patch [3] or an end-to-end technique. The use of prosthetic patch aortoplasty by way of a left thoracotomy to reconstruct severely hypoplastic aortas should be avoided.

It is essential for surgeons to examine their own results with a procedure and compare them with alternative approaches. Aortic coarctation should be reparable with low mortality (even in critically ill neonates) and with a low incidence of restenosis. When properly performed, prosthetic patch aortoplasty allows the achievement of both these goals. Use of the correct technique and proper materials should make long-term aneurysm formation as unlikely with this as with any alternative approach. Although it is sometimes difficult for a surgeon to defend the less fashionable option, current results with prosthetic patch aortoplasty suggest that it should be considered an acceptable and effective technique to fix a difficult problem with the aorta.

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EDITOR'S NOTE

Dr Ungerleider's viewpoint is included in this supplement for the sake of discussion, but I believe there is a wealth of published evidence that patch aortoplasty carries an unacceptable incidence of late aneurysm formation. This certainly has been our experience at the University of Nebraska, where Bill Fleming was an early advocate for this procedure, for many of the reasons cited by Ungerleider.

The aneurysms are "true" aneurysms involving all layers of the aortic wall, and they are usually asymptomatic but can reach impressive size. Repair frequently requires a tour de force of dissection and usually entails replacement of the aneurysmal segment with a prosthetic graft, with all of the concerns that that entails. We would therefore urge avoidance of patch aortoplasty for primary repair, and reserve it for the patient in whom recoarctation not amenable to balloon dilation develops after other techniques of repair.

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Coarctation: Do We Need to Resect Ductal Tissue?

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A review of the literature as well as a retrospective review of 100 neonates undergoing operation for coarctation at Children's Hospital in Boston between 1972 and 1984 has not established clear superiority for either resection and end-to-end anastomosis or subclavian flap aortoplasty with respect to risk of recurrent coarctation. However, there is histological evidence that the juxtaductal coarctation shelf is composed of smooth muscle of ductal origin, which subsequently fibroses. This abnormal tissue may be at risk for late aneurysm development, particularly if balloon dilation angioplasty is required. The fact that this abnormal tissue is not removed by the subclavian flap procedure is one of the inherent disadvantages of that procedure. Other disadvantages include the need to sacrifice the left subclavian artery and the fact that, unlike resection and end-to-end anastomosis, the subclavian flap procedure does not lend itself to augmentation of the hypoplastic distal aortic arch. Furthermore, occasionally a secondary coarctation membrane is present within the distal aortic arch, and though it is readily detected during the resection procedure, it can be missed with the subclavian flap procedure. Based on these considerations rather than on a demonstrated superiority of either procedure, my colleagues and I currently prefer resection and end-to-end anastomosis over subclavian flap aortoplasty.

(Ann Thorac Surg 1991;52:604–7)

Much of the pioneering work in the surgical management of coarctation was undertaken at Children's Hospital in Boston by Dr Robert Gross [1]. Gross recommended resection and end-to-end anastomosis with a continuous silk suture posteriorly and interrupted everting horizontal mattress sutures anteriorly. In 1962, in a review of the first 500 cases of coarctation at Children's Hospital, Gross and Schuster [2] concluded that the optimal age for coarctation repair was approximately 10 years. This was based on the concern that there may be a lack of growth potential of the circumferential anastomosis. The limited growth potential of circumferential anastomoses appeared to be confirmed by a 1980 study by the Toronto group [3]. This study suggested that by 7 years postoperatively, the risk of recurrent coarctation after resection and end-to-end anastomosis was 50% (Fig 1).

Thus, in 1983 when the neonatal arterial switch experience was begun in Boston [4], there was concern regarding the potential for stenosis at the two great-vessel anastomoses as well as the circumferential coronary artery anastomoses. At most recent follow-up, however, after more than 400 arterial switch procedures during an 8-year period, not 1 child has required reoperation for aortic anastomotic stenosis. Of the 9 children who have required reoperation for pulmonary artery stenosis up to September 1989, all were among the first 40 to undergo the arterial switch procedure. It was this finding that led my colleagues and me to reexamine our experience with circumferential anastomoses in the management of coarctation of the aorta.

Retrospective Review of 100 Neonatal Coarctation Repairs

In 1986, we [5] published a retrospective review of 100 neonates who had undergone operation for coarctation of the aorta at Children's Hospital between 1972 and 1984. All of them were less than 30 days of age. The mean age was 12 days and the mean weight, 3.1 kg. Twenty-nine patients had simple coarctation, 32 had coarctation with ventricular septal defect, and 39 had coarctation with associated complex anomalies such as single ventricle. Seventy patients had subclavian flap aortoplasty, 24 had resection and end-to-end anastomosis, and 6 had miscellaneous other procedures.

This analysis was undoubtedly complicated by the changing face of coarctation management throughout this time frame. For example, the majority of the resections and end-to-end anastomoses were performed before 1977 and the subclavian flap procedures, from 1977 onward. The introduction of prostaglandin E_1 in 1979 permitted smaller, sicker children with more complex anomalies to be resuscitated and undergo operation. In 1983, absorbable polydioxanone suture was introduced. Most importantly, in 1984, Dr James Lock introduced balloon dilation angioplasty for recurrent coarctation. Thus, after 1984 the threshold for intervention for recurrent coarctation was lowered considerably.

Figure 2 compares actuarial freedom from reintervention for recoarctation for the two operative techniques [5]. Freedom from reintervention for recoarctation at 5 years was 93% for neonates who underwent resection and end-to-end anastomosis, whereas it was 75% for those who underwent subclavian flap aortoplasty. In addition to the neonatal group of 100 patients, we also reviewed the cases of 22 infants who were between 1 month and 1

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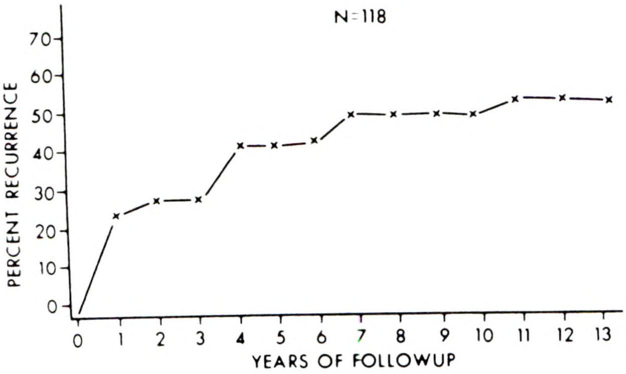

Fig 1. Actuarial incidence of recoarctation after resection and end-to-end anastomosis reported by the Toronto group in 1980. Incidence exceeds 50% after 7 years postoperatively. (Reprinted from Williams WG, Shindo G, Trusler GA, Dische MR, Olley PM. Results of repair of coarctation of the aorta during infancy. J Thorac Cardiovasc Surg 1980;79:603, 603–8; by permission of Mosby-Year Book, Inc.)

year of age at the time of subclavian flap angioplasty. In this group, the actuarial freedom from reintervention for recoarctation was 95% at 5 years.

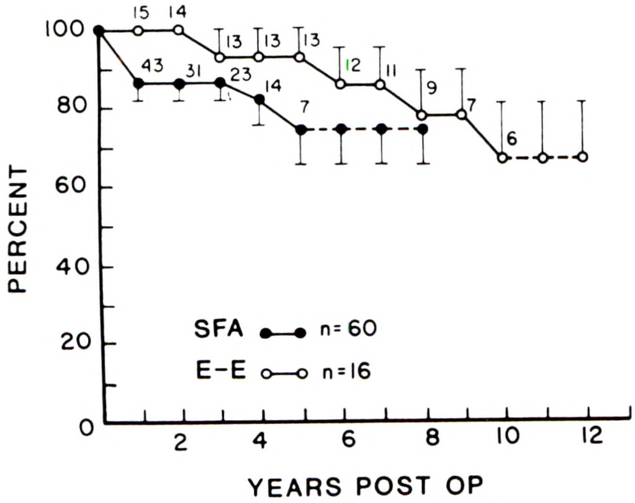

Fig 2. Actuarial freedom from reintervention for recoarctation after subclavian flap aortoplasty (SFA) and resection with end-to-end anastomosis (E-E) in neonates (≤30 days old), Children's Hospital, Boston, 1972 through 1984. (Reprinted from Ziemer G, Jonas RA, Perry SB, Freed MD, Castaneda AR. Surgery for coarctation of the aorta in the neonate. Circulation 1986;74[Suppl 1]:25–31; by permission of The American Heart Association, Inc.)

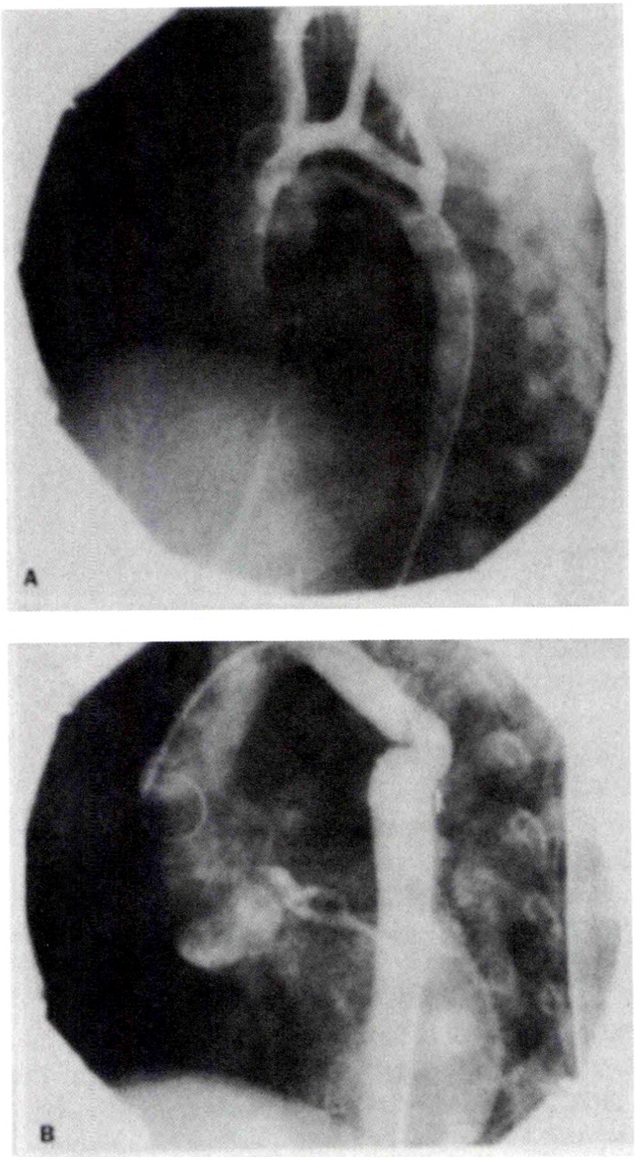

Fig 3. (A) Preoperative neonatal coarctation demonstrating patent ductus arteriosus. (B) Angiogram made 19 months after subclavian flap aortoplasty in the same patient demonstrates a prominent juxtaductal shelf. (Reprinted from Ziemer G, Jonas RA, Perry SB, Freed MD, Castaneda AR. Surgery for coarctation of the aorta in the neonate. Circulation 1986;74[Suppl 1]:25–31; by permission of The American Heart Association, Inc.)

These findings led us to speculate that molding of ductal tissue resulting in a posteriorly placed shelf was perhaps responsible for this difference in recurrence rate between the two groups having subclavian flap aortoplasty. In the older group, ductal tissue had already matured by the time of operation, making postoperative recurrence less likely. Our speculation tended to be supported by the typical angiographic appearance of children in whom recurrence developed after neonatal subclavian flap angioplasty. Figure 3 illustrates the obvious shelf projecting into the lumen despite the fact that the subclavian flap can be clearly seen to extend well past the area of recoarctation.

We attempted to find morphological predictors of recoarctation after the subclavian flap procedure. However, morphological factors such as length and diameter of the subclavian artery, the diameter of the distal aortic arch, the length of the isthmic segment (that is, from the subclavian takeoff to the coarctation area), and the diameter of isthmus were not predictors of recurrence after subclavian flap angioplasty.

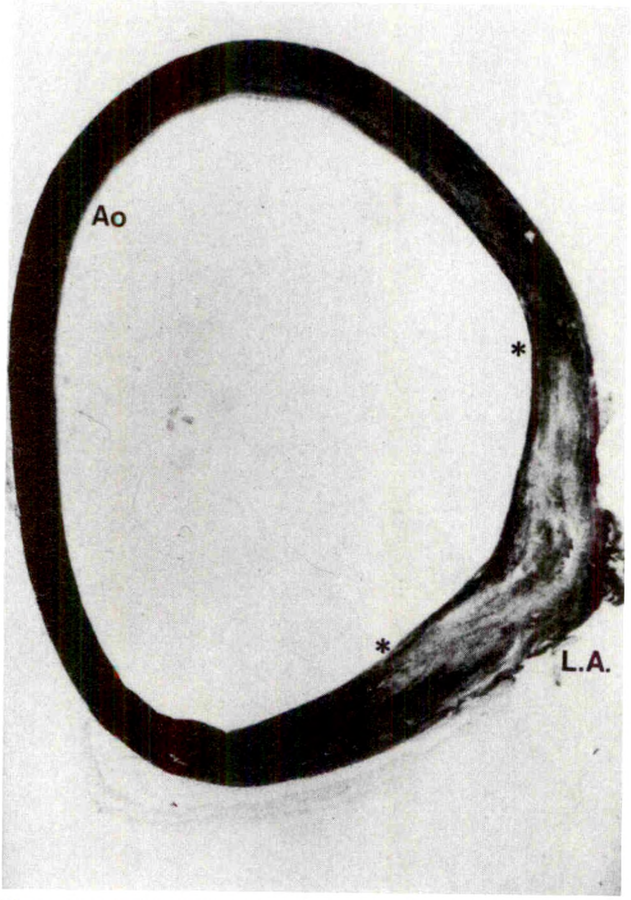

Fig 4. Histological examination of normal aorta reveals pale-staining ductal tissue extending only partially around circumference of aorta (Ao). (L.A. = *ligamentum arteriosum.) (Elastic tissue stain.) (Reprinted from Elzenga NJ, Gittenberger-de Groot AC. Localised coarctation of the aorta. An age dependent spectrum. Br Heart J 1983;49: 317–23; by permission of The British Medical Association.)*

Inherent Disadvantages of Subclavian Flap Aortoplasty

Although our 1986 review [5] did not clearly establish superiority of resection and end-to-end anastomosis relative to subclavian flap aortoplasty, our current practice is to undertake resection and end-to-end anastomosis using absorbable polydioxanone suture. This has been largely in response to what we perceive to be the inherent disadvantages of the subclavian flap procedure. First, is the inherent disadvantage of having to sacrifice the left subclavian artery. Second, use of the subclavian artery limits the options for dealing with a hypoplastic distal aortic arch [6]. Third, and rarely, there can be a secondary coarctation shelf in the distal aortic arch. During a subclavian flap aortoplasty procedure, this shelf can be missed, but it is readily detected during resection and end-to-end anastomosis. Fourth, there is the possibility of an increased risk of late aneurysm formation in the abnormal tissue that is left in the posterior aortic wall as part of the subclavian flap procedure but that is removed entirely with resection and end-to-end anastomosis. This risk can be exacerbated if there is a need for balloon dilation angioplasty [7, 8].

Ductal Tissue and Coarctation Histological Data

There is a considerable amount of histological evidence that ductal tissue plays an important role in the mechanism of coarctation formation. Dr Nynke Elzenga [9] in conjunction with Dr Gittenberger-de Groot has provided important documentation of the following hypothesis. They propose that in the presence of a left-sided obstructive lesion such as a bicuspid aortic valve, there is an increase in right-to-left flow through the ductus arteriosus during in utero development. This results in a migration of ductal tissue into the adjacent aortic wall. Conversely, if there is a right-sided obstructive lesion such as tetralogy, left-to-right flow through the ductus results in the commonly seen stenosis of left pulmonary artery origin. Presumably this latter problem should be dealt with in a fashion similar to aortic coarctation, that is, by resection and end-to-end anastomosis whenever possible.

In the normal aorta, the ductal tissue, which is pale staining and is composed largely of smooth muscle cells, extends only partially around the circumference of the aorta (Fig 4). In the child with coarctation, the ductal tissue extends around the full circumference of the aorta (Fig 5). As it contracts and subsequently fibroses, it creates constriction in that area.

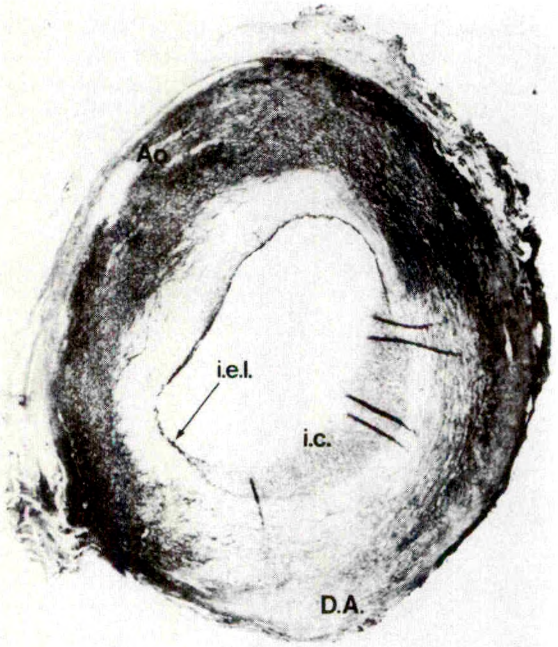

Fig 5. Histological section of juxtaductal area in the child with coarctation reveals pale-staining, ductal smooth muscle extending circumferentially around aorta (Ao). (D.A. = *ductus arteriosus;* i.c. = *internal cushion;* i.e.l. = *internal elastic lamina.) (Elastic tissue stain.) (Reprinted from Elzenga NJ, Gittenberger-de Groot AC. Localised coarctation of the aorta. An age dependent spectrum. Br Heart J 1983; 49:317–23; by permission of The British Medical Association.)*

Table 1. Recurrent Coarctation in Infancy

Year	Age < (mo)	LSCA (%)	Resect (%)	Follow-up (y)
1980	12	. . .	54	7
1980	1.5	. . .	2.6	4
1984	12	. . .	9	10
1984	12	17	. . .	1
1985	12	12	35	?
1985	3	25	. . .	5
1986	12	16	16	5
1986	3	12	. . .	5
1986	3	22	. . .	0.5
1988	3	11	14	5

Additional histological evidence has emerged from a study by Isner and colleagues [10], who reported that cystic medial necrosis was seen in all 33 coarctation specimens resected from children. They suggested that the necrosis, which was severe in two thirds of the patients, may explain the risk of aneurysm formation after balloon angioplasty. This may also explain aneurysm formation in the aortic wall opposite prosthetic patches used for synthetic patch aortoplasty [11, 12].

Review of Results of Operation for Coarctation

Table 1 illustrates the wide range of results that have been achieved with both the subclavian flap procedure and resection and end-to-end anastomosis. For the subclavian flap procedure, the reported risk of recurrent coarctation ranges between 11% and 25%, whereas for resection and end-to-end anastomosis, the range is between 2.6% and 54%. Thus, it is impossible to draw any clear conclusion from these reports as to which is the preferable procedure.

Conclusion

In the face of much conflicting information, I believe that at present there are no clear data as to which of the two techniques for coarctation repair has a lower risk of recurrence. However, the inherent disadvantages of the subclavian flap procedure currently make resection and end-to-end anastomosis the more attractive alternative.

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Coarctation and Hypoplasia of the Aortic Arch: Will the Arch Grow?

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Hypoplasia of the transverse aortic arch of various degrees of severity is commonly seen in infants who have coarctation of the aorta. It is more often present when the coarctation is associated with intracardiac lesions that diminish or limit forward flow in the ascending aorta and promote right to left flow through an arterial duct. The increased frequency of surgical treatment of infants with complex coarctation, which is in part related to the ability to stabilize their condition with prostaglandin E_1, has posed the question of the potential for growth and development of the originally hypoplastic aortic arch after conventional repair of aortic coarctation. Review of our experience with transverse aortic arch hypoplasia, found in 33 (32%) of 102 infants undergoing coarctation repair by subclavian flap aortoplasty or classic resection and end-to-end anastomosis, revealed excellent growth of the transverse arch after repair in all patients available for linear follow-up. The currently proposed extended arch repair should be reserved for the small group of infants with transverse aortic arch to ascending aorta diameter ratios (arch indices) of less than 0.25.

(Ann Thorac Surg 1991;52:608–14)

Poor growth and underdevelopment of various portions of the aortic arch are frequently associated with coarctation of the aorta. This is especially true in infants with associated major cardiac abnormalities who require surgical intervention during the first or second month of life. The abnormality in growth, when present, usually takes the form of some degree of hypoplasia of the distal aortic arch, that portion of the arch between the origin of the left carotid artery and the origin of the left subclavian artery. The arch in this region may be not only narrow in diameter but longer than normal. When such a long and narrow segment is present, it is natural to question whether operative procedures designed to correct the stenosis created by the coarctation segment will effectively and completely relieve pressure gradients between the ascending and descending aorta.

To answer the question whether the arch does, in fact, grow after correction of discrete coarctation stenosis, we examined the preoperative and postoperative "arch index" in a group of infants with coarctation of the aorta and transverse arch hypoplasia who were treated for coarctation by conventional means.

Material and Methods

Background

The etiological basis of tubular arch hypoplasia is largely related to morphological abnormalities of the left heart that result in diminished preductal aortic flow in the fetus [1–3]. That isolated coarctation can coexist with tubular hypoplasia is well established and would appear to be as much related to the presence of ductal tissue within the wall of the aortic isthmus as to fetal blood flow patterns [4–8]. Certainly, changes in the ductal tissue within the aortic wall when associated with spontaneous closure of the arterial duct would seem to be an additional etiologic factor for isolated coarctation. These changes can occur immediately after birth or can evolve over weeks, but usually become static by the fourth to sixth postnatal week [2].

The consideration of the varying degrees of arch hypoplasia demands the determination of the size limits of normality for the various parts of the thoracic aorta. Moulaert and associates [9] published normal values based on autopsy specimens from the Laboratory of Anatomy and Embryology of Leiden University (Fig 1). They found the diameter of the "normal" distal aortic arch to be *at least* 50% of the diameter of the ascending aorta measured at a prescribed distance proximal to the origin of the innominate artery.

Using two-dimensional echocardiograms to study various portions of the thoracic aorta and pulmonary artery in both neonates without cardiovascular disease and neonates with coarctation, Morrow and co-workers [10] found significant differences between the control group and the coarctation group in the diameter of the transverse arch. These measurements were standardized by the use of comparative ratios. The diameter of the transverse arch divided by the diameter of the ascending aorta was significantly larger in the control subjects (0.76) than in the neonates with coarctation (0.54). Although the control values are somewhat larger than the same ratios calculated from the normal values published by Moulaert and colleagues [9], the relative findings are consistent,

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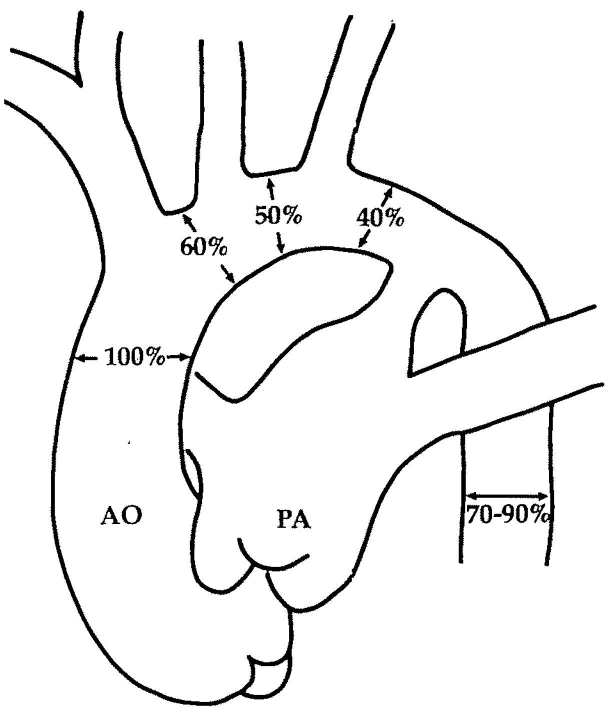

Fig 1. Minimal relative diameters of the normal thoracic aorta (AO) *in infancy.* (PA = *pulmonary artery.) (Adapted from Moulaert AJ, Bruins CC, Oppenheimer-Dekker A. Anomalies of the aortic arch and ventricular septal defects. Circulation 1976;53:1101–5, by permission.)*

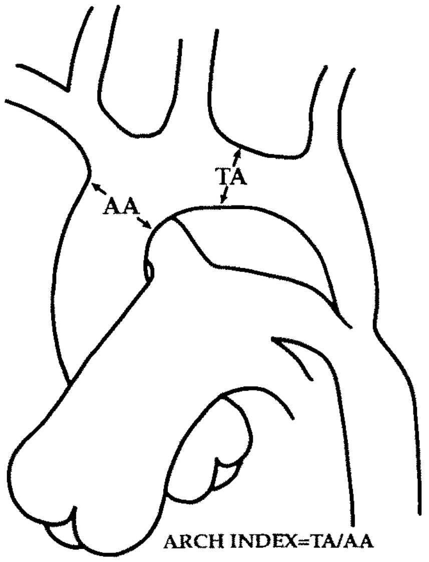

Fig 2. Aortic arch index, an adaptation of concepts of normality and methods of comparison of vessel size used by Morrow [10], Moulaert [9], and their colleagues. (AA = *ascending aorta;* TA = *transverse arch.)*

and the differences may be explained by the vastly different methods of study.

It is useful to standardize various descriptive categories of aortic arch hypoplasia (mild, moderate, severe) by means of the transverse aortic arch to ascending aorta diameter ratios, which we call the arch index (Fig 2). Transverse aortic arch hypoplasia can thus be defined as an arch index of less than 0.5.

Patient Population

During the 10-year period between December 1978 and December 1988, 229 patients underwent coarctation repair at Children's Hospital of Pittsburgh. One hundred twenty-nine (56%) were less than 12 months old at the time of repair, and 95 (41%) were less than 2 months of age. Of the 129 patients less than 12 months of age, 102 had preoperative cineangiograms that were suitable for retrospective review and determination of the comparative sizes of the ascending aorta and transverse aortic arch. These 102 infants constitute the study group (Table 1).

The determination of transverse arch hypoplasia and calculation of the arch index was conducted in a blinded fashion by two members (J.A.E. and E.P.) of the cardiology staff at our institution. Hypoplasia was noted when the transverse aortic arch diameter divided by the ascending aorta diameter yielded a ratio of less than 0.5 (see Fig 2). Angiographic images were used for determination of aortic size in 94 of the 102 preoperative determinations. The other 8 patients did not undergo preoperative catheterization but had adequate cross-sectional echocardiographic measurements to allow calculation of the arch index.

The operative procedures employed were subclavian flap aortoplasty (SFA) in 72 infants and resection and end-to-end anastomosis without any attempt to enlarge the transverse arch in 25 infants. A small group of 5 infants required other procedures, which included one reverse subclavian flap, three resections with an associated subclavian flap, and one bypass graft.

The presence of other cardiac anomalies was noted in each patient. Major anomalies including ventricular septal defect, double-outlet right ventricle, various forms of

Table 1. Mortality and Recoarctation in Infants After Coarctation Repair Relative to Presence of Major Associated Cardiac Anomalies and Hypoplasia of Transverse Aortic Arch[a]

		Mortality		Recoarctation[c]	
Variable	No. of Patients	Early[b]	Late	≥10 mm Hg	≥ 20 mm Hg
Major cardiac anomaly					
Present	62 (61)	9 (15)	14 (23)	6 (10)	3 (5)
Absent	40 (39)	1 (3)	1 (3)	3 (8)	2 (5)
p Value		0.06	0.01	NS	NS
Transverse arch					
Hypoplastic	33 (32)	3 (9)	12 (36)	5 (15)	3 (9)
Normal	69 (68)	7 (10)	3 (4)	4 (6)	2 (3)
p Value		NS	0.001	NS	NS

[a] Numbers in parentheses are percentages. [b] Early mortality = death within 30 days after operative repair. [c] Recoarctation = presence of specified systolic blood pressure difference between resting upper and lower extremity blood pressure measured at last follow-up.

NS = not significant: $p \geq 0.10$ by Fisher's exact test.

Table 2. Aortic Arch Index Before and After Standard Correction of Coarctation in Infancy

Patient No.	Age at Operation (mo)	Operative Procedure	Transverse Aortic Arch Index			Arm–Leg Blood Pressure Gradient (mm Hg)	Last Follow-up (mo postop)
			Preop	Postop	Change		
1	0.1	REEA	0.29	0.74	0.45	5	122
2	3.4	REEA	0.41	0.76	0.35	0	129
3	3.8	REEA	0.36	0.46	0.10	10	121
4	0.3	REEA	0.40	0.61	0.21	10	98
5	0.5	SFA	0.35	0.57	0.22	0	17
6	0.4	SFA	0.33	0.90	0.57	18	86
7	2.6	SFA	0.43	0.54	0.11	0	26
8	0.2	SFA	0.42	0.60	0.18	0	26
9	0.7	SFA	0.40	0.75	0.35	0	65
10	0.4	REEA	0.47	0.61	0.14	0	53
11	2.0	SFA	0.45	0.87	0.42	0	20
12	0.1	SFA	0.44	0.84	0.40	0	36
13	0.6	SFA	0.43	0.63	0.20	0	27
14	2.9	SFA	0.45	0.71	0.26	0	20
15	0.4	SFA	0.43	0.79	0.36	16	19
16	1.3	SFA	0.35	0.64	0.29	16	22
17	0.1	SFA	0.43	0.64	0.21	6	22
18	0.7	SFA	0.40	0.70	0.30	2	28
Mean	1.1		0.40	0.69	0.28[a]		55
SD	±1.2						±40
SE			0.01	0.03	0.03		
95% CL			0.38–0.42	0.63–0.75	0.22–0.34		

[a] $p < 0.001$.

CL = confidence limits; REEA = resection and end-to-end anastomosis; SD = standard deviation; SE = standard error; SFA = subclavian flap aortoplasty.

single ventricle, transposition with ventricular septal defect, and other complex anomalies were found in 62 (61%) of the infants. The presence of a bicuspid or stenotic aortic valve, an interatrial communication, or a patent arterial duct was regarded as a minor associated anomaly.

Of the 102 infants in the study group, there were 92 who survived more than 30 days after repair and from whom reliable postoperative resting arm and leg blood pressure data were obtained. The pressure gradients recorded were those measured at the most recent visit. Follow-up was available for all 92 survivors, with a mean follow-up of 50 ± 37 months (± standard deviation).

Of the 29 infants operated on during the study period who preoperatively had a hypoplastic transverse aortic arch and who survived longer than 12 months after repair, 18 had one or more satisfactory postoperative studies that allowed calculation of the postrepair arch index (Table 2). This provided an individual patient comparison of the preoperative and the postoperative arch index. These measurements were also made in a blinded fashion from existing studies, seven from angiography and 11 from one or more cross-sectional echocardiographic studies, a mean of 39 ± 29 months postoperatively.

Statistical Analysis

Statistical analysis of the data was by the Fisher exact test for group comparisons and Student's *t* test for the differences in measured values of the preoperative and postoperative aortic arch indices. When indicated, 95% confidence limits were calculated by ±2 × the standard error.

Results

Thirty-three (32%) of the 102 infants had a hypoplastic arch (an arch index of less than 0.5) (see Table 1). Of these 33 infants, 27 (82%) had major associated cardiac anomalies, and 6 did not. A normal transverse arch diameter (arch index of 0.5 or greater) was present in 69 of the 102 infants, half of whom had no major associated cardiac anomalies. This difference is highly significant ($p = 0.002$). Although the early (30-day) mortality was slightly greater for the infants with major associated anomalies than for those without these anomalies ($p = 0.06$), there was no difference in early survival between infants with and infants without arch hypoplasia. There was a significantly increased late mortality for the infants with major associated anomalies ($p = 0.01$) and infants with hypoplastic arches ($p < 0.001$) (see Table 1). However, of the 12 late deaths in the hypoplastic arch group, 11 were ascribed to complex associated cardiac malformations. There was no evidence that the arch hypoplasia itself in any way contributed to the late deaths.

Of the 92 infants (90%) who survived longer than 30 days after coarctation repair, 5 (5%) have required an additional procedure to relieve restenosis or have been found to have a resting arm–leg blood pressure gradient of 20 mm Hg or greater. However, 14 (15%) of the 92 patients met the more strict criterion of a resting arm–leg gradient of 10 mm Hg or greater to connote persistent or recurrent stenosis. There was no significant difference in the restenosis rate at either the 10- or 20-mm Hg gradient level between the 30 early survivors with hypoplastic arches and the 62 early survivors with a normal arch (see Table 1).

There were no significant differences in survival or restenosis on the basis of type of operation used for the coarctation repair in either the normal arch or the hypoplastic arch group. In another study (unpublished data), we found that in our institution, SFA when used in infants between 2 and 12 months of age has a significantly lower restenosis rate, as evidenced by resting cuff blood pressure measurements in the right arm and legs, than does repair by resection and end-to-end anastomosis, irrespective of the size of the transverse aortic arch. In this study, the 3 patients who had SFA repair and had postrepair resting arm–leg blood pressure gradients greater than 10 mm Hg underwent operation when they were less than 6 weeks of age (see Table 2).

The mean prerepair arch index in the 18 patients for whom a preoperative and postoperative arch index comparison was available was 0.40 (95% confidence limits, 0.38 to 0.42). The mean postrepair arch index was 0.69 (95% confidence limits, 0.63 to 0.75) when measured a mean of 39 ± 29 months after repair. The mean increase in arch index of 0.28 (95% confidence limits, 0.22 to 0.34) represents a highly significant change ($p = 0.001$). There was no relationship between the type of operation and the postoperative change in arch index. There was also no difference in the observed degree of change in the arch index and the age at which the repair was done. The mean age at operation for the 18 patients was 1.1 ± 1.2 months with a range of 0.1 month to 3.8 months.

In this group of 18 patients, 1 had an arm–leg gradient of 18 mm Hg at 86 months after repair, 2 had gradients of 16 mm Hg at 19 and 22 months after repair, and 2 had gradients of 10 mm Hg at 98 and 121 months postoperatively. Ten have no arm–leg pressure gradient, and the remaining 3 have gradients of 2 to 6 mm Hg. The 3 infants with postrepair pressure gradients of 16 to 18 mm Hg all experienced excellent arch growth with postrepair arch indices of 0.64, 0.79, and 0.90. Their pressure gradients are most likely secondary to failure to achieve long-lasting relief of the discrete coarctation stenosis. These 3 infants all underwent SFA repairs at 0.4 month, 0.4 month, and 1.3 months of age.

Comment

Hypoplasia of various portions of the aortic arch can be severe enough to produce obstruction to blood flow and cause measurable pressure gradients [11]. Such examples of arch hypoplasia can coexist with discrete coarctation of the aorta.

Several investigators [11–17] have questioned the growth potential of a hypoplastic transverse aortic arch and have advocated more extensive operative procedures designed to surgically enlarge the arch segment in question. Others [18–21], however, who have studied infant coarctation have not identified the small transverse arch as an isolated contributor to persistent or recurrent arm–leg pressure gradients. These authors imply that correction of the coarctation stenosis alone by well-established procedures without extending the repair into the arch is adequate. They suggest that conventional repair that allows increased forward blood flow across the distal arch into the descending aorta and perhaps the left subclavian artery will provide appropriate inducement to arch growth, especially when the operation is performed early in infancy.

It is extraordinarily unusual to find obstruction to blood flow within an underdeveloped transverse arch sufficient to produce a documented blood pressure gradient between the ascending and descending aorta in patients who have had a prior coarctation repair during infancy. At our institution, we have not seen arch hypoplasia that produced persistent blood pressure gradients between the ascending and descending aorta in children who, during infancy, required coarctation repair by standard resection and anastomosis or by SFA. Review of several reported series [22–24] of children with recurrent coarctation reveals that no incidence of the "recurrence" was found to be due to hypoplasia of the transverse arch. Most of the patients included in these reviews were suspected to have recurrence by virtue of arm–leg blood pressure gradients after coarctation repair. On subsequent invasive investigations, no recurrence was, in fact, due to a persistently hypoplastic and flow-restrictive transverse aortic arch.

With the reported incidence of arch hypoplasia as high as 25% to 50% (32% in our series) in infants with coarctation, a substantial group of patients having coarctation repair could be expected to experience failure on the basis of unacceptable arm–leg blood pressure gradients (recoarctation) when conventional resections or subclavian flap repairs have been done. An impressive number of these failures, one might suspect, would be not on the basis of persistence or recurrence at the coarctation site but on the basis of the originally diagnosed hypoplastic aortic arch. This simply is not the case, neither in the present series nor in other long-term follow-up reports of infant coarctation repair [18, 20, 21].

We are aware of the patient reported by Kopf and associates [25] who had subaortic stenosis, single ventricle, and transposition of the great vessels and underwent SFA and pulmonary artery banding at 8 days of age. The child died at 2½ years of age with a persistent hypoplastic arch and stenotic isthmus despite balloon dilation of the isthmus. Death was related to the subaortic stenosis, but Kopf and associates thought that the failure of the arch to grow contributed to the poor outcome. The failure of the transverse arch to grow may, however, have been related to the complex intracardiac anomaly and the persistent isthmic narrowing, both of which would have contributed to a chronic low cardiac output state and poor growth of the aortic segments.

Some may contend that infants with a hypoplastic transverse arch who receive conventional coarctation repair have a higher early mortality and that extended arch enlargement may improve survival. The reports [13, 17] recommending extended arch enlargement have not provided data to support this view. We found no difference in early survival between patients with and patients without arch hypoplasia in our series.

We recognize that there are infants with symptomatic coarctation and associated complex cardiac disease who have extreme degrees of aortic arch hypoplasia with arch indices of 0.25 or less. These infants who are dependent on ductal blood flow for lower body perfusion, as many neonates with coarctation of the aorta are, have functional atresia of the arch segment between the left carotid and left subclavian arteries. Their operative management is more akin to that of infants with arch interruption or arch atresia. These infants represent an extremely small minority of patients with symptomatic coarctation, and they may require some type of extended arch repair incorporated with the coarctation repair, as advocated by Lansman and colleagues [14], Elliott [12], Vincent and coworkers [15], and others.

In the group of patients reported in this series, the smallest preoperative arch index was 0.29. This infant, patient 1, had excellent arch growth after simple coarctation excision. In the group of 18 patients whose preoperative and postoperative arch indices could be compared, there was no relationship between the severity of arch hypoplasia and the tendency for growth after coarctation repair as determined by the postoperative arch index (see Table 2). There was no difference in the growth in arch index between the 13 patients operated on before 8 weeks of age and the 5 operated on between 2 and 3.8 months of age.

On the basis of the patients in this study and our interpretations of the reports of those who advocate aggressive surgical enlargement of the hypoplastic aortic arch at the time of infant coarctation repair, we believe there is no justification for this more radical operation in the great majority of infants with coarctation and hypoplasia of the aortic arch. We recognize that these extended operations can be helpful in highly selected patients. However, the majority of infants with coarctation and transverse aortic arch hypoplasia, currently defined as a transverse arch diameter less than half the diameter of the ascending aorta, do not require any surgical intervention designed to enlarge the transverse arch.

Because all techniques designed to enlarge the transverse aortic arch and relieve the coarctation stenosis can be expected to carry some degree of increased morbidity and a higher operative mortality, they should be reserved for infants with extreme degrees of arch hypoplasia, perhaps with arch indices of 0.25 or less. Under these circumstances, a more extensive operation may be appropriate in this very unusual subset of infants with coarctation. The more frequently seen infants with aortic coarctation and arch hypoplasia with an arch index of 0.3 to 0.5 do not need surgical arch enlargement.

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COMMENTARY: Indications for Extended Aortic Arch Reconstruction

We congratulate Dr Siewers for his interesting and clarifying work on this controversial pathology. Our experience with coarctation and transverse arch hypoplasia comprises 66 neonates who underwent aortic arch reconstruction [1].

Management of coarctation associated with transverse arch hypoplasia has continued to evolve over the last 5 years. It can no longer be approached simply in terms of surgical techniques for repair of the coarctation alone. The preoperative use of prostaglandin, the surgical strategy for the repair of associated complex lesions, and the increasing development of one-stage repairs through a sternotomy are also involved.

Tubular hypoplasia of the transverse aortic arch has been extensively described by pathologists and cardiologists. According to the definitions of arch hypoplasia of Moulaert and associates [2], there are three types of hypoplasia: type I, hypoplasia of the distal arch (82%); type II, hypoplasia of the entire arch (13%); and type III, absence of the proximal arch (5%).

Coarctation with severe aortic arch hypoplasia is a ductus-dependent cardiopathy. Spontaneous closure of the ductus usually causes severe deterioration in clinical status, with impaired left ventricular function, absence of femoral pulses, and acidosis. Infusion of prostaglandin may dramatically improve the situation, permitting safe operation on an infant with restored ventricular function and femoral pulses. Survival beyond the age of 1 month is unlikely in patients with severe transverse arch hypoplasia. Since the introduction of prostaglandin, a greater number of these neonates may survive until the operation.

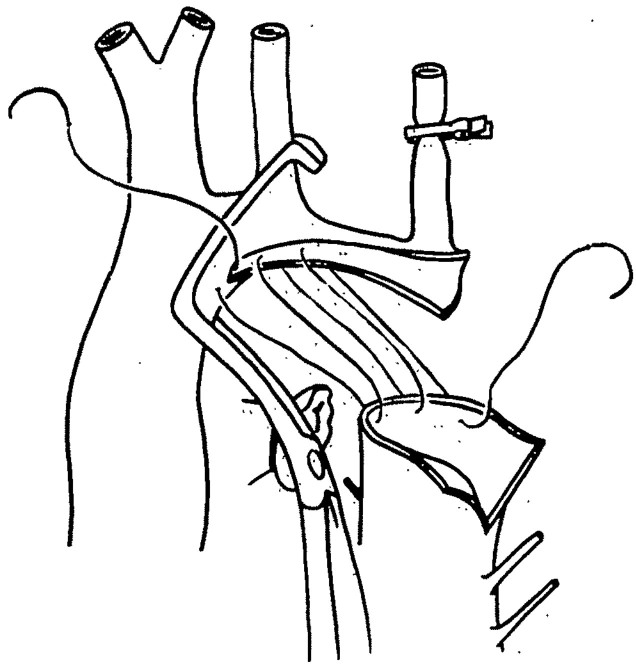
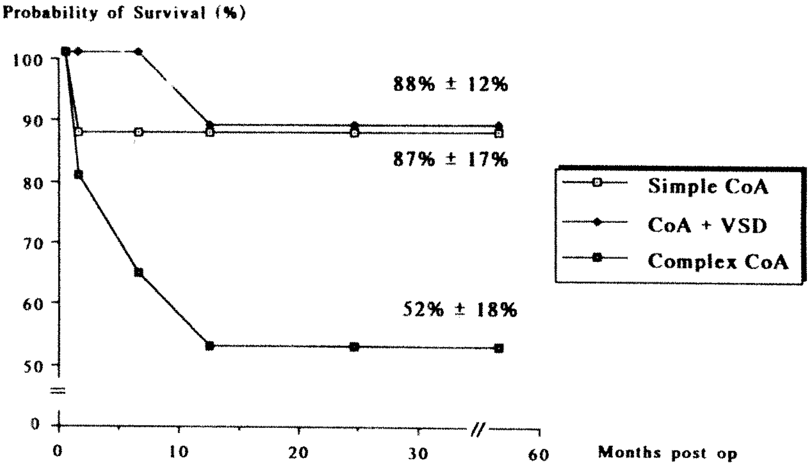


Fig 1. Repair of coarctation with hypoplastic transverse arch. All abnormal tissue has been resected, the descending aorta mobilized, and the underside of the hypoplastic aortic arch incised back to the normal-sized ascending aorta to completely relieve all areas of obstruction in the aortic arch. (Reprinted from Lacour-Gayet F, Bruniaux J, Serraf A, et al. Hypoplastic transverse arch and coarctation in neonates. J Thorac Cardiovasc Surg 1990;100:808, by permission.)

Coarctation and severe aortic arch hypoplasia is currently associated with intracardiac lesions in 78% of patients, half with ventricular septal defect and half with complex lesions including transposition of the great arteries or double-outlet right ventricle with ventricular septal defect, complete atrioventricular canal, and hypoplastic left ventricle. Concomitant or subsequent repair of an associated "simple" ventricular septal defect can be carried out with low mortality. On the other hand, coarctation associated with complex intracardiac lesions represents a challenging repair in which a reconstructed normal arch is an important factor of success, as it is in operations for hypoplastic left heart syndrome.

The surgical technique of extended aortic arch reconstruction through a thoracotomy or sternotomy has been well described (Fig 1). The repair is performed without any foreign material, the elasticity of the neonatal tissue allowing easy mobilization of the descending aorta, even in the most severe types of hypoplasia (types II and III). The operation can be hazardous through a thoracotomy,

Fig 2. Survival among 66 neonates with coarctation (CoA) with hypoplastic arch. Overall early mortality rate was 14%. The mortality rate for patients with complex coarctation includes deaths at subsequent intracardiac repair if they occurred within 30 days of the coarctation repair, even if the coarctation repair itself went well. (VSD = ventricular septal defect.)



and total repair of the coarctation and the intracardiac lesion through an anterior approach should be considered. We used that approach four times in our series.

The overall early mortality rate in our series of 66 neonates was 14% (Fig 2). It was 13% for simple coarctation, 0% for coarctation plus ventricular septal defect, and 28% for complex coarctation (including four deaths within the first postoperative month at a concomitant or subsequent repair of the associated intracardiac anomaly). The overall combined early and late mortality was 23%. By group, it was 14% for simple coarctation, 8% for coarctation plus ventricular septal defect, and 44% for complex coarctation. All late deaths were related to repair of the associated intracardiac anomaly. Five patients have required reoperation for recurrent coarctation. Reoperation was performed without any foreign material and without any deaths. Freedom from reoperation for recurrent coarctation among all survivors was 89% at 5 years. We believe this low incidence of residual or recurrent obstruction justifies continued and possibly expanded use of extended aortic arch reconstruction.

Neonates with coarctation and hypoplastic transverse arch represent a high-risk group. Preoperative improvement by reopening the ductus with prostaglandin E_1 infusions is useful in this ductus-dependent cardiopathy. The overall mortality rate in the group with complex coarctation remains elevated (44%), but death was associated with repair of the associated intracardiac lesion, not the extended arch reconstruction. One-stage total repair through an anterior sternotomy is one alternative to reduce the mortality. The surgical technique of aortic arch reconstruction that was developed in this series is also suitable for the anterior approach and requires no foreign material. Safe development of this type of repair might be the real surgical challenge of neonatal coarctation.

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Role of Extended Aortoplasty Related to the Definition of Coarctation of the Aorta

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One hundred thirty-nine patients underwent operation for coarctation of the aorta. Age ranged from 1 day to 21 years and weight, from 1.5 to 70.4 kg. Numerous methods of repair were used. The operative mortality was low (1.3%), and 17 patients (11.3%) died late. Recoarctation occurred in 13 patients (9.4%). We attempted to correlate mortality and recoarctation with the surgical procedure. A review of the literature revealed no classifications of coarctation that applied to the anatomical and pathological variations we found at the time of operation. Therefore, we devised a surgical classification to separate the various entities in the spectrum of coarctation: type I = primary coarctation; type II = coarctation with isthmus hypoplasia; and type III = coarctation with tubular hypoplasia involving the isthmus and segment between the left carotid and left subclavian arteries. Each of these types has subtypes: A = with ventricular septal defect and B = with other major cardiac defects. We believe that rather than labeling one procedure as "the procedure of choice," providing this classification will allow the surgeon to use a method of repair that is suited to the anatomical variation.

(Ann Thorac Surg 1991;52:615–20)

Coarctation of the aorta remains controversial not only in its etiology but also in regard to the optimal method of repair. This condition, especially in neonates, remains an enigma because of the high rates of recoarctation and mortality. Thus, a multitude of techniques including end-to-end anastomosis, subclavian flap turndown, subclavian angioplasty, and extended end-to-end anastomosis have been compared. It is our belief that the anatomical classification of coarctation, presented to the surgeon in this spectrum of pathological entities, has not been adequately addressed. The purpose of this report is threefold: to present a surgical classification of the coarctation spectrum; to review our data on coarctation repair and discuss the correlation of recoarctation and anatomical operative findings; and to discuss indications for the various operative techniques and some disadvantages of each method.

Material and Methods

From January 1979 to September 1990, there were 151 procedures performed on 139 patients, 85 male and 54 female. Age ranged from 1 day to 21 years and weight, from 1.5 kg to 70.4 kg. Of the 139 patients, 88 had no associated anomalies, 27 had ventricular septal defect, and 24 had other major cardiac defects. Patent ductus arteriosus and atrial septal defect were not considered associated anomalies.

Our series of 139 patients comprised 66 newborns. Twenty-four of them were seen with only coarctation, 21 had coarctation with associated ventricular septal defect, and 21 had coarctation with other associated complex defects. Therefore, 42 (63.6%) of the 66 neonates had associated anomalies. Of the 151 procedures performed in the overall series, 43.7% (66/151) were on neonates less than 1 month of age, 53.6% (81/151) were on infants less than 3 months of age, and 60.3% (91/151) were on infants less than 1 year of age (Fig 1).

The surgical repair included multiple techniques (Fig 2). The end-to-end repair, as first described by Crafoord and by Gross, was performed on 27 patients (17.9%). The subclavian flap turndown repair, introduced by Waldhausen in 1966, was done on 79 patients (52.3%) and the subclavian flap turnup, on 4 (2.6%). An interposition graft for replacement of the entire coarctation segment was used in 3 adolescents (2.0%). Enlargement of the coarctate segment was accomplished with a patch, as described by Vosschulte in 1957, of either Dacron or polytetrafluoroethylene in 23 patients (15.2%) or with a free segment of subclavian artery in 3 (2.0%). The technique of subclavian reimplantation, described by de Mendonca and colleagues [1] in 1985, was used in 4 patients (2.6%), 3 newborns and 1 2-year-old child. Other angioplasties performed in conjunction with an end-to-end anastomosis included subclavian flap turnup in 6 (4.0%), subclavian flap turndown in 1 (0.7%), and subclavian reimplantation in 1 (0.7%). This last angioplasty procedure was presented

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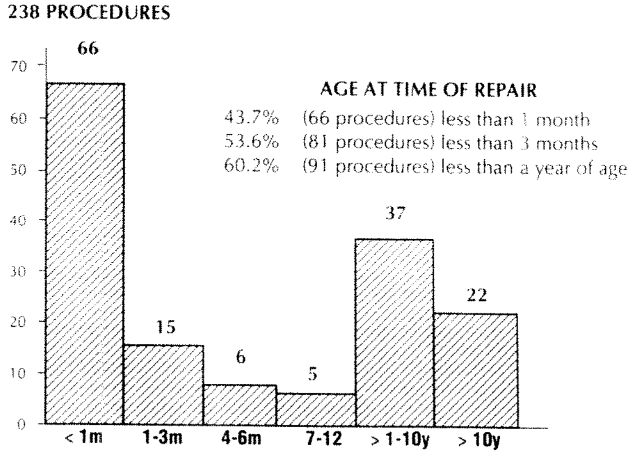

Fig 1. Age of the 151 patients at the time of surgical repair.

by one of us (J.J.A.) in 1977 [2]. The age group for each of these repairs is shown in Table 1.

The suture material used for the anastomoses beginning in 1979 was 5-0 or 6-0 Prolene (Ethicon, Somerville, NJ). In 1986, we converted to monofilament absorbable suture. At first we used polydioxanone (PDS; Ethicon), but technical difficulty with handling the memory of the suture and inability to tighten the suture line led to the use of polyglyconate (Maxon; Davis + Geck, Danbury, CT) in 1988. Continuous suture technique was used in all anastomoses.

Results

Mortality

The operative mortality rate was 1.3% (2 patients/151 procedures). One of the 2 patients who died was a newborn not considered to be in the classification of hypoplastic left heart syndrome, although a hypoplastic aortic arch, unrecognized severe aortic stenosis, and mild hypoplasia of the left ventricle were present. This patient died of low cardiac output on the night of operation. The other patient who died was a newborn in whom coarctation of a right-sided aortic arch was diagnosed preoperatively. At postmortem examination, an associated vascular ring was discovered. During the end-to-end anastomosis, the patient became acidotic because of poor ventilation and died in the operating room.

The late mortality rate was 11.3% (17 patients/151 procedures). Eight patients (5.3%) died at a later operative correction. Causes of death not related to the initial operation in the other 9 patients (6.0%) included sepsis (4), meningitis (1), biliary infection (1), and respiratory problems (2).

Recoarctation

Recoarctation, by definition, is recurrence of coarctation or restenosis. Some authors [3] have stated that recoarctation is defined as a resting gradient of 10 mm Hg or more between the arm and the leg, but others have chosen a gradient of 20 mm Hg [4] or even 30 mm Hg [5]. An exact clinical determination of the gradient is difficult. At Schneider Children's Hospital, recoarctation has been

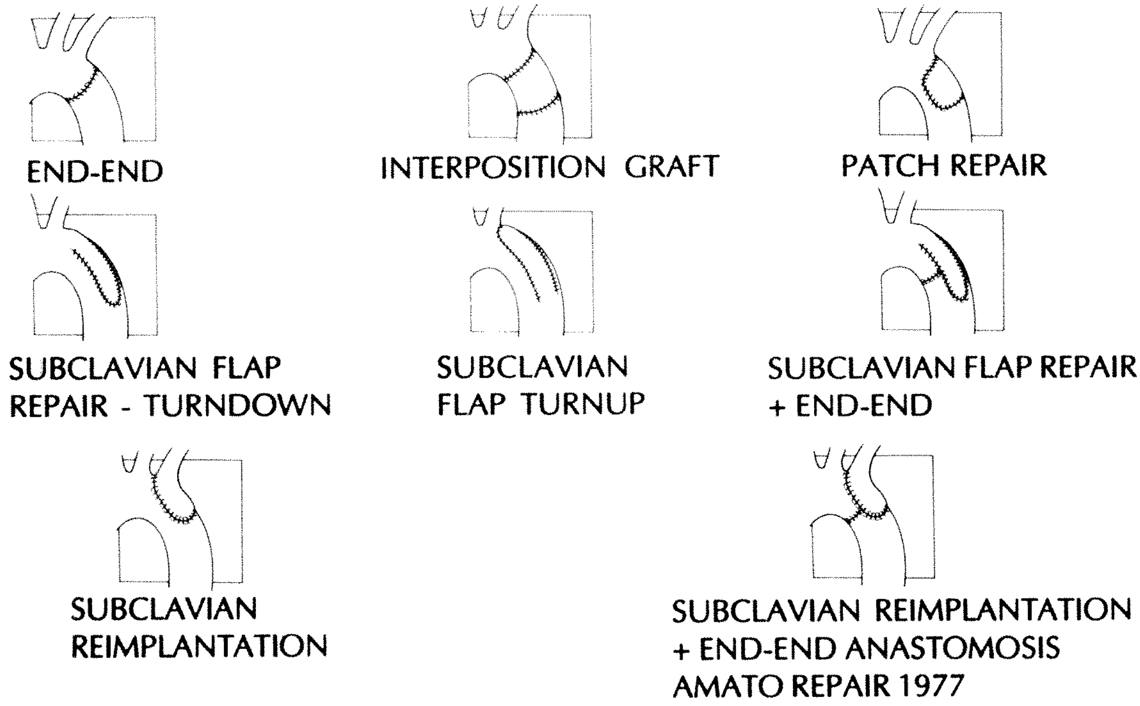
END-END
INTERPOSITION GRAFT
PATCH REPAIR
SUBCLAVIAN FLAP REPAIR - TURNDOWN
SUBCLAVIAN FLAP TURNUP
SUBCLAVIAN FLAP REPAIR + END-END
SUBCLAVIAN REIMPLANTATION
SUBCLAVIAN REIMPLANTATION + END-END ANASTOMOSIS AMATO REPAIR 1977

Fig 2. Methods of repairing coarctation of the aorta at Schneider Children's Hospital.

Table 1. Types of Repair for Coarctation of the Aorta[a]

	Repair									
Age	E-E	SFTD	SFTU	Patch	IG	SFTU + E-E	SR	SR + E-E	SFTD + E-E	Total
<1 Month	7	44	2	3	0	5	3	1	1	66
1–3 Months	2	8	2	2	0	1	0	0	0	15
4–6 Months	3	2	0	1	0	0	0	0	0	6
7–12 Months	0	4	0	1	0	0	0	0	0	5
>1–10 Years	12	15	0	9	0	0	1	0	0	37
>10 Years	3	6	0	10	3	0	0	0	0	22
Total	27	79	4	26	3	6	4	1	1	151
	(17.9)	(52.3)	(2.6)	(17.2)	(2.0)	(4.0)	(2.6)	(0.7)	(0.7)	(100)

[a] Numbers in parentheses are percentages.

E-T = end-to-end anastomosis; IG = interposition graft; SFTD = subclavian flap turndown; SFTU = subclavian flap turnup; SR = subclavian reimplantation.

defined as a gradient of 20 mm Hg or greater between the right arm and the legs.

Thirteen (9.4%) of the 139 patients in this series had recoarctation (Table 2). When we analyzed recoarctation by type of repair (Table 3), only 1 of the 27 patients with end-to-end anastomosis had recurrence of coarctation. Five of the 79 patients with subclavian flap turndown needed a second procedure. In 3 patients whose anatomy did not lend itself to a subclavian flap turndown, a free patch graft of subclavian artery was used. There was recoarctation in 1 patient. Six patients had a long hypoplastic segment between the subclavian and left carotid arteries for which a subclavian turnup with end-to-end anastomosis was performed; 2 (33%) had recoarctation. The most devastating results were seen with the subclavian reimplantation technique; 3 (75%) of the 4 patients had restenosis. The 3 affected were newborns, whereas the fourth was a 2-year-old child who did well. Of the 4 patients having a subclavian flap turnup, 1 infant (25%) had recoarctation. No recoarctation occurred in the 23 patients having patch repair with Dacron or polytetrafluoroethylene or in the 3 with an interposition graft. No patient in the patch repair group had aneurysm formation. The 1 patient who had subclavian reimplantation with end-to-end repair, as reported in 1977 [2], did not

Table 2. Patients with Restenosis of Coarctation Repair

Patient No.	Original Repair	Age at First Repair (days)	Interval (mo)	Second Repair
1	E-E	13	8	SFTD
2	SFTD	24	20	Dacron patch graft
3	SFTD	16	15	Dacron patch graft
4	SFTD	10	14	E-E
5	SFTD	26	4	E-E
6	SFTD	15	2	E-E
7	SFTU	10	4.5	Gore-Tex patch
8	SFTU + E-E	23	2	Gore-Tex patch
9	SFTU + E-E	5	2	Dacron patch
10	Free subclavian artery patch	11	5	E-E
11	SR	9	9	SFTD
12	SR	14	2	Unknown[a]
13	SR	10	. . .	Late death

[a] This repair was done at another center.

E-E = end-to-end anastomosis; SFTD = subclavian flap turndown; SFTU = subclavian flap turnup; SR = subclavian reimplantation.

Table 3. Restenosis by Type of Repair[a]

Repair	No. of Procedures	Restenosis
E-E	27	1 (3.7)
SFTD	79	5 (6.3)
SFTU + E-E	6	2 (33.3)
Patch	23	0 (0)
SR + E-E	1	0 (0)
IG	3	0 (0)
Free subclavian graft	3	1 (33.3)
SR	4	3 (75)
SFTU	4	1 (25)
SFTD + E-E	1	0 (0)
Total procedures	151	. . .
Total patients	139	13 (9.4)

[a] Numbers in parentheses are percentages.

E-E = end-to-end anastomosis; IG = interposition graft; SFTD = subclavian flap turndown; SFTU = subclavian flap turnup; SR = subclavian reimplantation.

have recoarctation, nor did the patient who had subclavian flap turndown with end-to-end anastomosis.

Comment

Since the first repair of coarctation by Crafoord and by Gross, there has been considerable controversy about the choice of operation for coarctation, particularly in newborns. We analyzed our data to try to determine critical factors for low operative mortality and low incidence of recurrence. In the last two decades, numerous authors judged the end-to-end anastomosis to be less favorable than the Waldhausen subclavian flap turndown. Within the last 5 years, however, there has been a resurgence of favor toward the end-to-end anastomosis.

To compare results, numerous methods have been used to determine the "success" of the operations. In some reports [6, 7], patients were placed into three anatomical groups based on the presence of "pure" coarctation, coarctation with ventricular septal defect, and coarctation with associated anomalies [8, 9]. However, these groups addressed mortality statistics rather than recoarctation. Bergdahl and colleagues [10] chose to place their patients into three anatomical groups and three functional groups. The anatomical groups again were divided into pure coarctation, coarctation with ventricular septal defect, and coarctation with major anomalies. Ziemer and associates [8], after analyzing the data on patients with either end-to-end anastomosis or subclavian flap turndown repair, came to no conclusion as to the merits of either technique. Others grouped patients according to age to determine the procedure of choice: the first month [9], the first 3 months [11], the first 6 months [12], and the first year [13].

In reviewing the literature, we began to question methods that grouped patients by age or anatomy, namely, pure coarctation, coarctation with ventricular septal defect, or coarctation with other associated cardiac anomalies. We searched the literature for various classifications of coarctation of the aorta. It became apparent that, from the first description of coarctation in 1760 by Morgagni to that of Ho and Anderson [14] in 1979, the classifications were ideal to fit the needs of the anatomist, pediatrician, or pathologist. Waldman and co-workers [13] stated in 1983 that the choice of operation should be based on anatomy rather than personal preference. In 1985, Pellegrino and colleagues [15] defined an anatomicopathological study with respect to treatment. Several types of obstructive lesions, which increased in severity from a shelflike lesion in coarctation to tubular hypoplasia, were noted. The feasibility of correlating surgical repair with the anatomy was discussed, and it was stated that such a study would have value only if the information obtained was applicable to clinical practice.

It seems logical that repair of coarctation be based primarily on the anatomical variations seen in the aorta. Therefore, we composed a classification for coarctation of the aorta that allows the surgeon to choose a suitable operation based on the anatomy with which he or she is presented at the time of operation. We also determined that together with the anatomical variations, the classification should be amplified to consider the associated lesions and thus the increased risk of death. Our pro-

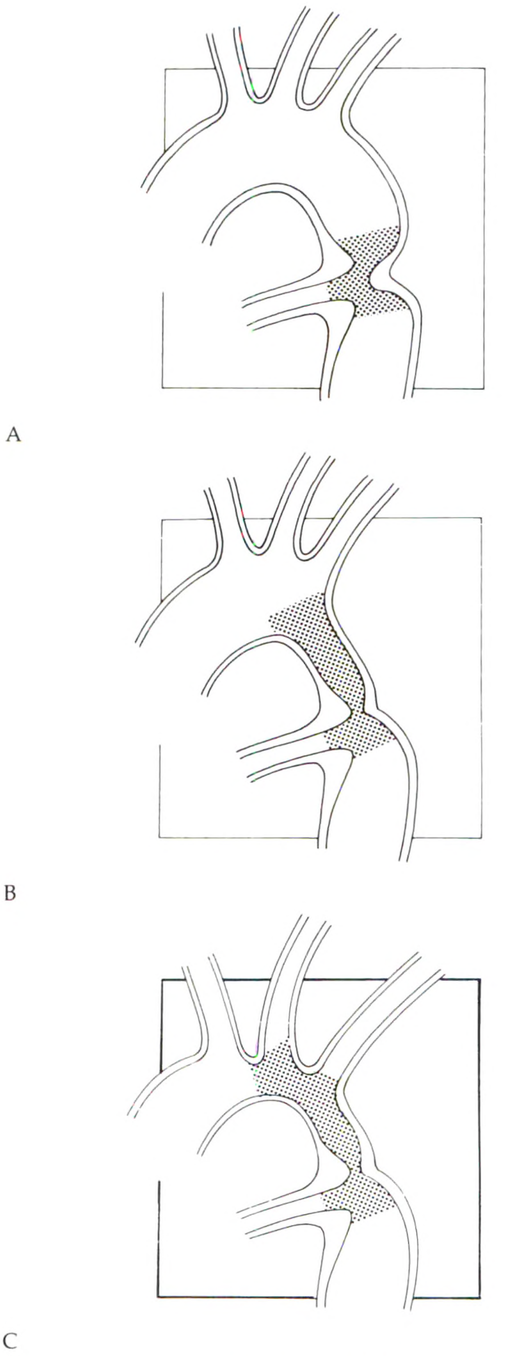

Fig 3. (A) Surgical classification of coarctation: type I = primary coarctation with or without patent ductus arteriosus with no other cardiac defects; IA = with ventricular septal defect; and IB = with other major cardiac defects; (B) type II = coarctation with isthmus hypoplasia with or without patent ductus arteriosus; IIA = with ventricular septal defect; and IIB = with other major cardiac defects; (C) type III = coarctation with tubular hypoplasia involving isthmus and segment between the left carotid and left subclavian arteries with or without patent ductus arteriosus; IIIA = with ventricular septal defect; and IIIB = with other major cardiac defects.

Table 4. Analysis of Late Deaths in Terms of Proposed Classification[a]

Classification[b]	No. of Procedures	Deaths: At Later Open Heart Operation	Deaths: Other Causes
I	35	0 (0)	0 (0)
IA	6	0 (0)	0 (0)
IB	5	1 (20)	0 (0)
II	55	0 (0)	1 (1.8)
IIA	23	1 (4.3)	3 (13.0)
IIB	16	2 (12.5)	5 (31.3)
III	6	0 (0)	0 (0)
IIIA	2	1 (50)	0 (0)
IIIB	3	3 (100)	0 (0)
Total	151	8 (5.3)	9 (6.0)

[a] Numbers in parentheses are percentages. [b] See text for explanation of classification.

posed surgical classification of coarctation of the aorta is as follows:

Type I	Primary coarctation with or without patent ductus arteriosus (Fig 3A)
IA	With ventricular septal defect
IB	With other major cardiac defects
Type II	Coarctation with isthmus hypoplasia with or without patent ductus arteriosus (Fig 3B)
IIA	With ventricular septal defect
IIB	With other major cardiac defects
Type III	Coarctation with tubular hypoplasia involving isthmus and segment between left carotid and left subclavian arteries with or without patent ductus arteriosus (Fig 3C)
IIIA	With ventricular septal defect
IIIB	With other major cardiac defects

With this classification in mind, we analyzed our 151 procedures with regard to mortality. Our early mortality of 1.3% comprised 2 patients who would be considered type IB. The classification of the 17 patients who died late is described in Table 4. We also retrospectively reviewed the cases of our patients with recoarctation in terms of our classification. The 13-day-old neonate with the end-to-end repair belonged in type II and most likely should have had a subclavian flap turndown. The 5 patients who had an initial subclavian flap turndown belonged in type II, and should have had either a longer segment of the subclavian flap turned down or, preferably, an end-to-end resection combined with subclavian flap turndown. The patient with a subclavian flap turnup and the 2 patients with a subclavian flap turnup with end-to-end anastomosis belonged in type III and, perhaps, needed a more extensive resection of the coarctation segment before the end-to-end anastomosis. We believe in the theory of Skoda as described by Wielenga and Dankmeijer [16] and advanced by others [14, 17]. The patient with a free subclavian artery graft belonged in type II and should have had a subclavian flap turndown with an end-to-end anastomosis.

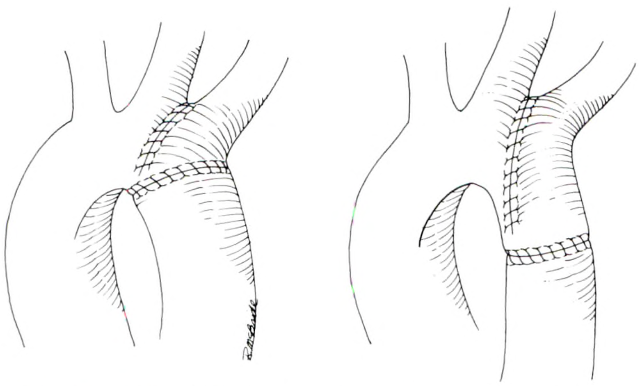

Fig 4. Comparison of (A) Amato repair of 1977 and (B) proposed Meier repair of 1987.

The 3 patients with subclavian displacement require discussion. Isthmosubclavioplasty was described by de Mendonca and associates [1] in 1985 in a case report. In 1986, Meier and co-workers [18] reported that this technique was used in 28 patients with good results, but only 2 patients were less than 3 months old. The 3 neonates in our series, who were 9, 10, and 14 days old, all had recoarctation. Other authors [19–21] have also reported recoarctation when the de Mendonca technique was used.

Meier [22] subsequently presented a modification of this technique to prevent recoarctation in newborns by suggesting end-to-end resection combined with reimplantation of the subclavian artery. This type of subclavian angioplasty was described by one of us (J.J.A.) in 1977 [2] (Fig 4); and Binet [23], in 1986, referred to this operation as the Amato technique. Several other techniques followed the principles of the Amato technique, namely, by enlarging the hypoplastic segment combined with an extended end-to-end anastomosis [24]. A similar procedure was described as a new technique by Dietl and Torres [25] in 1987. We believe that the Amato technique or its modification should be used in infants with type III coarctation with tubular hypoplasia.

Conclusion

We have presented a surgical classification for coarctation of the aorta by which the surgeon can correlate the anatomy of each patient with the operation performed. There should be no preconceived ideal or classic procedure. In many infants, especially newborns, type I—discrete coarctation—can be repaired equally well with an end-to-end anastomosis or a subclavian flap turndown or a patch repair. We do not recommend the free patch graft of subclavian artery, as the patch has a tendency to retract and cause recoarctation. Type II—coarctation with isthmus hypoplasia—can be repaired preferably and easily by a subclavian flap turndown or a subclavian flap turndown with end-to-end anastomosis; or with extended resection, it can be managed with an end-to-end anastomosis. If a patch is used, we recommend that the posterior ridge of the coarctation not be resected because this could weaken the wall and lead to aneurysm formation [26]. Type III—coarctation with tubular hypoplasia involving the isthmus and segment between the left carotid and the left subclavian arteries—is the most difficult to repair, especially as there are some [27] who believe that the hypoplastic segment might grow; there is no evidence to establish this. We agree that some hypoplastic segments will grow, but the likelihood of occurrence cannot be predicted.

As the best repair, we believe that an extended angioplasty using the left subclavian artery as part of the patch repair or the technique of resection and extended anastomosis of the transverse arch, which was derived from our technique and endorsed by others [23], could be used. We are concerned, however, about the subclavian turnup with end-to-end anastomosis because 2 of 6 patients had recoarctation. Our conclusion is that subclavian reimplantation should not be used without an end-to-end resection in newborns. This technique, however, might be feasible in children more than 1 year of age.

We propose that the anatomicopathological condition or aortic arch in each patient should dictate the method of repair. There is no one procedure that we would label "the procedure of choice."

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Role of Balloon Angioplasty in the Treatment of Aortic Coarctation

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Since the initial report of coarctation balloon angioplasty in 1982, several groups have used this technique for native coarctations in neonates, infants, and children and for postoperative recoarctations. However, recommendations for use of balloon angioplasty as a treatment procedure of choice are clouded by reports of aneurysm development at the site of coarctation. Here we review our experience as well as that published in the literature, including Valvuloplasty and Angioplasty of Congenital Anomalies Registry data, and present evidence in support of balloon angioplasty as a therapeutic procedure of choice for treating native and recurrent postoperative aortic coarctations. Balloon angioplasty of native aortic coarctations in 20 neonates and infants 1 year old or less reduced peak systolic pressure gradient across the coarctation from 40 ± 12 mm Hg (mean ± standard deviation) to 11 ± 8 mm Hg ($p < 0.001$); no patient required immediate surgical intervention. The residual gradient at follow-up (mean follow-up, 12 months) in 16 infants was 18 ± 16 mm Hg, a significant improvement ($p < 0.01$) compared with preangioplasty values. In none of the patients did an aneurysm develop. Recoarctation developed in 5 (31%) of the 16 infants and was successfully treated either by surgical resection (in 2) or by repeat balloon angioplasty (in 3). A comparison of mortality and recurrence rates between the balloon angioplasty and surgical groups was made with the help of data pooled from the literature published since 1980. The initial (7% versus 23%) and late (2% versus 25%) mortality and recoarctation (11% versus 18%) rates were higher ($p < 0.025$) after surgical intervention than after balloon therapy. When only reports in which patients were operated on after 1979 were included in this type of analysis, the initial and late mortality rates remained higher ($p < 0.01$) after operation than after angioplasty, and the recoarctation rates became similar ($p > 0.1$). Thirty-two children (>1 year old) underwent balloon angioplasty of native coarctation with a resultant reduction in peak systolic pressure gradient from 48 ± 19 mm Hg to 10 ± 9 mm Hg ($p < 0.001$), which continued to remain low (14 ± 11 mm Hg; $p < 0.001$) at follow-up catheterization in 24 children 13 months (mean) later. There were no immediate or late deaths. A small aneurysm developed in 1 patient (4%) but did not require intervention. Recoarctation developed in 2 patients (8%), and in both, repeat balloon angioplasty was performed with good results. Hypertension decreased. Comparison with pooled surgical data revealed that initial (0% versus 1.6%; $p < 0.025$) and late (0% versus 8%; $p < 0.001$) mortality rates were lower after balloon angioplasty and that rates of recoarctation (6% versus 5.5%) and aneurysm formation (12% versus 13%) were similar ($p > 0.1$). Postoperative aortic recoarctation in 10 children was treated by balloon dilation. The coarctation gradient was reduced from 52 ± 21 mm Hg to 15 ± 7 mm Hg ($p < 0.001$), which decreased to 6 ± 6 mm Hg ($p < 0.01$) at a mean follow-up of 17 months. There were no deaths, recoarctation, or aneurysm formation in this small group of patients. Compared with pooled data of repeat operation from the literature, the balloon angioplasty group had a lower mortality rate, but the recoarctation rates were similar. Although we have compared pooled surgical data with pooled balloon therapy data in all three groups presented, we recognize that there are limitations to the comparison of "older" surgical studies with "current" balloon angioplasty experience. Based on our experience and that in the literature, we surmise that coarctation balloon angioplasty is the treatment of choice for symptomatic native coarctation and recoarctation, especially in neonates and infants 1 year old or less.

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Sos and colleagues [1] demonstrated in a neonate post mortem that the coarctate aortic segment could be dilated by balloon angioplasty. Lock and associates [2–4] extended these observations and showed that surgically excised coarctate aortic segments and experimentally created coarctation in lambs and dogs could be dilated by balloon angioplasty. Since then, several short-term and a few intermediate-term results of balloon angioplasty of aortic coarctations not previously operated on ("native" coarctations) as well as postoperative recoarctations have been published. However, there is considerable controversy among cardiologists and surgeons in regard to whether the aortic coarctations should be balloon dilated or surgically treated and, if balloon angioplasty is chosen, which coarctations should be dilated.

We support the view that balloon angioplasty is an effective therapeutic alternative to surgical treatment of

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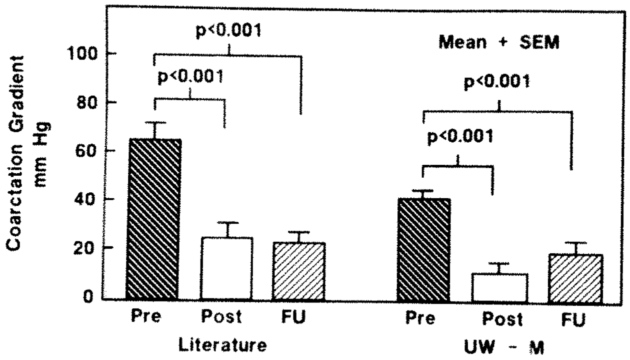

Fig 1. Peak systolic pressure gradients across aortic coarctation before balloon coarctation angioplasty (Pre), *immediately after angioplasty* (Post), *and at follow-up* (FU) *in neonates and infants* (≤*1 year old*) *for the literature group and our group* (UW − M). *Note significant* ($p < 0.001$) *decrease in gradient immediately after angioplasty in both groups. The gradients at follow-up remained low and continued to be significantly lower* ($p < 0.001$) *compared with preangioplasty values.* (SEM = *standard error of the mean.*)

aortic coarctation. Data from our experience with balloon angioplasty during the last 5½ years in 62 consecutive patients between 3 days and 13 years old for relief of native aortic coarctations (n = 52) and postoperative recoarctations (n = 10), including some patients previously reported [5–12], and the experience reported in the literature will be used as supportive material.

The description of the patients, technique of coarctation percutaneous balloon angioplasty, data gathered, and statistical analysis used have been described previously [5–7] and will not be detailed here except for three important technical aspects of the procedure. First, heparin sodium, 100 U/kg (maximum of 2,000 U), was administered immediately after the introduction of the arterial catheter. The heparin effect was neither reversed nor continued after balloon angioplasty. Second, the size of the balloon chosen for angioplasty was two times (or more) the size of the coarctate segment but no larger than the size of the descending aorta at the level of the diaphragm as measured from a frozen frame of the video recording. Third, at no time was a catheter or guidewire manipulated over the area of freshly dilated coarctation of the aorta. For the purpose of discussion, the coarctations are divided into three groups: native coarctation, usually associated with other defects, in the neonate and young infant (≤1 year old); native coarctation in children (>1 year old); and postoperative recoarctations.

Native Coarctation in the Neonate and Young Infant

Immediate Results

In our case material, 20 infants aged 3 days to 12 months (median age, 2.7 months) underwent balloon angioplasty with a resultant reduction in peak systolic pressure gradient across the coarctation from 40 ± 12 mm Hg (mean ± standard deviation) to 11 ± 8 mm Hg ($p < 0.001$) (Fig 1). Thirteen (65%) of the 20 infants had associated major cardiac defects. One infant died 2 days after balloon angioplasty and will be discussed later. All the other infants had improvement in symptoms and were discharged home within 24 to 48 hours. The condition of the infants who were in heart failure improved, and their systemic hypertension decreased. The femoral pulses, which had either been absent or markedly reduced and delayed compared with the brachial pulses, became palpable with increased pulse intensity after angioplasty.

Review of the literature (excluding our reports and the Valvuloplasty and Angioplasty of Congenital Anomalies [VACA] Registry patients [13]) revealed that 39 neonates and infants underwent coarctation balloon angioplasty; they were derived from eight reports [14–21]. Twenty-two (76%) of the 29 infants for whom data on associated anomalies were available had severe associated defects, including moderate to large ventricular septal defect, mitral valve atresia, Shone's anomaly, and subvalvar aortic stenosis. Discrete and not so discrete coarctations were dilated, but long-segment coarctations were excluded for the most part. Adequate pressure data were available for 25 infants ranging between 4 days and 12 months old with a median age of 4 weeks. Balloon angioplasty in these infants reduced the peak systolic pressure gradient across the coarctation from 64 ± 34 mm Hg to 23 ± 26 mm Hg ($p < 0.001$) (see Fig 1). Two of the 39 infants required immediate surgical intervention [14, 16]. There were three deaths [14, 16, 18], which will be discussed later. The condition of the remaining infants improved clinically, and they were discharged home. The pressure gradient reduction data in 27 neonates and infants from the VACA Registry [13] were equally impressive.

Follow-up Results

From our group of 20 neonates and infants who underwent balloon angioplasty, 16 had follow-up catheterization and angiography 12 ± 4 months after angioplasty [11]. The residual gradient was 18 ± 16 mm Hg (see Fig 1), a significant improvement compared with their preangioplasty gradient of 40 ± 12 mm Hg ($p < 0.01$). Recoarctation, defined as a gradient greater than 20 mm Hg [11], developed in 5 (31%) of these patients; 2 underwent surgical repair of residual coarctation early in our experience, and the remaining 3 underwent repeat balloon angioplasty with reduction of gradient from 30, 39, and 46 mm Hg to 0, 10, and 8 mm Hg, respectively.

Follow-up catheterization and angiographic data were available for 8 neonates and infants [17, 19, 20] from among the 39 infants in the literature group. These data were obtained 11 ± 4 months after the procedure. The residual gradient at restudy was 22 ± 10 mm Hg (see Fig 1), a significant improvement compared with the preangioplasty gradient of 50 ± 31 mm Hg ($p < 0.05$) in these 8 infants. All 39 patients had clinical follow-up, and surgical resection of coarctation was performed in 1 infant because of critical recoarctation [18]. The remaining infants seem to have done well clinically. No follow-up data were reported from the VACA Registry [13].

The high recurrence rate in infants 1 year old or younger is not too dissimilar to that seen after surgical intervention. In a previous study, our group [8] identified four factors for recurrence of aortic coarctation after balloon angioplasty: age less than 12 months; aortic isthmus less than two thirds the size of the ascending aorta; coarctate aortic segment less than 3.5 mm before angioplasty; and coarctate aortic segment less than 6 mm after dilation. We also observed that the larger the number of risk factors, the higher the chance for recurrence. The majority of the infants in this series had several of the risk factors, and therefore it is not surprising that the recoarctation rate was high. Despite this high recurrence, we believe balloon angioplasty is a worthwhile procedure because repeat intervention to relieve residual or recurrent obstruction can be safely undertaken when the infant is older and not acutely ill.

Immediate Complications

Of the 20 infants in our study group, one 11-day-old infant died 2 days after balloon angioplasty while awaiting surgical palliation of a complex cyanotic heart defect (double-inlet left ventricle with severe subaortic [bulboventricular foramen] obstruction and hypoplastic transverse aortic arch), a mortality rate of 5%. There were three deaths [14, 16, 18] among the 39 neonates and infants in the literature, giving a mortality rate of 7.7%. One infant did not improve after angioplasty and died during banding of the main pulmonary artery for associated atrioventricular canal defect [14]. Another infant with associated ventricular septal defect died after perforation of the aorta caused by manipulation of an angiographic catheter immediately after balloon angioplasty [16]. The third infant died of recurrent ventricular fibrillation on the day of balloon angioplasty [18]; whether or not this was related to transient prolongation of the QTc interval observed after balloon dilation [22] is not known. One 8-day-old infant from the VACA Registry [13] died 3 weeks after angioplasty, giving a 3.7% mortality rate. The cause of death was not reported. A 5-day-old infant required resuscitation immediately after angioplasty and was later referred for surgical ligation of patent ductus arteriosus.

Among the 20 neonates and infants in our balloon angioplasty group, 2 neonates had lost their pulse but had good perfusion of the lower extremity. The following morning, the pulse returned to normal. No immediate arterial complications were reported for the 39 neonates and infants collected from the literature [14–21]. It is not clear whether the arterial complications were absent or whether the format of the reports did not allow mention of such complications. Description of complications in the VACA Registry patients was not conducive to separating the arterial complications between the infant and children groups.

Other complications during and immediately after balloon angioplasty were remarkably minimal. Blood loss necessitating transfusion was reported in several studies.

Follow-up Complications

There was one late death in our study group, and it was related to severe mitral and aortic stenosis and moderate hypoplasia of the left atrium and left ventricle, a forme fruste hypoplastic left heart syndrome. There was one late death in the infant group from the literature [20], and no late deaths were reported for the VACA Registry patients [13].

The recommendations for use of balloon angioplasty of native aortic coarctations have been clouded by reports [19, 23–27] of development of aneurysms at the site of coarctation dilation. However, to our knowledge, aneurysms have not been reported after balloon angioplasty of native coarctation in the infant group; detailed review of patients reported to have aneurysm development [19, 23–27] revealed that none of them were infants less than 1 year old at the time of angioplasty. Also, the VACA Registry [13] did not have any children less than 4 years old in whom aneurysms developed. Although these data are encouraging in regard to lack of aneurysms in the infant group, longer-term follow-up results than are currently available must be scrutinized before declaring freedom from aneurysm development in this age group.

Comparison With Operation

Twenty-five reports published since 1980 (Appendix 1) were examined to assess the results of operation for coarctation in neonates and infants less than 1 year old [11]. The authors operated on 24 to 191 infants from 1953 to 1986. Associated major cardiac defects were present in 1,260 (68%) of 1,846 infants for whom such data were available. Operative mortality rates ranged between 0% and 50% with an overall mortality rate of 23% (451/1,940) (Table 1). The investigators followed up 18 to 144 infants and observed a 3% to 59% late mortality rate. The average late mortality rate was 25% (349/1,425 infants) during an average follow-up of 11 months to 25 years. Recurrence of coarctation ranged from 0% to 33% with an average recurrence rate of 18% (211/1,204 infants) (see Table 1).

In an attempt to have comparable time periods during which both surgical and balloon interventions were performed, we [11] examined the results for infants who underwent operation for coarctation between 1979 and 1986. Prevalence of associated major heart defects was 74% (151/204 infants), similar ($p > 0.1$) to the prevalence in the balloon angioplasty group (73%; 35/48 infants). The mortality rates and recoarctation rates in these two surgical groups (25 reports published since 1980 and five reports on surgical intervention performed between 1979 and 1986) were compared with pooled data from our study plus eight balloon angioplasty reports [14–21] dealing with infants less than 1 year old in Table 1. The mortality rates are higher for operation than for balloon angioplasty, and recoarctation rates are similar, especially when the more recent surgical results are compared with balloon angioplasty results.

In conclusion, our data and data in the literature indicate that balloon angioplasty is effective in relieving aortic obstruction in neonates and young infants with an acceptable complication rate. The recoarctation rate is high, but recoarctation can be relieved by repeat balloon angioplasty or surgical intervention when the infant is in stable condition and less acutely ill. Because of these results and

Table 1. Comparison of Mortality and Recoarctation Rates Between Surgical and Balloon Angioplasty Groups of Neonates and Infants Less than 1 Year Old

Variable	25 Surgical Reports, 1953–1989[a]	*p* Value	Balloon Angioplasty 1982–1989[b]	*p* Value	5 Coarctation Surgical Reports, 1979–1986[c]
Initial mortality	451/1,940 (23%)	<0.005	4/59 (7%)	<0.01	30/204 (15%)
Late mortality	349/1,425 (25%)	<0.005	1/55 (2%)	<0.005	26/174 (15%)
Recoarctation	221/1,204 (18%)	<0.025	6/55 (11%)	>0.1	14/174 (8%)

[a] Data were pooled from these 25 reports published in the 1980s; see Appendix 1. [b] Data were pooled from eight reports in the literature [14–21] plus one case material. [c] Data were pooled from five reports (Appendix 1: [10, 17, 22, 24, 25]) presenting results of coarctation operations performed between 1979 and 1986.

Adapted from Rao PS, Thapar MK, Galal O, Wilson AD. Follow-up results of balloon angioplasty of native recoarctation in neonates and infants. Am Heart J 1990;120:1310–4, by permission of Mosby-Year Book, Inc.

the reported high mortality and morbidity after surgical repair in neonates and young infants, we recommend balloon angioplasty as the procedure of choice for relief of symptomatic native coarctation in neonates and infants 1 year old or younger.

Native Coarctation in Children (>1 Year Old)

Immediate Results

From our case material, 32 children aged 14 months to 13 years (median age, 6.5 years) underwent balloon angioplasty of native coarctation with a resultant reduction in peak systolic pressure gradient across the aortic coarctation from 48 ± 19 mm Hg to 10 ± 9 mm Hg ($p < 0.001$) (Fig 2). No patient required immediate surgical intervention. The femoral pulses became palpable with increased pulse intensity after angioplasty, and hypertension decreased. All patients were discharged home within 24 hours after the procedure.

Review of the literature excluding our reports and VACA Registry material revealed that 99 children underwent balloon coarctation angioplasty; they were derived from nine studies [17, 19, 21, 24, 25, 27–30]. One report [26] presented the results of balloon angioplasty in 33 patients aged 2 months to 29 years, but their description did not indicate the numbers of neonates, children and adults in the study population. Adequate data on predilation and postdilation coarctation gradients were available for 43 of the 99 children. These 43 children ranged in age from 1 year to 18 years (median age, 5.8 years). The gradient across the coarctation was reduced from 52 ± 16 mm Hg to 12 ± 11 mm Hg ($p < 0.001$) after angioplasty (see Fig 2). Three reports [21, 25, 27] did not provide pressure data for individual patients but did have group data; in each of these studies there was an impressive decrease in peak systolic pressure gradient from a mean of 29, 46, and 51 mm Hg to a mean of 6, 8, and 22 mm Hg, respectively. Of all 99 children, 1 child required surgical resection on the day after balloon dilation [30]. One patient from among the 33 not categorized by age also needed surgical resection 1 month after angioplasty [26]. The remaining patients did well with reduction in hypertension and improvement in symptoms when present [17, 19, 24, 27–30].

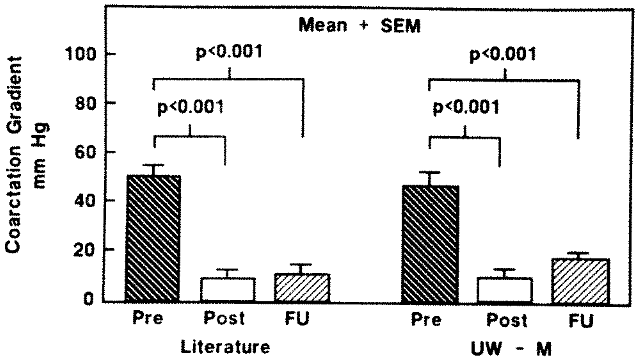

Fig 2. Peak systolic pressure gradients across aortic coarctation before balloon coarctation angioplasty (Pre), immediately after angioplasty (Post), and at follow-up (FU) in children (>1 year old) for the literature group and our group (UW − M). Note significant (p < 0.001) decrease in gradient immediately after angioplasty in both groups. The gradients at follow-up remained low and continued to be significantly lower (p < 0.001) compared with preangioplasty values. (SEM = standard error of the mean.)

From the VACA Registry [13], pressure gradient data were available for 97 children. The gradient for the entire group (including neonates and infants) decreased from 48 ± 19 mm Hg to 12 ± 11 mm Hg after balloon angioplasty.

Follow-up Results

From our study group, 24 children underwent repeat catheterization 13 ± 8 months after angioplasty and had a residual gradient of 14 ± 11 mm Hg (see Fig 2). The preangioplasty gradient in these 24 patients was 50 ± 22 mm Hg ($p < 0.001$). Repeat balloon angioplasty was required in 2 children and led to good results in both. The remaining children were asymptomatic with relief of hypertension (138 ± 27 mm Hg before angioplasty versus 110 ± 15 mm Hg after angioplasty; $p < 0.01$) on follow-up.

Follow-up catheterization and angiographic data with pressure gradient information on individual patients were available for 26 [17, 19, 24, 30] of 99 patients from the literature group. The follow-up data were obtained 13 ± 4 months after angioplasty. The residual gradient at the time of follow-up study in these 26 patients was 13 ± 11 mm Hg (see Fig 2), significantly lower ($p < 0.001$) than

Table 2. Prevalence of Recoarctation and Aneurysm Formation at Follow-up After Balloon Angioplasty of Native Coarctation in Children

Reference	Year	No. of Patients With Follow-up	Recoarctation Gradients (mm Hg) 20–30	>30	Total	Aneurysms
Lababidi et al [17]	1984	5	1	1	2	0
Allen et al [30]	1986	1	0	0	0	0
Cooper et al [24]	1987	7	0	1	1	3
Wren et al [25]	1987	14	2	0	2	1
Beekman et al [19]	1987	13	2	0	2	1
Brandt et al [27]	1987	11	1	2	3	4
Morrow et al [26]	1988	17	0	0	0	2
Suarez de Lezo et al [21]	1989	16	0	1	1	1
Rao and Chopra [this report]	1991	24	2	1	3	1
Total		108	8	6	14	13

their preangioplasty value of 47 ± 15 mm Hg. In the three studies providing only group data and comprising 44 patients [21, 25, 27], the residual gradients were low; mean values were 5.6, 11.0, and 7.6 mm Hg versus 29, 46, and 51 mm Hg before angioplasty. Adequate clinical follow-up information was available for all 99 patients [17, 19, 21, 24, 25, 27–30]. Repeat intervention for recoarctation was required in 6 children. Elective surgical intervention was performed in 4 to excise the recoarctation [19, 27], and repeat balloon angioplasty was performed in 2 to relieve the residual coarctation [25]. The other children were asymptomatic with reduced hypertension at follow-up. No follow-up data were available from the VACA Registry [13].

Immediate Complications

To our knowledge, no deaths have been reported immediately after balloon coarctation angioplasty in children; this is true for the literature group, the VACA Registry group, and our group.

Of the 32 children in our study group, 1 child required thrombectomy, and 1 had a decreased femoral pulse, which improved the following morning. Of the 127 children from the literature group [17, 19, 21, 24, 25–30], 3 required thrombectomy and 5, heparin or streptokinase therapy to improve the arterial pulse on the side of balloon angioplasty.

Other complications including balloon rupture at high inflation pressure [6, 7], cerebrovascular accident [19, 21], and hypertension with forme fruste postcoarctectomy syndrome [5, 21, 30], though rare, have been reported.

Follow-up Complications

No late deaths have been reported in children after balloon angioplasty. Data on the incidence of femoral artery occlusion at follow-up are not readily available from the literature. In our study, we [7] systematically looked at femoral artery complications at follow-up. At follow-up catheterization, arterial catheterization was performed on the side opposite that used for angioplasty in two thirds of the patients; the same side was used in the other third. In the former group, iliac arteriography was done with visualization of the femoral artery previously used for angioplasty. Arterial entry in the latter group is considered evidence of arterial patency. Of the 24 children who underwent follow-up study, 2 had femoral arteries that were found to be partially obstructed, but there was good collateral flow.

The data on recoarctation in children from the literature and from our study are listed in Table 2. Major recoarctation occurred in only 6 of 108 patients, giving a recurrence rate of approximately 6%, although the recurrence rate is much higher if the gradient cutoff is lowered to 20 mm Hg.

Aneurysms at the site of balloon angioplasty have been reported with native coarctations in children, the incidence ranging from 6% to 43% [19, 23–27]. In our material and in our review of the literature, we found aneurysms in 13 of 108 patients, giving a 12% incidence (see Table 2). None of the aneurysms required therapy nor did any aneurysm rupture, although elective resection and repair has been advocated by some authors [24, 27].

Comparison With Operation

Ten reports published since 1980 (Appendix 2) were scrutinized to examine the results of operation for aortic coarctation in children older than 1 year. A total of 1,137 children had surgical intervention from 1957 to 1985; the number of children in each report ranged between 19 and 263. The average operative mortality was 1.6% (18/1,137), although this ranged from 0% to 5%. The authors followed up 11 to 255 children for a period of 11 months to 25 years and observed a late mortality rate of 0% to 27%; the mean was 8% (60/793 children). The recoarctation rate ranged between 0.7% and 18% with an average of 5.5% (57/1,037). The mortality and recoarctation rates in the balloon angioplasty and surgical groups are compared in Figure 3. The recoarctation rates are similar, and the mortality rates are slightly higher in the surgical series.

Aneurysms occurring after balloon angioplasty [19,

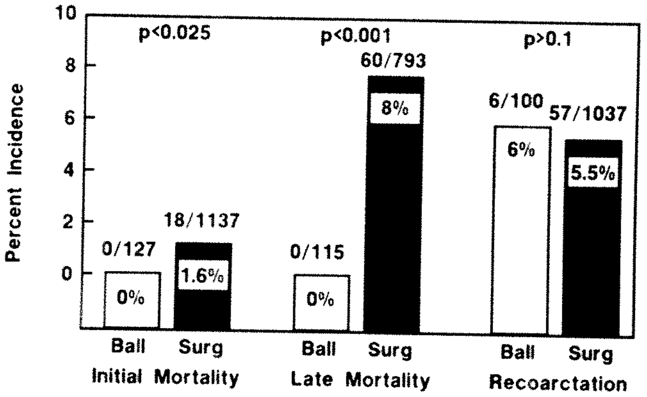

Fig 3. Comparison of initial (operative) mortality, late mortality, and recoarctation rates after balloon angioplasty (Ball) *and surgical correction* (Surg) *in children (>1 year old). The data for balloon angioplasty are derived from published literature plus our data. The data for surgical correction are derived from ten reports published in the 1980s (see Appendix 2). Note that the initial and late mortality rates are better with balloon angioplasty* ($p < 0.025$ *to* < 0.001) *than with surgical intervention, although the recurrence rates are comparable* ($p > 0.1$) *between the two groups.*

23–27], though of concern, have also been seen after surgical correction of aortic coarctation. Both false aneurysms and true aneurysms have been reported. Development of early false aneurysms appears not to be related to the type of coarctation repair but rather to the surgical technique or infection. Late aneurysms have been well documented to occur with Dacron tube grafts and onlay synthetic patch grafts. Although the exact etiology of these aneurysms is not known, compliance mismatch between the normal aortic wall and the synthetic patch, intimal resection, and translocated ductal tissue on the aortic wall have been postulated as possible causes. Review of seven studies published since 1980 (Appendix 3) revealed an incidence of aneurysm formation of 5% to 100% with a mean of 13% (67/501 patients) after synthetic patch angioplasty of aortic coarctation. The true incidence is probably on the order of 24%, as observed by Bromberg and associates [31], who determined the prevalence rate prospectively and with objective, measurable criteria. Sudden deaths secondary to aneurysm rupture have been reported. Elective surgical repair of aneurysms has been carried out with good success.

The prevalence of aneurysm formation after balloon angioplasty and Dacron patch angioplasty is similar at 12% and 13%, respectively. Occurrence of aneurysms after subclavian flap angioplasty is also well documented, although the prevalence rate is not yet determined. It must be remembered, however, that aneurysms are seen mostly with patch angioplasty and not as frequently with resection and end-to-end anastomosis.

Other complications such as paraplegia and paradoxical hypertension are seen with variable frequency after surgical repair but are either rare or, if present, very mild and inconsequential after balloon angioplasty. The rate of femoral artery occlusion may be higher with balloon angioplasty than with surgical therapy. Vascular function and growth of the left upper limb after subclavian flap aortoplasty repair of coarctation [32, 33] have been studied. The vascular function appears to be preserved, whereas definitive reduction in the length and muscle mass of the upper arm [32] and forearm [33] has been observed.

Thus, this review of comparison of surgical versus balloon therapy of aortic coarctation suggests that balloon angioplasty is no worse than surgical intervention when mortality and major complication rates are compared. Longer hospital stay and the attendant increased costs, need of intubation and general anesthesia, and residual scar are additional disadvantages with surgical intervention.

Postoperative Recoarctation

Immediate Results

Our own experience with recurrent coarctation including that reported previously [12] is limited to 10 patients. Recoarctation developed 6 months to 7 years (26 ± 25 months) after surgical repair of coarctation. Previous operations included end-to-end anastomosis after resection of coarctation in 5; patch angioplasty in 4 (Dacron, 2; subclavian flap, 2); and repair of interrupted aortic arch in 1. Surgical repair of coarctation was performed in the neonatal period in 7 patients and at 2, 10, and 18 months of age in 3. At the time of balloon angioplasty, they were 6 months to 7 years old (median age, 22 months). The peak-to-peak systolic pressure gradient across the recoarctate area decreased from 52 ± 21 mm Hg to 15 ± 7 mm Hg ($p < 0.001$) after angioplasty. As a result of angioplasty, the coarctate aortic segment increased from 3.4 ± 1.4 mm Hg to 6.1 ± 1.6 mm Hg ($p < 0.01$).

There are several reports in the literature of balloon angioplasty for recoarctation [9, 14, 16, 17, 30, 34–41]. Immediate results (excluding single case reports) were tabulated and are comparable with our results (Table 3). Thus, review of data from the literature and from our experience indicates that balloon angioplasty can be successfully performed and the aortic obstruction relieved in the vast majority of patients. Balloon angioplasty produces significant pressure gradient relief irrespective of the type of previous operation for coarctation. Data from the VACA Registry [41] suggest the pressure gradient relief is inversely proportional to the age at angioplasty, although there was considerable overlap between the groups.

Follow-up Results

Seven children from our study group had follow-up catheterization and angiography 8 to 20 months (16 ± 7 months) after balloon angioplasty. Clinical and Doppler echocardiographic follow-up data were obtained in 9 patients 8 to 23 months (17 ± 6 months) after angioplasty. Residual gradient across the dilated postsurgical recoarctation was calculated by combining the catheterization-derived peak-to-peak pressure gradient in 7 children with arm and leg cuff systolic pressure difference in the other 2 children. The residual gradient in these 9 children was 6 ±

Table 3. Immediate Results of Balloon Angioplasty of Postoperative Recoarctation[a]

Reference	Year	No. of Patients Undergoing BA	Age[b] (mo)	Types of Previous Operations	Interval Between Operation and BA (mo)	Gradient Before and After BA (mm Hg)	No. of Patients With Residual Coarctation[c]	Complications
Lock et al [14]	1983	5	90 ± 107 (1.5–264)	EE, 3 SF, 2	. . .	51 ± 19 22 ± 19	2 (40%)	Operation done within 1 week of BA, 2 patients
Kan et al [35]	1983	7	113 ± 64 (10–204)	EE, 3 TG, 2 PA, 1 IA, 1	95 ± 59 (10–198)	58 ± 12 13 ± 9	1 (14%)	Death due to ventricular fibrillation, 1 patient
Lababidi et al [17]	1984	7	121 ± 53 (36–192)	EE, 3 PA, 4	74 ± 36 (36–132)	39 ± 21 8 ± 6	1 (14%)	None reported
Allen et al [30]	1986	8	97 ± 102 (7–240)	EE, 5 SF, 2 TG, 1	. . .	51 ± 18 15 ± 10	2 (25%)	Severe hypertension, after BA, 1 patient
Hess et al [36]	1986	5	8.2 (mean) (1.5–14.3)	EE, 3 SF, 1 ST, 1	. . .	27 (mean) 12 (mean)	. . .	Balloon rupture, 3 patients
Lorber et al [37]	1986	5	4 ± 2 (3–9)	SF, 5	4 ± 2 (3–9)	59 ± 16 22 ± 16	2 (40%)	Femoral artery thrombosis, 1 patient; hypotension, 1
Saul et al [38]	1987	27	67 ± 75 (3–264)	EE, 14 SF, 7 PA, 3 NP, 3	24 (median) (3–180)	42 ± 14 14 ± 15	5 (19%)	Blood loss, 6 patients; femoral artery occlusion, 5; procedures, 3 failed
Cooper et al [39]	1989	44	288 ± 64 (2–240)	EE, 21 SF, 13 PA, 5 MO, 5	49 ± 54 (1.5–180)	37 ± 16 14 ± 11	. . .	Femoral artery occlusion, 4 patients; aneurysm, 1 patient; deaths, 2
Hellenbrand et al[d] (VACA Registry) [41]	1990	200	84 (mean) (1–312)	EE, 84 SF, 48 PA, 40 NP, 12 TG, 4 UK, 12	65 (mean) (1.5–420)	42 ± 20 13 ± 12	41 (22%)	Deaths, 5 patients; femoral artery occlusion, 17; hypertension, 4; neurological event, 3; fair to poor result, 41; balloon rupture, 19
Rao and Chopra [this report]	1991	10	29 ± 25 (6–84)	EE, 5 SF, 2 PA, 2 IA, 1	26 ± 25 (6–84)	52 ± 21 15 ± 7	2 (20%)	Blood loss, 2

[a] Single case reports were not included. [b] Data are shown as the mean ± the standard deviation with range in parentheses. [c] Residual coarctation was defined as a peak to peak gradient higher than 20 mm Hg. [d] Only mean values without the standard deviation were available.

BA = balloon angioplasty; EE = end-to-end anastomosis; IA = interrupted aortic arch repair; MO = multiple operations; NP = Norwood procedure; PA = patch angioplasty (Dacron, Gore-Tex, or pericardial patch); SF = subclavian flap; ST = subclavian artery–thoracic aorta anastomosis, end-to-side; TG = tube graft; UK = unknown; VACA = valvuloplasty and angioplasty of congenital anomalies.

Adapted from Rao PS, Wilson AD, Chopra PS. Immediate and follow-up results of balloon angioplasty of postoperative recoarctation in infants and children. Am Heart J 1990;120:1315–20, by permission of Mosby-Year Book, Inc.

6 mm Hg with a range of 0 to 18 mm Hg. The gradients averaged 52 ± 20 mm Hg before angioplasty and 16 ± 8 mm Hg immediately after angioplasty; thus there was a further decrease ($p < 0.01$) in gradient at follow-up. No aneurysms were seen in the area of previously dilated recoarctation.

The literature has a limited number of studies with a limited number of patients for whom follow-up data were recorded. The available studies are listed in Table 4. Of the 53 patients for whom follow-up data were available, 11 (21%) had major residual gradient or restenosis at follow-up. Aneurysms were found in 5 patients (9%) at follow-up. Follow-up information on a larger number of patients for a longer duration may be necessary before

Table 4. Follow-up Results of Balloon Angioplasty of Postoperative Recoarctation

Reference	Year	No. of Patients Followed Up	Duration of Follow-up^a (mo)	Follow-up Gradient^a (mm Hg)	Aneurysm	Restenosis^b	Comment
Lock et al [14]	1983	3	3.4 ± 2.9 (0.25–6.0)	15 ± 13 (5–30)	0	1 (33%)	. . .
Lababidi et al [17]	1984	4	. . .	10 ± 6 (5–18)	0	0	Duration of follow-up not given
Allen et al [30]	1986	6	7 ± 2 (4–11)	7 ± 8 (0–20)	0	1 (17%)	Femoral artery occlusion at follow-up, 1 patient
Lorber et al [37]	1986	5	12 ± 12 (2–30)	24 ± 17 (0–45)	0	4 (80%)	Repeat BA, 2 patients
Saul et al [38]	1987	5	. . .	24 ± 22 (3–50)	2 (40%)	2 (40%)	Repeat BA, 2 patients (duration of follow-up not given)
Cooper et al [39]	1989	21	12 ± 8 (2–24)	12 ± 9	3 (14%)^c	3 (14%)	Repeat BA, 3 patients
Rao and Chopra [this report]	1991	9	17 ± 6 (8–23)	6 ± 6 (0–18)	0	0	Femoral artery occlusion, 1 patient
Total		53	. . .	. . .	5 (9%)	11 (21%)	. . .

[a] When available, data are shown as the mean ± the standard deviation with range in parentheses. [b] Restenosis was defined as a peak to peak gradient higher than 20 mm Hg. [c] This includes one aneurysm that developed immediately after BA.

BA = balloon angioplasty.

Adapted from Rao PS, Wilson AD, Chopra PS. Immediate and follow-up results of balloon angioplasty of postoperative recoarctation in infants and children. Am Heart J 1990;120:1315–20, by permission of Mosby-Year Book, Inc.

surgeons can be certain of the long-term favorable effects of balloon angioplasty of postoperative recoarctation.

Complications

In the present small series, the complications were modest. In the VACA Registry [41] involving 200 patients, five deaths (2.5%) were reported after balloon angioplasty. This death rate is higher than that seen with native coarctations (0.7%, 1/141) in the VACA Registry [13], although this difference did not attain significance ($0.05 < p < 0.1$). Other major complications reported by the registry [41] include balloon rupture (9.5%), femoral artery complications (8.5%), postcoarctectomy syndrome (2%), and neurological event (1.5%).

Development of aneurysms at follow-up is another complication that needs to be addressed. The incidence of this complication appears to be 9% (see Table 4), similar to that seen after balloon angioplasty of native coarctation [9]. It has been thought that circumferential scar tissue at the recoarctation site after previous surgical repair might prevent formation of aneurysms and, if formed, prevent their rupture [36, 41–43]. Such a hypothesis may not be tenable, at least in some patients; in the VACA Registry [41], it was observed that there was little scar tissue surrounding the previous surgical repair site in a patient with aortic rupture after balloon angioplasty.

Comparison With Operation

The operative mortality rate for recoarctation after initial surgical repair of aortic coarctation is high and ranges between 3% and 33% (Appendix 4). The mortality rate after balloon angioplasty of aortic recoarctations varies between 0% and 2.5% [14, 40, 41] which compares favorably with the operative mortality after a second operation. Recoarctation rates after the second operation for postsurgical recoarctation are also high, ranging between 6% and 30%, and these recoarctation rates appear comparable with those reported for balloon angioplasty for postoperative recoarctation (21%, 11/53; see Table 4) [14, 17, 30, 37–39]. However, there are limitations to comparing the surgical data with balloon angioplasty data because of the small number of balloon angioplasty patients available for follow-up, the shorter duration of follow-up, and the possible inaccuracy of comparing "older" surgical studies with "current" balloon angioplasty studies. Nonetheless, there are advantages to balloon therapy, ie, avoidance of intubation, anesthesia, repeat thoracotomy, possible bleeding while isolating the recoarctate segment, and stay in the intensive care unit. Shorter duration of hospitalization and less expense are additional advantages of balloon angioplasty.

Conclusion

Immediate results of balloon angioplasty of native aortic coarctation and postoperative aortic recoarctation appear encouraging. Follow-up results are available on only a small number of patients and after only a short follow-up. The magnitude of the problem of aneurysms at follow-up is unknown. However, mortality and morbidity appear to be better for balloon angioplasty than for surgical treatment. Based on our experience and this review, we recommend balloon angioplasty as a procedure of choice in the treatment of native coarctation and postoperative recoarctation with critical hypertension, congestive heart failure, or both. The technique is particularly valuable in neonates and infants 1 year old or younger. Final and definitive recommendation for application of balloon angioplasty of native coarctation in children and recoarctation should probably await longer-term follow-up of a larger number of patients.

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Appendix 1. Results of Operation for Native Aortic Coarctation in Neonates and Infants: 25 Reports Published in the 1980s

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Appendix 2. Results of Operation for Native Aortic Coarctation in Children: 10 Reports Published in the 1980s

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EDITOR'S NOTE

This report presents an interesting counterpoint to the surgical papers in this supplement, but the role of balloon angioplasty in the management of native coarctation remains controversial.

It is noteworthy that the incidence of aneurysm after balloon aortoplasty is minimal in the infant group. In the group of patients greater than 1 year of age, the incidence is similar to that seen after Dacron patch aortoplasty—a procedure abandoned by most surgeons who found the incidence of this complication unacceptable. Furthermore, it is a risk that progressively increased with longer follow-up in that surgical experience. Whether a similar pattern will be seen in the balloon group will need close evaluation.

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Staged Repair of Interrupted Aortic Arch and Ventricular Septal Defect in Infancy

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Staged repair of interrupted aortic arch and ventricular septal defect was carried out in 20 infants from 1979 through 1990. Among the important associated cardiac defects were transposition of the great arteries, truncus arteriosus, and anomalous origin of the right pulmonary artery. The first stage, usually consisting of the placement of an 8- or 10-mm polytetrafluoroethylene graft, pulmonary artery banding, and ligation of the patent ductus arteriosus, resulted in 20 survivors (100%) There were two interim deaths (10%) before the second stage of ventricular septal defect closure and pulmonary artery band removal, which had 15 survivors (83%, 15/18). Because the major morbidity and mortality early in this experience could be traced to leaving the pulmonary artery band on too long, early removal (within 2 to 3 months) was begun. Since 1985, 8 (100%) of 8 infants have survived both stages and are now doing well. Because of the relatively large polytetrafluoroethylene graft, only 1 child (aged 9 years) has experienced substantial late aortic arch obstruction and undergone placement of an 18-mm Dacron graft without difficulty. Of interest is the finding that in only 1 (5%) of the 20 patients has major (≥40-mm Hg gradient) left ventricular outflow tract obstruction developed. In summary, the staged repair of interrupted aortic arch with ventricular septal defect has become very reliable despite the condition of the infant or major associated cardiac anomalies and can be recommended for infants at high risk for primary repair. More long-term information will be needed to determine which approach will be the best choice for the majority of infants.

(Ann Thorac Surg 1991;52:632–9)

The outlook has greatly improved for infants born with interrupted aortic arch (IAA), ventricular septal defect (VSD), and patent ductus arteriosus (PDA), which, until relatively recently, was usually lethal [1]. Several series [2–9; Bowman FO Jr, unpublished results] have reported survival of more than 50% of infants after operation either by a staged approach or by primary repair, and the trend is to further improvement. Complete repair in which the PDA is excised, a primary aortic anastomosis is carried out, and the VSD is closed has considerable appeal, and successful series have been reported from several institutions [5–7, 9; Bowman FO Jr, unpublished results]. On the other hand, excellent results have been reported using a staged procedure in which the arch is repaired first, usually with a conduit, and the pulmonary artery (PA) banded. Later, the VSD is closed and the PA band removed [2–4, 8].

Infants with an IAA may have a constellation of problems, and longer follow-up and evaluation will be necessary to accurately determine the advantages and disadvantages of each approach. Despite the attraction and apparent increasing success rate of primary repair, there may be a subset of infants for whom the staged approach would be the best choice. Moreover, our midterm results continue to suggest there may be advantages to this approach for the entire group. The purpose of this report is to analyze the 20 infants who have undergone staged repair at the University of Minnesota in an attempt to better define the advantages and disadvantages of this approach as well as the circumstances under which it may be preferable.

Material and Methods

Patient Population

The patient population consisted of all 20 infants who underwent staged repair of an IAA at the University of Minnesota Hospitals during the period 1979 through 1990. During this period, 2 additional infants underwent complete primary repair, and a third, older child was found to have pulmonary hypertension and uncorrectable intracardiac defects.

The condition of each patient was stabilized by infusion of prostaglandin E_1 and other standard methods of resuscitative and supportive care. In general, the older infants were in cardiac failure, and the younger infants were acidotic from severe failure and poor perfusion after ductal closure. The preoperative use of prostaglandin E_1 infusion has allowed the condition of these infants to be greatly improved, particularly when ductal closure has occurred. Lower body perfusion was restored, and acidosis and shock were corrected. Inotropic and ventilator support were often also used to further improve their condition before the infants were taken to the operating room. For the 20 infants in this series, age at operation ranged from 1 day to 39 days (median age, 4 days), and

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Table 1. Summary of Data on 20 Patients With Interrupted Aortic Arch

Variable	Value
Weight at first stage (kg)	2.4–3.8 (mean, 3.2)
Age at first stage (d)	1–39 (median, 4)
Type of IAA	
B	18
A	2
Additional major cardiac defects	
Bicuspid aortic valve	4
Truncus arteriosus	2
Transposition of great arteries	1
Anomalous origin of right pulmonary artery	1

IAA = interrupted aortic arch.

weight at operation ranged from 2.4 to 3.8 kg (mean weight, 3.2 kg) (Table 1).

Cardiac catheterization and angiography were carried out in 18 of the 20 infants and for 6 infants was the only method of diagnosis. Since 1982, all infants have been studied by echocardiography, and for 2 infants, echocardiography was the only method used to establish the diagnosis. All had VSDs and, after infusion of prostaglandin E_1, PDAs. For 18, the aortic interruption was type B and for 2, type A. A number of additional major cardiac defects were found including a bicuspid aortic valve (4 patients), truncus arteriosus (2), transposition of the great arteries (1 patient), additional muscular VSD (1), and anomalous origin of the right PA from the aorta (1) (see Table 1).

Surgical Procedures

The first stage consisted of placement of a polytetrafluoroethylene (PTFE) conduit between the ascending and descending aorta, ligation of the PDA, and PA banding. In all patients, pressures in the ascending aorta were monitored distal to the site of the partially occluding clamp on the ascending aorta. Because the right subclavian artery came off the descending aorta in 8 infants with type B interruption, other methods besides right radial artery monitoring were used when necessary. These included direct distal monitoring and temporal artery cannulation to allow monitoring after placement of the partially occluding clamp. The ascending aorta is usually very narrow; therefore, the aorta was dissected free circumferentially where the partially occluding clamp was to be placed, which allowed the vessel outside the clamp to expand. The distal pressures often rose while the clamp was in place and always exceeded 50 mm Hg initially. Before clamp removal, the systolic pressures were typically in the range of 60 to 80 mm Hg. For type B IAA, either an 8-mm (9 patients) or 10-mm (8 patients) thin-walled PTFE (Gore-Tex) conduit was used after the first operation in the series, in which only a 6-mm graft was selected. More recently, a 10-mm graft was more commonly placed, even in infants as small as 2.4 kg. Both anastomoses were carried out using 7-0 polypropylene interrupted or running suture. The graft was cut as short as possible and beveled to prevent kinking. For the 2 infants with type A IAA, a subclavian artery flap repair was used. The PDA was ligated and the PA banded to reduce the distal pressure to 40% or less of the systemic systolic pressure.

The second stage consisted of Dacron patch closure of the VSD and PA debanding with PA reconstruction if necessary. The second stage for 1 infant with truncus arteriosus also consisted of placement of a 12-mm xenograft valved conduit, later changed to an 18-mm graft when the patient was 5 years old (the other patient with truncus arteriosus died before the second stage). The infant with transposition of the great arteries underwent repair elsewhere. In the infant with the right PA arising from the aorta, correction was accomplished by direct anastomosis to the main PA at the second-stage repair 6 days after the first operation. For the other infants, the second stage was carried out 2 months to 1.6 years after the first stage. Recently, we have been performing the second stage at an earlier date and, in general, recommend this be done at 2 to 3 months of age.

Only 1 child (9 years old) has had development of a major gradient (>40 mm Hg) across the conduit and undergone augmentation of the aortic repair. In this patient, an 18-mm Dacron graft was placed between the ascending and descending aorta through the previous left thoracotomy incision. The graft was positioned proximal to the initial conduit, which was left in place, and therefore, cardiopulmonary bypass was not needed. The operation was straightforward and the postoperative hospitalization short (4 days).

Results

All 20 infants survived the first-stage repair despite the complexity of their condition, several having major additional cardiac defects (Table 2). Only the first 2 patients went to the operating room in less than hemodynamically stable condition. As stabilization with prostaglandin E_1 infusion, ventilator control, and inotropic support measures have improved, the condition of these infants before operation has benefited. Two infants had also sustained critical periods of cardiac arrest during cardiac catheterization.

Table 2. Results for Repair of Interrupted Aortic Arch in Infants[a]

Survival	No. of Patients
After first stage	20/20 (100)
Late deaths	2/20 (10)
After second stage	15/18 (83)
1985–1990	8/8 (100)
After third stage	1/1 (100)

[a] Numbers in parentheses are percentages.

Table 3. Recent Results With Staged Repair (1985–1990)

Patient No.	Year of Operation	Associated Lesions	Age at Operation		Follow-up Gradients[a]	
			Stage 1	Stage 2	Conduit	Subaortic
1	1985	VSD	3 d	5 mo	None	None[b]
2	1985	VSD	3 d	6 mo	None	None
3	1985	VSD, truncus	1 mo	6 wk	None	None[b]
4	1987	VSD, aortic stenosis	3 d	2 mo	None	None
5	1987	VSD	2 d	2 mo	None	None
6	1988	VSD	8 d	10 mo	None	None
7	1989	VSD, TGA	7 d	6 mo	None	None[b]
8	1990	VSD, RPA from aorta	2 d	12 d	None	None[b]

[a] None means that no measurable gradient was demonstrated by echocardiography. [b] Gradient data also were obtained from cardiac catheterization.

RPA = right pulmonary artery; TGA = transposition of the great arteries; VSD = ventricular septal defect.

There were two late deaths before the second-stage repair. One infant with severe subaortic obstruction and biventricular hypertrophy died after cardiac catheterization and angiography in preparation for the second-stage operation. The other infant, who also had truncus arteriosus, died unexpectedly at home.

Fifteen (83%) of 18 infants survived the second-stage operation (see Table 2). Two of these operative deaths were due to low cardiac output. The first infant in this series was in unstable condition at the time of the first operation, and the PA band could not be adequately tightened. Pulmonary vascular resistance increased and led to her postoperative death from low cardiac output. One infant had marked right ventricular hypertrophy; with debanding, the severe cavitary hypoplasia resulted in poor filling and very small stroke volume, which was readily documented by echocardiography. No substantial improvement in the right ventricle had occurred by the time the infant died 3 days later. This child also had moderate (25- to 30-mm Hg gradient) subaortic obstruction, which may have contributed to the severity of the right ventricular hypertrophy that developed despite a relatively short period (10 weeks) between stages. The third child had multiple cerebral infarctions of unknown etiology, and support was withdrawn.

In the earlier part of the series, the second stage was often delayed until the patient had outgrown the PA band. This led to a variety of problems and accounted for virtually all the instances of major morbidity and mortality after the second-stage operation. Among the problems produced by undue delay were (1) severe right ventricular hypertrophy, (2) substantial PA scarring, and (3) marked dilation of the PA after removal of the band, which, in turn, caused severe tracheobronchial malacia. Similar problems have been avoided by earlier debanding, and these deaths were all in the early part of the series (infants born before 1985). For a variety of reasons including early debanding, the success rate has continued to improve and from 1985 to 1990, the last 8 infants have all successfully undergone first- and second-stage repair (8/8, 100%) and are getting along well (Table 3).

In only 1 patient did severe (≥40-mm Hg gradient) left ventricular outflow tract obstruction develop. This patient died after cardiac catheterization in preparation for the second stage. A moderate gradient (20 to 40 mm Hg) was present in 1 patient (5%) after staged repair, and no gradient was found in 18 patients (90%). The 9 long-term (5 to 10 years) survivors have had no evidence of left ventricular outflow tract obstruction by Doppler echocardiography or cardiac catheterization.

Despite the relatively large size of the PTFE conduits that were placed at the first operation, we anticipated that they would be outgrown within a few years. Only 1 child (aged 9 years), however, has required augmentation of the aortic conduit to date (1990). As described, this proved to be a straightforward procedure in which an 18-mm Dacron conduit was placed without need of cardiopulmonary bypass. As these children become older, it is anticipated that all will require an additional aortic conduit. When a substantial gradient (40 mm Hg) has developed across the conduit, the ascending aorta should be relatively large, and only one additional operation will probably be required.

Comment

The surgical results of treatment of infants born with IAA, VSD, and PDA have shown great improvement. As a reflection of the previous difficulties in achieving initial success, a variety of procedures have been used. At present, however, two methods of operative repair are most widely used and consist of either a primary repair or a staged repair. In the latter, the IAA is corrected first, and later the intracardiac repair is carried out. From a highly lethal condition where success was documented by individual cases [10, 11], to small series [2–4] in which 50% of infants survived, to more recent reports [6–9; Bowman FO Jr, unpublished results] with even greater promise, the surgical treatment has become increasingly successful.

Despite the improvement, however, substantial operative mortality persists, and a number of late problems have been reported. Complete primary repair has obvious appeal, but the complexities encountered in achieving long-term success in this group of infants mean the results with primary and staged repairs must be compared to determine the indications for each. Because of our interest

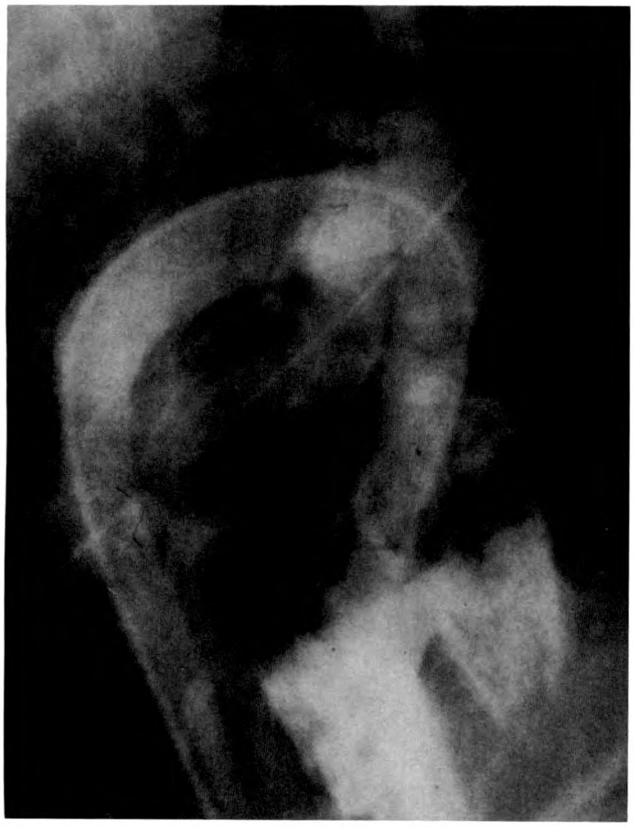

Fig 1. Follow-up angiogram made 14 months after first-stage placement of a 10-mm polytetrafluoroethylene graft, which remains larger than ascending or descending aorta.

in the staged repair, the purpose of this report is to update our results and further evaluate the current role of the staged repair. Most of the problems associated with both the primary repair and the staged repair have been defined in previous reports. As experience with these procedures has grown, some problems have decreased in incidence while others have persisted or even increased with longer follow-up. This report presents the results with a relatively large series of infants who have undergone staged repair, some of whom now approach a medium-term follow-up. Our analysis concentrates on the advantages and disadvantages of the staged repair.

An early appeal of the staged repair was the ability to get these infants through the first operation no matter what their preoperative condition. Our experience of uniform survival has been duplicated by others (Lamberti JJ: see commentary; Elkins RC: personal communication). This advantage of the staged repair has diminished somewhat as preoperative preparation has become better. Techniques to establish preoperative stabilization have improved, and the majority of these infants go to the operating room in relatively good condition. Nevertheless, there will be some infants who may be adversely affected by cardiopulmonary bypass and the attendant anticoagulation. Infants who have sustained a critical cerebral insult and particularly those who may have already had intracranial bleeding would be at risk. Other consequences of an episode of shock, such as anuria or necrotizing enterocolitis, can add hazard to complete repair. For infants whose condition is especially precarious or for whom other cardiac anomalies make a primary repair inordinately difficult, the staged repair would seem to be the better choice. But aside from the initial advantages, more long-term information will be needed to determine which approach will lead to fewer late problems and will be the best choice even for infants who can be well prepared for the first operation.

Some infants have associated cardiac defects that would increase the difficulty of carrying out a successful primary repair. An example in our series is the infant with anomalous origin of the right PA from the aorta. The right PA arose from the right side of the aorta, and the main PA was directed along the course of the left PA. The primary repairs of the PA and the IAA would be under tension in opposite directions and would tend to decrease the lumen of the right PA. If the PA gap was bridged with an allograft or PTFE segment, at least one more operation would be necessary later to enlarge the vessel. Other lesions, such as truncus arteriosus, offer less in the way of complicating technical factors. The magnitude of the operation required for complete repair, however, increases the overall risk.

There are several disadvantages to the staged repair. The first operation is more difficult technically than the primary repair. The ascending aorta is relatively narrow, particularly when both subclavian arteries take off from the descending aorta, and placement of a partially occluding clamp must be precise. Dissection of the aorta circumferentially seems to allow the aorta excluded from the clamp to expand and ensure adequate distal perfusion. Nevertheless, the rim of tissue left for anastomosis is narrow, and the anastomosis is more exacting than for the primary repair carried out under circulatory arrest.

The most persistent problem, whether primary or staged repair is used, appears to be late aortic obstruction. Placement of an 8- or 10-mm PTFE conduit assures that at some point, a gradient will develop across this arch segment. Because balloon dilation cannot be used, relief will necessitate an operation. To date, only 1 child (aged 9 years) has shown a significant gradient (>40 mm Hg) across the aortic arch, although another (aged 9 years) has a 20-mm Hg gradient by Doppler echocardiography. It appears that arch augmentation will more likely take place at 7 to 10 years of age than at 3 to 5 years of age as previously predicted. The operative choices would appear to be replacement of the previously placed PTFE graft or placement of a second conduit. We chose the latter, which resulted in a relatively straightforward operation without the need of cardiopulmonary bypass and with a very short hospitalization. This approach appears to be the best choice for the third stage.

In most infants who undergo primary anastomosis, relative obstruction at the anastomotic site will also develop. The actuarial time-related freedom from evidence of aortic arch obstruction 18 months after repair was only about 36% in the Boston experience [7]. Direct repair of this anastomotic obstruction is a major operation, and a 50% mortality was reported in that limited experience [7]. Because of these early results, balloon dilation is being used to relieve these anastomotic obstructions and has been reported to usually be effective over the short term [7]. The technology for dilating obstructions should continue to improve, and this may prove to be a satisfactory solution. The long-term success of balloon dilation, however, will be important to establishing an advantage of primary repair. If operative relief of obstructions after the primary repair is required, then placement of an aortic conduit will probably be the best choice. Therefore, the staged and primary repairs may end up very similarly with an artificial graft for the transverse aorta, which would tend to obscure the differences between these approaches.

The posterior malalignment of the outlet septum that seems to be common in infants with IAA and VSD often contributes to the development of left ventricular outflow tract obstruction [12]. It has been predicted that this would be aggravated by the placement of a PA band and the development of biventricular hypertrophy including the subaortic muscle that causes the obstruction [13]. The incidence of critical left ventricular outflow tract obstruction (≥40 mm Hg) has been low (1/20, 5%) in our series and was absent among the 35 staged repairs of the Boston study [7]. In contrast, all cases of subaortic obstruction occurred in infants undergoing primary repair, and subaortic obstruction was found within 3 years in 42% of survivors [7]. Clearly, the presence of a PA band does not seem to increase the chance of subaortic obstruction. Unexpected and unexplained, these data also suggest that the incidence may be increased in the primary repair group. The difficulty in relieving left ventricular outflow tract obstruction means the incidence of this complication in regard to the reparative approach should be documented in future series.

Other technical problems seem to be increased with the usual primary repair. Primary anastomosis of the aortic segments may compromise the right PA or even the tracheobronchial tree and can be difficult to treat over the long term. The occurrence of these problems has been acknowledged, but the incidence has not been reported. When an interposition aortic graft is used, compression of the right PA does not occur. Tracheobronchial compression can also be avoided by the staged repair, particularly with division of the PDA.

From our early experience [2], our survival rate of 57% for the staged repair was the first reported with greater than 50% survival of infants with type B IAA. All infants survived the first stage; however, the PA band was the primary source of difficulty when left on too long. With this realization and consequent early second-stage repair, our results have improved substantially. Since 1985, 8 (100%) of 8 infants have survived the second stage and appear to be getting along well without significant residual cardiac abnormalities. Results with primary repair have also improved, and the most recent studies [7, 9] had 82% and 83% operative survival rates. Other series have been reported with excellent results with staged repair, indicating that this approach compares favorably with primary repair in the survival of these infants (Lamberti JJ, Elkins RC: personal communications). Because of these results, we have continued to use the staged repair.

In summary, we have presented our series of 20 infants born with IAA and VSD who have undergone a staged repair. With early PA artery debanding, our results improved, and since 1985, the last 8 infants have survived complete intracardiac repair and are doing well. The very high rate of success with the staged repair supports its use for infants at high risk for primary repair or in the presence of additional anomalies that would inordinately increase the technical difficulty. Staged repair will also be a very acceptable alternative to primary repair, particularly if the obstruction that develops frequently at the primary aortic repair site proves difficult to treat over the long term. At present, the overall results with both the staged and primary repairs continue to improve, and longer follow-up will be necessary to define more precisely the relative advantages and disadvantages of these two approaches and accurately judge which repair should be used for the majority of these infants. The end results may become very similar. Therefore, the lower initial mortality and greater flexibility in the face of additional cardiac lesions may continue to favor the staged repair.

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COMMENTARY: Staged Repair of Interrupted Aortic Arch

At the Children's Hospital and Health Center of San Diego, my colleagues and I have followed a policy of staged repair of interrupted aortic arch since 1979. We have encountered 23 infants with interrupted aortic arch and a ventricular septal defect. Nineteen infants were born with ventricular septal defect and normally related great arteries. Three infants had transposition of the great arteries and ventricular septal defect, and 1 infant had truncus arteriosus. My comments are limited to the group with ventricular septal defect and normally related great arteries (19 patients); all had type B interruption. This patient group is remarkably similar to the patients of Irwin and Foker. Eleven of the 19 had an aberrant right subclavian artery. Only 4 patients underwent preoperative cardiac catheterization.

The stage I operation is virtually identical to the Minnesota approach, with 14 patients receiving an 8- or 10-mm graft. Five patients had smaller grafts; 4 of the 5 were critically ill, and 1 weighed 1.7 kg. Seventeen of the 19 patients underwent simultaneous pulmonary artery banding.

The only operative death (5%) with the stage I procedure occurred in a 2-day-old female neonate who remained profoundly acidotic after aggressive resuscitation. Persistent acidosis in the presence of documented patency of the ductus arteriosus is probably a contraindication to operation, as this presentation suggests an additional pathological process. The infant died of a clotted aorta with bowel necrosis.

Seventeen of the 18 stage I survivors underwent ventricular septal defect closure and debanding when necessary. There was one early death at 6 hours and one late death (from sepsis) on the 56th postoperative day. One patient died suddenly at home while awaiting stage II repair; the cause of death was probably subaortic stenosis. The median time of stage II repair was 26 days after the stage I procedure.

In summary, 15 (79%) of the 19 patients are alive and well 6 months to 10 years after presentation. Seven patients have undergone additional operations after the stage II procedure. Three patients had operations on the left ventricular outflow tract. Two patients, age 12 and 19 months, underwent resection of subaortic stenosis; 1 had a discrete subaortic membrane and the other, tunnel subaortic stenosis. Another patient underwent aortic valvotomy at 10 months of age. Two additional patients have mild, stable left ventricular outflow tract gradients.

Four patients have undergone a reoperation designed to relieve proximal compartment hypertension. Such operations might be termed stage III procedures. They were performed when the patients were between 18 and 36 months of age (mean age, 26 months). All 4 patients underwent initial repair with a 5- or 6-mm Teflon graft. None of the patients with an 8- or 10-mm graft has required reoperation for aortic arch obstruction yet.

Stage III operations were individualized. Two patients underwent repeat left thoracotomy with placement of a 12-mm prosthetic graft from the ascending aorta to the descending aorta. One patient underwent placement of a left ventricular apical conduit for the treatment of persistent tunnel subaortic stenosis. In addition, that patient also had placement of a 10-mm graft from the ascending aorta to the descending aorta. Finally, 1 patient underwent replacement of the aortic arch using the technique of deep hypothermia and circulatory arrest. This patient, who had a birth weight of 1.7 kg, underwent initial implantation of a 6-mm graft. The graft was replaced with a 12-mm tube graft when the patient was 30 months old.

Our long-term results are quite similar to those of Irwin and Foker. We believe that a long-term survival rate of 79% is acceptable compared with rates in the literature. At present, we recommend early stage II operation. Pulmonary artery banding for a period of a few weeks should not cause critical distortion of the pulmonary artery, nor should banding produce progressive subaortic stenosis if the band is removed in less than 1 month. The staged approach avoids a long operation associated with a substantial period of deep hypothermia and circulatory arrest. We have not seen compression of the pulmonary artery or the bronchi by the tube graft. When the child outgrows the stage I prosthetic tube, an additional graft can be placed with low risk and low morbidity (no deaths in our series). When early debanding is performed, the incidence of subaortic stenosis should be identical to that seen with primary (one-stage) repair.

Although other groups have reported good short-term success with primary repair, we await complete analysis of the natural history of patients undergoing primary reconstruction of the aortic arch. Accurate comparison of one-stage versus two-stage repair must include early and late mortality data as well as timing and number of repeat interventions. In addition, the occurrence of associated morbidity such as neurodevelopmental delay or pulmonary complications secondary to airway compression must be noted.

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COMMENTARY: The Argument for One-Stage Repair

I am grateful for the opportunity to comment on our current approach of one-stage repair with direct aortic arch anastomosis for interrupted aortic arch. My colleagues and I believe that it would be a regressive step to revert to a two-stage approach that includes placement of an ascending to descending aortic conduit.

In 1988 [1], we published the results of a review of 63 children who had undergone repair of interrupted aortic arch. This study was undertaken with the kind cooperation of Dr John Kirklin and colleagues. It was clear from that review that the results for repair of interrupted aortic arch have improved dramatically over the last decade. Figure 1 illustrates that much of this improvement has resulted from an improved metabolic status of children immediately before the operation. Although the cornerstone of this metabolic resuscitation is the use of prostaglandin E_1 to maintain ductal patency, this is only one aspect of the resuscitation process. It is essential that ventilation be carefully manipulated to maximize pulmonary vascular resistance, thereby maximizing systemic perfusion. In general, at least 2 days of resuscitation are required after admission of the child to the hospital; on occasion, particularly when sepsis is present, resuscitation can extend for up to 1 week. Following such an approach, we have been able to decrease the early mortality risk in the current era for children with interrupted aortic arch and ventricular septal defect (VSD) to less than 10%. This improvement in outcome has occurred despite moving away from two-stage to one-stage repair.

Between 1985 and 1987, 82% of patients underwent one-stage repair of interrupted aortic arch with VSD at the Children's Hospital, compared with 44% of patients between 1981 and 1983. Concurrently, there has been a shift to direct anastomosis: between 1985 and 1987, 82% of patients underwent aortic arch repair by direct anastomosis, whereas between 1981 and 1983, none of 9 patients underwent repair by direct anastomosis. Previous concerns regarding development of left bronchial obstruction due to a bowstring effect across the left main bronchus have not materialized. We believe that the avoidance of left bronchial obstruction is due to extensive mobilization of the ascending aorta and the head vessels in addition to wide mobilization of the descending thoracic aorta [2]. In type B interrupted aortic arch, it is also not uncommon for us to divide the left subclavian artery to further decrease tension on the aortic anastomosis. Direct aortic arch anastomosis has been complicated by a relatively high incidence of anastomotic gradient at follow-up catheterization. However, this can generally be dealt with by balloon dilation angioplasty.

The other important current issue with respect to interrupted aortic arch is the approach to be taken when subaortic stenosis is present [3]. Our approach has been a very conservative one in that we have rarely altered our initial approach in the neonatal period based on the suspected presence of left ventricular outflow tract obstruction. Within 3 years, the actuarial freedom from evidence of left ventricular outflow tract obstruction in our series was 58% (Fig 2). However, at this point it has been possible to deal with obstruction by relatively conventional procedures such as resection of subaortic tissue or a Konno-type operation. It should rarely be necessary to baffle left ventricular outflow through the VSD and to use the proximal pulmonary artery as a neoaorta. Such an

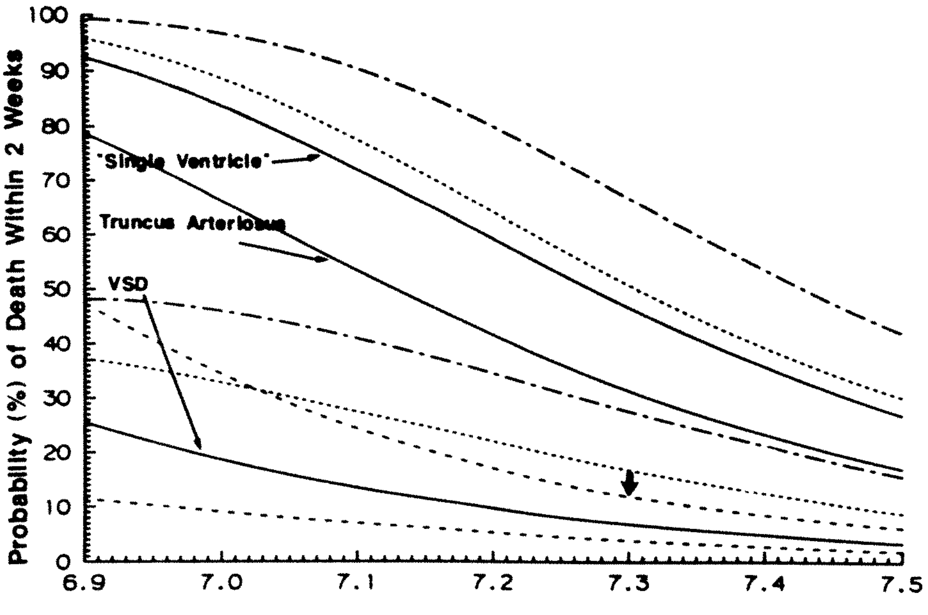

Fig 1. Relationship of probability of early death within 2 weeks of operation to immediate preoperative pH highlights the importance of preoperative resuscitation before undertaking reparative operation. This applies both for simple interrupted aortic arch with ventricular septal defect (VSD) as well as for more complex anomalies.

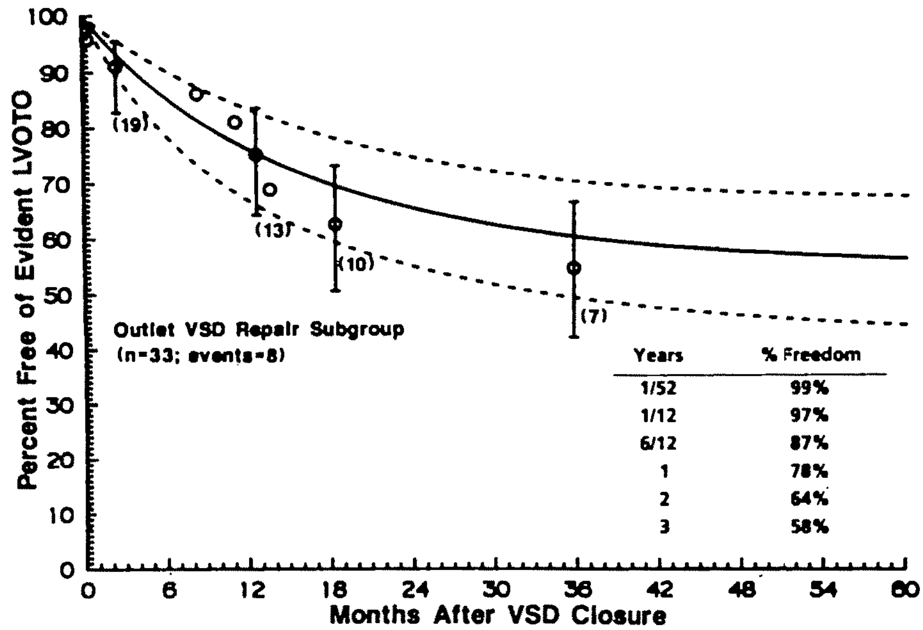

Fig 2. Actuarial freedom from evident left ventricular outflow tract obstruction (LVOTO) after repair of interrupted aortic arch with ventricular septal defect (VSD) in the outlet portion of the right ventricular aspect of the septum.

operation is undoubtedly palliative in the sense that a right ventricular to pulmonary artery conduit is required.

We attempted to find predictors of subsequent development of subaortic stenosis and found that the dimension of the left ventricular outflow tract during the neonatal period was *not* a useful predictor. This is in spite of finding a narrowest dimension as small as 3 mm. Perhaps dynamic factors are involved analogous to those seen with left ventricular outflow tract obstruction in transposition. After the VSD has been closed, there may be a rightward shift of the conal septum, thereby decreasing the degree of subaortic stenosis. An interesting finding of our study was that the presence of an aberrant right subclavian artery with type B interrupted aortic arch was a weak predictor ($p = 0.13$) of subsequent development of left ventricular outflow tract obstruction (Table 1). Perhaps this anomaly should be thought of as an intermediate morphological subtype between type B and type C interrupted arch. Presumably, during development, less blood passes out the ascending aorta relative to the type B interrupted aortic arch, which may influence in utero development of the left ventricular outflow tract.

I would like to make one final point regarding operative technique. Dr Bowman (unpublished results) referred to his preference for the transventricular approach to the posterior malalignment VSD. In a morphological study of approximately 40 specimens of interrupted aortic arch with VSD (unpublished data), we concluded that the conal septum was not only posteriorly malaligned but also frequently moderately to severely hypoplastic, that is, there was very little tissue between the aortic and pulmonary valves. This contributes to difficulty with closure of the upper edge of the VSD, particularly if one attempts to approach the VSD by a transatrial, transtricuspid valve approach. Under such circumstances when the preoperative echocardiogram demonstrates hypoplasia of the conal septum, we have found that an approach through the pulmonary valve allows satisfactory exposure and closure of the VSD.

Table 1. Aberrant Right Subclavian Artery With Type B Interrupted Aortic Arch[a]

Present	No. of Patients	LVOTO	
		n	%
Yes	9	5	(56)
No	19	5	(26)

[a] $p = 0.13$.

LVOTO = left ventricular outflow tract obstruction.

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The Myth of the Aortic Annulus: The Anatomy of the Subaortic Outflow Tract

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Surgical repair of the small aortic root is limited in part by the very structure of the outflow tract from the left ventricle. The root is not constructed on the basis of a ringlike annulus supporting the leaflets of the aortic valve. The only truly circular structure within the outflow tract is the junction of the aortic wall with the underlying ventricular structures, themselves partly muscular and partly fibrous. This circular ventriculoarterial junction is crossed by the semilunar attachments of the leaflets of the aortic valve, producing an interlinking arrangement between the expanded aortic sinuses and three triangles of fibrous tissue placed beneath the apexes of the commissures between the valve leaflets. The triangles form extensions of the left ventricle that are related, in part, to the pericardial cavity surrounding the heart. The arrangements of the attachment of the leaflets in malformed valves with two (or only one) effective leaflets are highly abnormal, although these valves are usually produced on the template of three aortic sinuses. The valve with two leaflets rarely gives problems during childhood. In valves producing "critical stenosis," there is usually only one effective leaflet, a condition due to incomplete liberation of two of the anticipated three commissures. Detailed study shows that, in these malformed hearts, the attachment of the leaflets is much more annular than in normal valves, with inadequate formation of the fibrous triangles.

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The surgical literature abounds with descriptions of the "annulus" of the arterial valves and with accounts of the optimal way of enlarging this purported structure to treat patients with stenotic and obstructive subarterial outflow tracts. All those who operate on hearts with malformed arterial valves are well aware that, in reality, the attachments of the leaflets are not arranged in ringlike fashion around the circumference of the ventricular outlets. Indeed, if the bases of the leaflets were attached in such annular formation, it would be impossible for the leaflets to open properly during ventricular systole. Instead, they are attached in semilunar fashion.

Is it necessary that these semilunar attachments be described in terms of an annulus? Those who pay scant attention to matters of style and description are likely to argue that it matters little whether there is or is not a true annulus as long as those who operate on the malformed hearts understand the anatomy of the structures on which they work. We take a different stance because we believe it important to use words that reflect accurately the features of the structures they describe. This is particularly true for those who are beginning in the field and are attempting to understand the complex morphology of normal and abnormal arterial valves.

In this review, therefore, we will present our understanding of the arrangement of the arterial valves, which is founded on the concept that the leaflets are attached in semilunar fashion rather than attached to a ringlike annulus [1]. We are well aware that the anatomy to be emphasized is already well recognized. The semilunar arrangement of the leaflets is described in current textbooks of anatomy [2] and is depicted and described as a tricorn (a hat with three peaks) in a popular textbook of surgery [3], where appropriate precedence is given to the important work of Zimmerman [4]. Accurate descriptions of the histology of the semilunar attachments of the leaflets of the arterial valves were also provided long ago by Gross and Kugel [5] and by Lewis and Grant [6]. Nevertheless, the cardiological world at large continues to describe an "annulus" within the outflow tracts of the ventricles. Our review is an attempt to put such terminology into the context of the observed anatomy of the leaflets of the normal valves and to show how their normal semilunar arrangement is distorted when the valves are congenitally malformed.

Material and Methods

The material on which our review is based comprises the large number of normal hearts that we have seen over the

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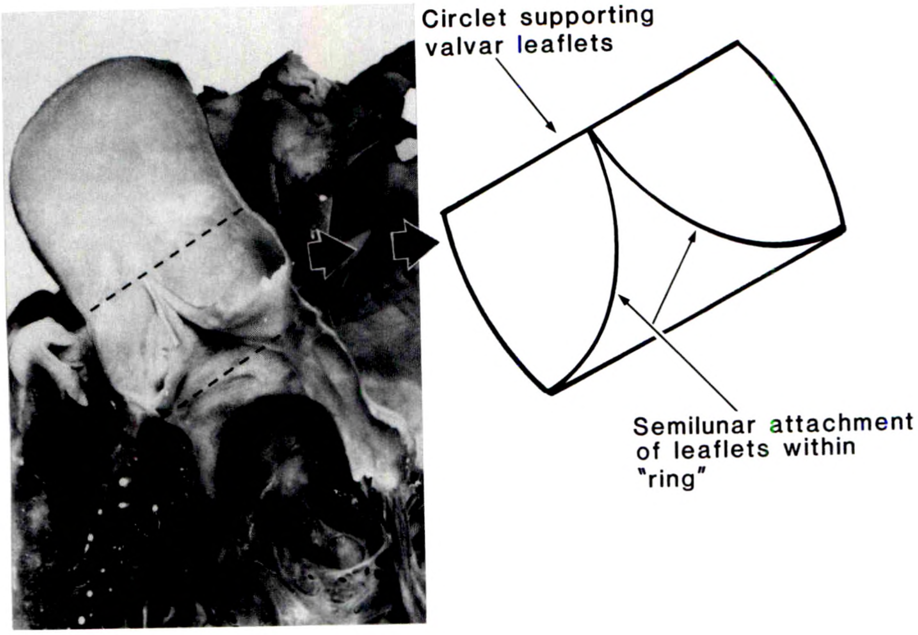
Circlet supporting valvar leaflets
Semilunar attachment of leaflets within "ring"

Fig 1. Long-axis section through the outflow tract of the left ventricle showing the cylindrical section that must be removed so as to take out the entire aortic valve. Within this circlet, which could be slipped on the finger like a ring, the leaflets of the valve are attached in semilunar fashion (see Fig 7).

years and the abnormal hearts with valvar aortic stenosis in the cardiopathological collections of our institutes. None of the normal hearts have been formally analyzed, but some have been sectioned histologically to determine the mode of attachments of the valve leaflets and to ascertain their connections to the fibrous skeleton of the heart. We will make reference to two more detailed and formalized studies of hearts with malformed valves. One [7] has been published, and one is in preparation (McKay R, Smith A, Leung MP, Arnold R, Anderson RH. Morphology of critical aortic stenosis in infancy with reference to haemodynamic function and clinical management. Unpublished observations). The published account was devoted to the deformed aortic valve having two leaflets. This material was analyzed by Angelini primarily, we being part of her collaborative team. The other series involved 21 hearts with "critical aortic stenosis" seen in infancy. They have been analyzed in depth by ourselves and colleagues at the Institute of Child Health, University of Liverpool. This material has, as yet, not been published and, therefore, will be considered only in the broadest terms as they apply to the concepts to be discussed.

Glossary and Etymology

It should be clear from our introduction that part of the problem of providing an adequate description of the anatomy of the subarterial outflow tracts is an inappropriate use of words. It is important, therefore, to establish the precise meaning of the words used in our descriptions, so those who disagree will have clear definitions on which to base their dissent.

ANNULUS. According to the *The Shorter Oxford English Dictionary* [8], *annulus* is the diminutive form of the Latin word *anus*, the latter meaning a ring. Although rings as worn nowadays can take various forms, the same dictionary defines this word as a circle or circlet as carried on the finger. This is how we will use the word.

SEMILUNAR ATTACHMENTS. If the area of outflow tract supporting the leaflets of an arterial valve is removed in its entirety, it takes the form of a ring that could be slipped on the finger (Fig 1). Within this circlet, however, the valve leaflets themselves are attached in semilunar fashion, with three such half-moons being present in the normal valve. The attachment of each leaflet rises to its zeniths at the peripheral attachments of the commissures and is at its nadir at the base of the leaflet.

CUSP. Although used by most pathologists and surgeons as synonymous with the word *leaflet*, the word *cusp* means a projection or point [9] and is used appropriately by dentists when describing elevations on the surfaces of the teeth.

COMMISSURE. This word means a seam or the place where two bodies touch or unite [10]. When applied to an arterial valve, therefore, the commissure should, logically, describe the line from the periphery to the center of the valve orifice over which the surfaces of adjacent leaflets coapt (Fig 2). Often, however, pathologists describe only the peripheral part of this junction in terms of the commissure.

VENTRICULOARTERIAL JUNCTION. Perhaps the most important point requiring emphasis for the understanding of the anatomy of arterial valves is that the semilunar attachments of the leaflets (see Fig 1) produce a discrepancy between the anatomical and physiological ventriculoarterial junctions. The *anatomical* junction, made between the ventricle and the arterial trunk it supports, takes the form of a circle over which the fibrous wall of the trunk is attached to the supporting ventricular structures. In the left ventricle, these supporting structures are in part muscular and, because of the fibrous continuity between the leaflets of the aortic and mitral valves, in part fibrous (Fig 3). The *physiological* junction, in contrast, is marked by the semilunar locus of attachment of the leaflets. The entire area beneath this locus is, in terms of hemodynamic pressures, part of the left ventricle. The area distal to the locus is, in the same hemodynamic terms, part of the aortic sinus. The arrangement of the attachments, therefore, results in three segments of ventricular wall becoming part of the aortic sinus and three triangular areas of arterial wall being incorporated into the outflow tract of the ventricle (Fig 4).

TRIANGLES VERSUS TRIGONES. To understand the complex arrangement of the aortic root, it is also necessary to emphasize the relationship of the semilunar attachment of the leaflets to the fibrous skeleton of the heart. The skeleton is often displayed as an extensive structure enclosing the attachments of all four cardiac valves [11]. Dissections as well as histological studies [6, 12] show this not to be the case. In the aortic root, the fibrous skeleton forms the posterior wall of the outflow tract in the area where the leaflets of the aortic valve are in fibrous continuity with those of the mitral valve (Fig 5; see Fig 3). The two ends of this area of fibrous continuity are thickened to form the left and right fibrous trigones, respectively. These trigones are the best-formed parts of the skeleton. The right fibrous trigone itself is continuous with the membranous part of the septum, and these two structures together form the central fibrous body through which the axis of atrioventricular conduction tissue penetrates to pass from the muscular atrioventricular septum to the crest of the ventricular septum (see Fig 5).

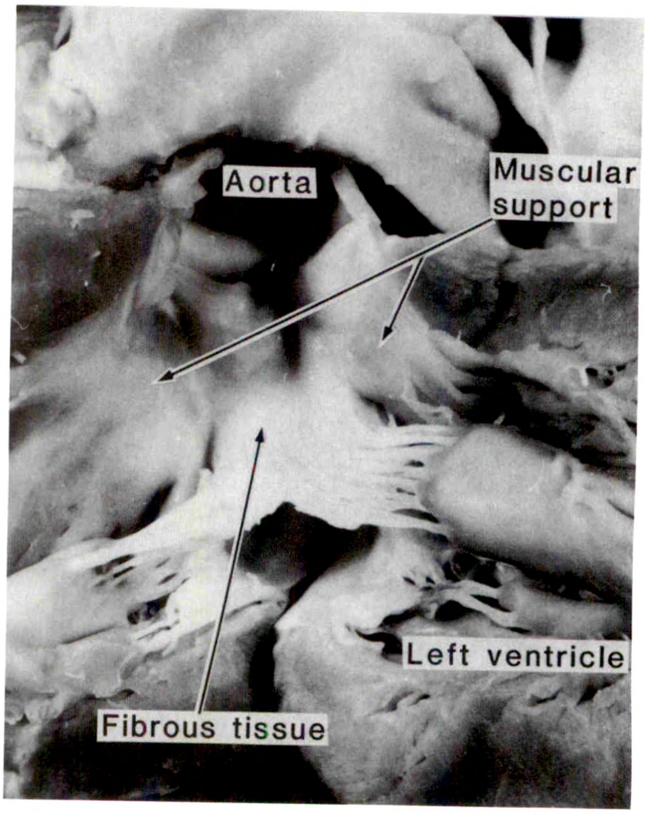

Fig 3. View of the opened outflow tract of the left ventricle showing how the leaflets of the aortic valve are supported in part by the musculature of the left ventricle and in part by fibrous continuity with the aortic leaflet of the mitral valve.

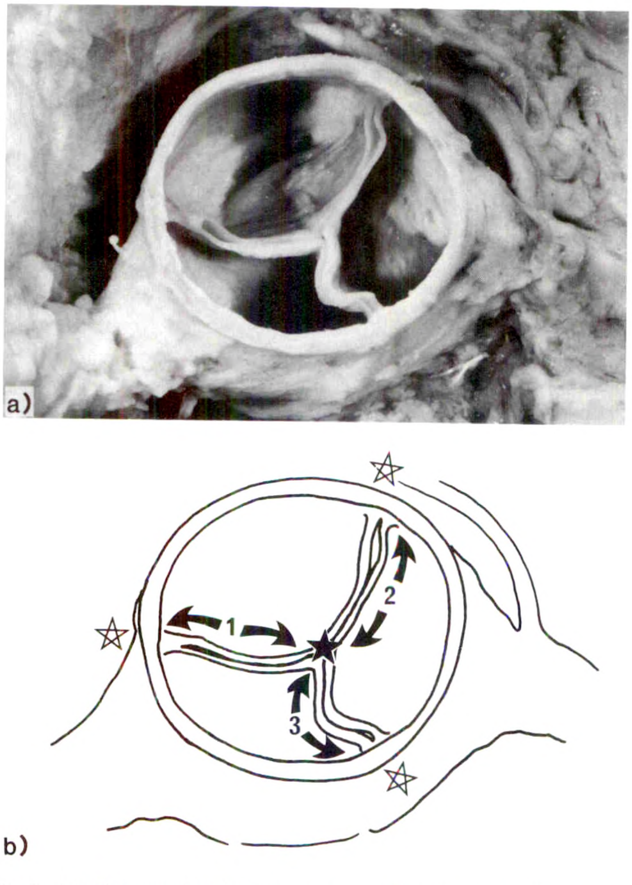

Fig 2. (a) Photograph and (b) line interpretation of aortic valve as seen from above with the leaflets in closed position. Note how the lines of coaptation, or commissures, extend from the periphery to the center of the valve orifice (open and closed stars).

Sections can be taken from the base of the leaflets of the aortic valve where they are attached to the muscular walls of the left ventricle to give the spurious impression that a cordlike collagenous ring does provide support for them. Serial sections, however, show that this cordlike structure is also arranged in semilunar fashion. It becomes incorporated in the substance of the arterial wall as the attachments of the leaflets rise to the peripheral extent of the commissures (Fig 6). Indeed, beneath the apex of the commissures, only a relatively thin layer of the arterial wall separates the inside of the left ventricle from the extracardiac space. These thinner areas of fibrous wall can accurately be described as the interleaflet triangles (see Fig 4) [7].

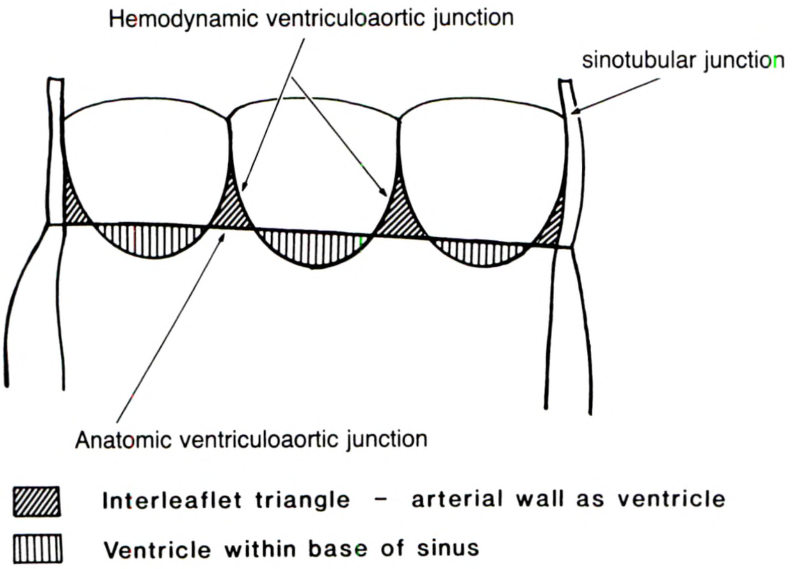
Hemodynamic ventriculoaortic junction
sinotubular junction
Anatomic ventriculoaortic junction
▨ Interleaflet triangle – arterial wall as ventricle
▥ Ventricle within base of sinus

Fig 4. Discrepancy between the anatomical and hemodynamic ventriculoarterial junctions. The discrepancy is due to the semilunar attachments of the leaflets.

Results

The Normal Aortic Root

As described in our glossary, the area of the outflow tract of the left ventricle supporting the leaflets of the aortic valve takes the form of a cylinder (see Fig 1) in which the leaflets are attached in the fashion of three half-moons. This can best be demonstrated by spreading out the cylinder so that its surface is seen as a flat strip (Fig 7a). Removing the leaflets from their attachment demonstrates the discrepancy between the anatomical and physiological ventriculoarterial junctions (Fig 7b).

When seen in the intact heart, either from below or above, there is an interlinking of the sinuses of the aorta and the supporting structures of the ventricle. The three interleaflet triangles extending beneath the apices of the commissures and formed by the relatively thin wall of the aorta are incorporated as extensions of the ventricular outflow tract. These three extensions "point" toward the transverse sinus of the pericardium, toward the roof of the membranous septum, and toward the infundibulum of the right ventricle, respectively [7]. With this arrangement of the outflow tract, the leaflets of the arterial valve, which have shorter free edges than attached margins, are free to fall back into the aortic sinuses during ventricular systole to ensure unobstructed flow of blood into the aorta. During diastole, the leaflets are forced together to coapt along a line away from their free edge. These areas of coaptation, or commissures, extend from the periphery to the center of the valve orifice (see Fig 2). As discussed, the entire cylinder forming the root and supporting the valve leaflets can be considered to represent a ring. The only circular structure within the overall ring, however, is the anatomical ventriculoarterial junction.

The Aortic Valve With Two Leaflets

Careful examination of these valves shows that, in most instances, there is evidence of fusion of two leaflets in a

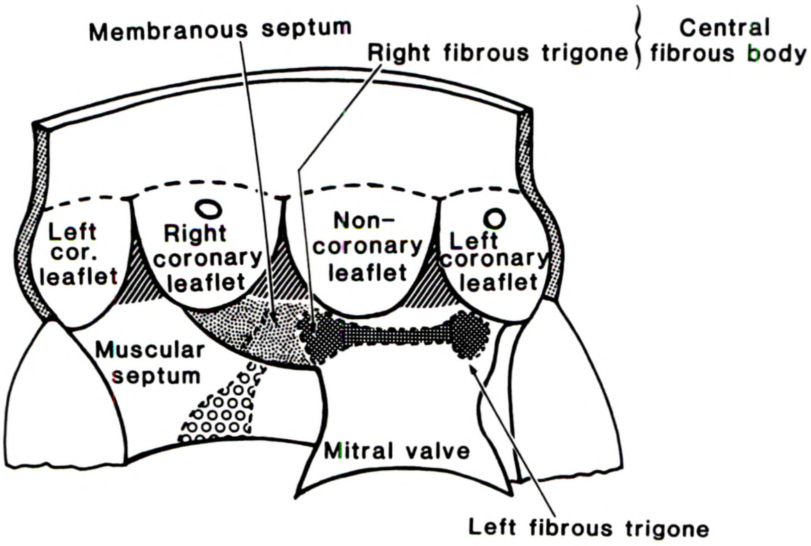

Fig 5. Relationships of the leaflets of the aortic valve to the components of the fibrous skeleton and the axis of atrioventricular conduction tissue.

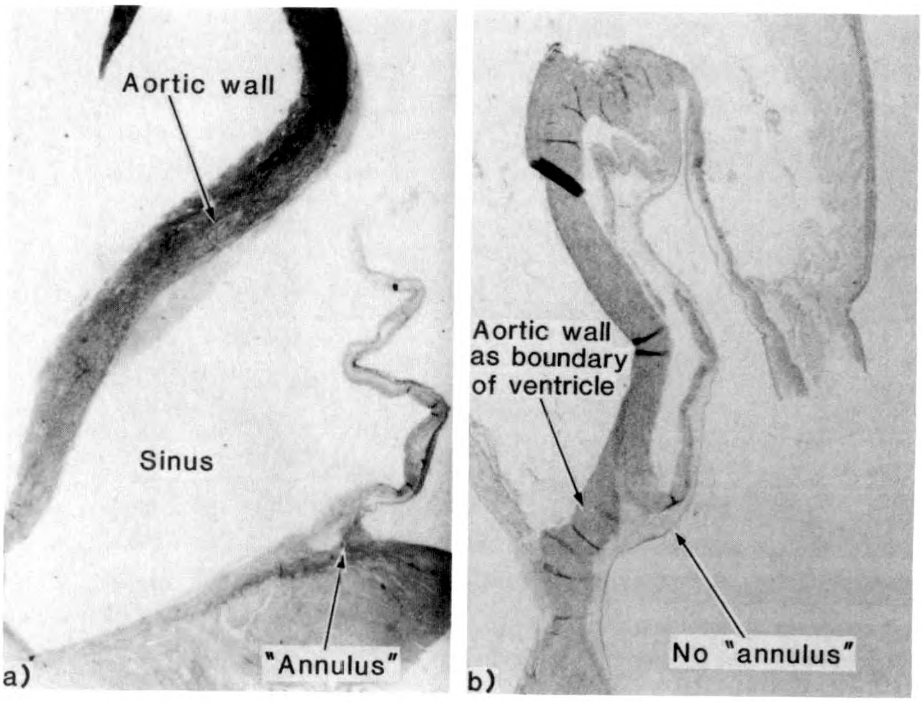

Fig 6. Histological sections showing how a fibrous cord (a) reinforces attachment of the valve leaflet to the ventricular musculature at the base of attachment but (b) becomes incorporated in the wall of the aorta at the level of the peripheral attachment of the commissures.

valve that initially had three leaflets (Fig 8a). Study of the ventricular outflow tract also supports the concept of formation of one vestigial commissure. In the majority of cases [7], there is evidence of three interleaflet triangles, although one of the triangles is rudimentary and is located between the raphe representing the fused commissure in the conjoint leaflet. In a small minority of hearts, however, the valve with two leaflets also possesses only two aortic sinuses, with no evidence of either a raphe in one of the leaflets or a vestigial third triangle within the outflow tract (Fig 8b). Irrespective of whether the valve with two effective leaflets was derived from a template with two or three leaflets initially, it is most unusual for these structures to be stenotic or incompetent in early life. As Edwards [13] pointed out, it is the onset of acquired disease, usually producing gradual fusion of the commissures from the periphery toward the center, that produces problems in valvar function.

The Critically Stenotic Aortic Valve of Infancy

Analysis of a series of such valves from the collection of the Institute of Child Health at the University of Liverpool (McKay R, Smith A, Leung MP, Arnold R, Anderson RH. Morphology of critical aortic stenosis in infancy with reference to haemodynamic function and clinical management. Unpublished observations) shows that the entire pattern of formation of the leaflets is distorted compared with normal. Almost all of the valves show a relatively circular arrangement of attachment of an effectively single leaflet, with full development of only one commissure and one deep interleaflet triangle (Fig 9). Within the solitary leaflet, there is usually evidence of initial (or attempted) formation of additional commissures, a vesti-

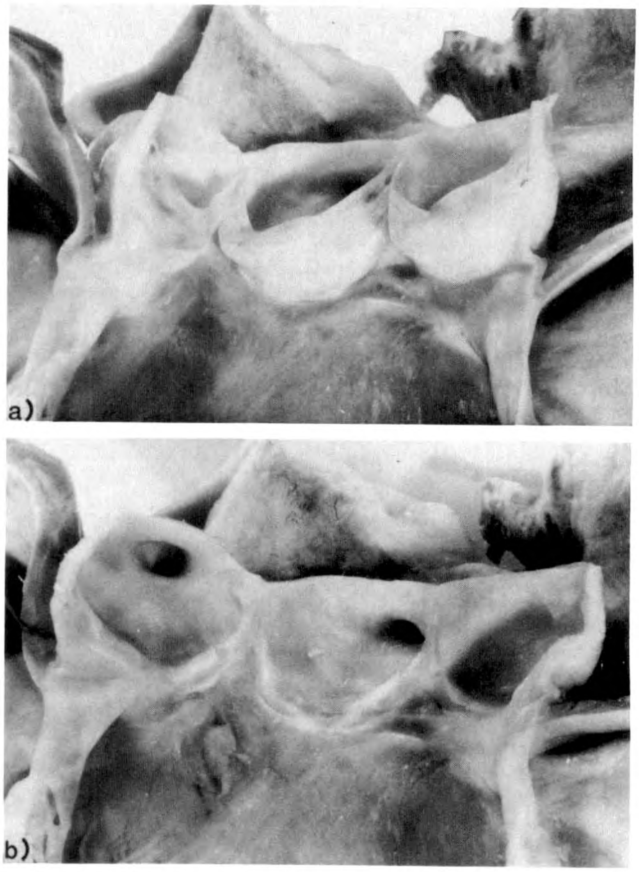

Fig 7. In these preparations of the aortic root, the circlet shown in Figure 1 has been spread to show its full circumference (a) and then the leaflets of the valve have been removed (b).

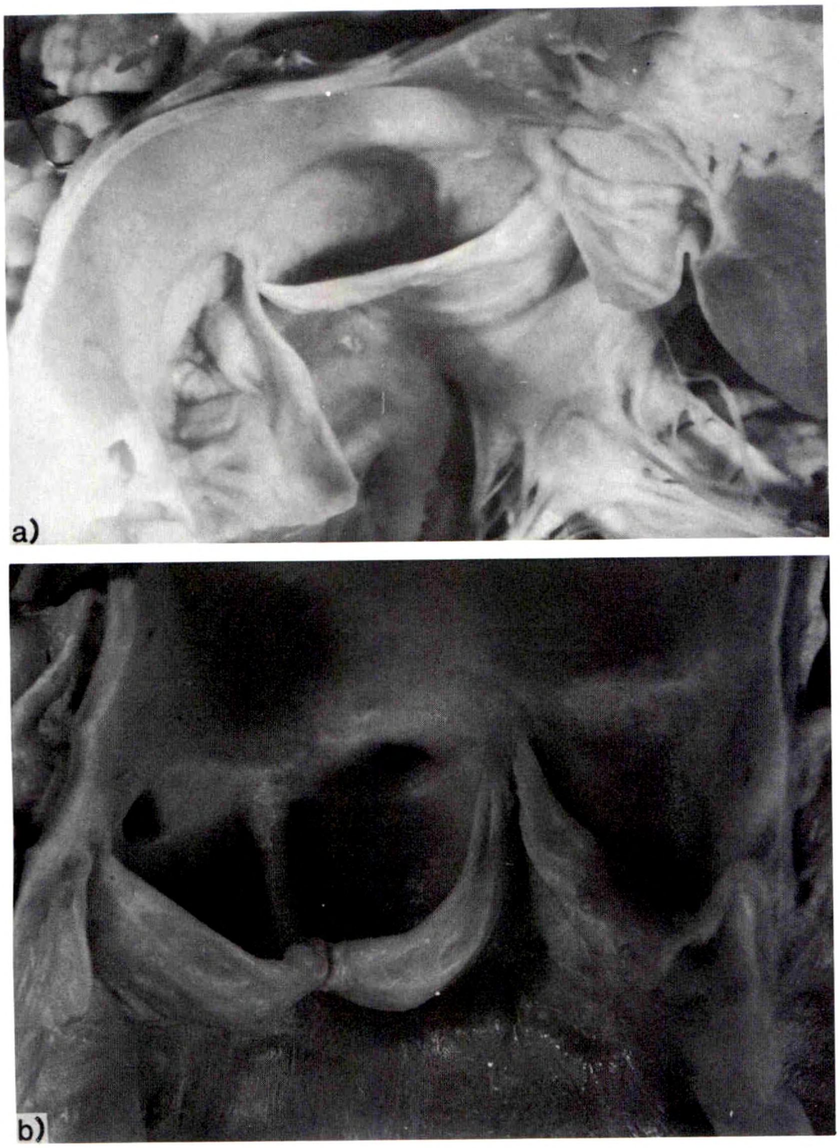

Fig 8. Aortic valves having two leaflets but (a) with one conjoint leaflet and three sinuses and (b) with two leaflets and two sinuses.

gial remnant of an interleaflet triangle being seen on the ventricular aspect. The putative commissures, however, are represented only as folds in the surface of the leaflet and the aortic aspect of the wall of the sinus, dividing the aortic root into three potential sinuses (Fig 10). Only one of these structures is related to a properly formed interleaflet triangle and thus has the potential to function as a true commissure. Usually this commissure extends to the area of fibrous continuity with the mitral valve. It should be emphasized that the folds are on the aortic aspect of the attachment of the leaflet and correspond to raphes within the leaflet. Incision of the leaflet limited to the length of the raphes, however, would extend only to its abnormal ventricular attachment. Such cuts would be unlikely to liberate the leaflet to permit it to open in unobstructed fashion.

Comment

Although the concept of an annulus supporting the valve leaflets is ingrained in the surgical vocabulary and unlikely to be displaced for some time, if ever, it seems to us that its acceptance provides a poor basis for the understanding of normal and abnormal valvar function. It is paradoxical that, among the valves we have examined, those with an almost annular attachment of their leaflets are the most stenotic and produce problems the earliest during life. The understanding of the function of the normal arterial valve must be based on the knowledge that its leaflets are arranged in semilunar fashion and that the commissures between the leaflets function properly only when their peripheral attachment to the arterial wall is well above the basal attachment of the leaflets they separate. Study of the arterial root in this fashion also emphasizes the important discrepancy to be found between the anatomical and hemodynamic ventriculoarterial junctions. This, in turn, highlights the relationships of the triangles found immediately beneath the apexes of the commissures. These areas are formed by the relatively thin wall of the arterial trunk between the arterial sinuses and can be the site of aneurysmal protrusions from the outflow tract [14]. In short, the understanding of normal

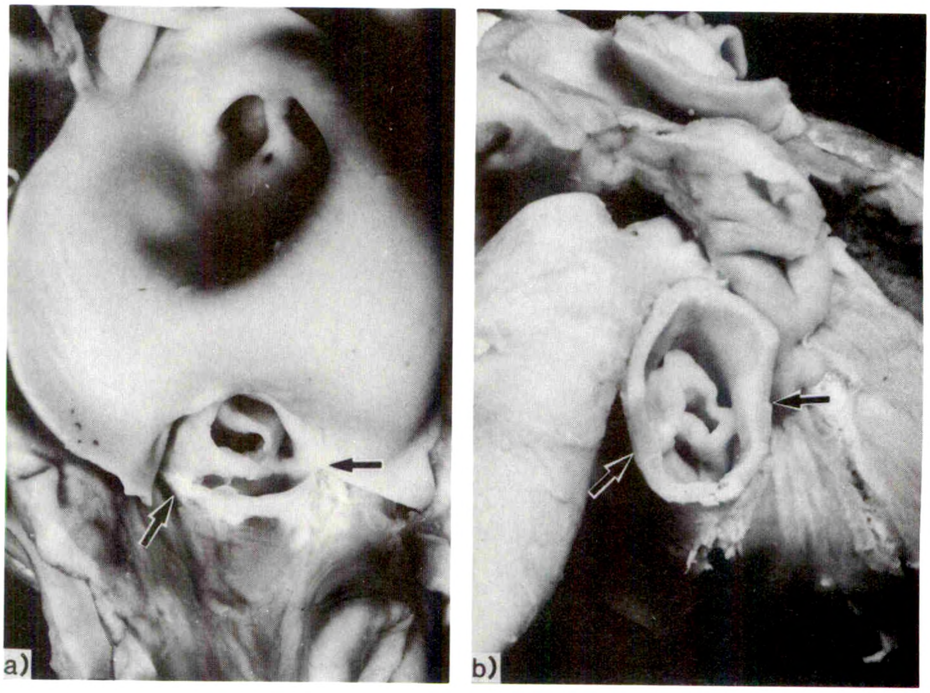

Fig 9. Formation of the so-called unicuspid and unicommissural valve seen most frequently when aortic stenosis is present in infants. The valve has three potential commissures, but two are vestigial, being marked by raphes in the leaflet and by rudimentary triangles on their ventricular aspect.

and abnormal arterial valves is enhanced by the appreciation of the semilunar attachment of the leaflets. Such knowledge would, in our opinion, be advanced by the abandonment of the myth of the valve annulus.

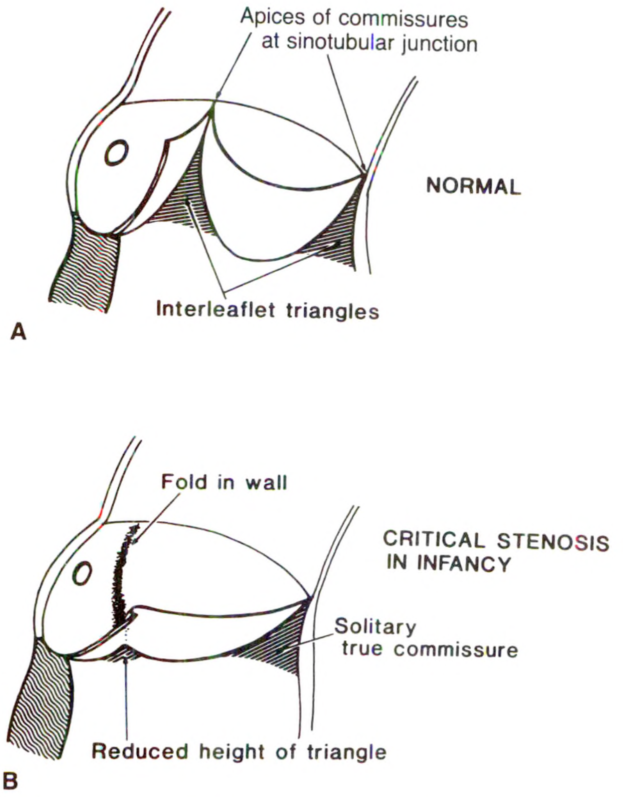

Fig 10. Fundamental differences in arrangement of (a) the normal valve leaflets and (b) the pattern seen in most of the hearts with critical aortic stenosis seen during infancy in the Liverpool study.

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Growth of the Hypoplastic Ascending Aorta After Radical Palliation

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Surgical treatment of hypoplasia of the ascending aorta is a difficult problem. An approach using proximal pulmonary artery to distal aorta bypass with distal pulmonary artery banding was employed in 19 patients with an ascending aorta less than 6 mm in diameter but a patent aortic outflow. There were 11 male and 8 female neonates and infants, and weight ranged from 2 to 4.5 kg (median weight, 3.5 kg). Four patients had transposition of the great arteries, 12 had subaortic stenosis, and 5 had interrupted aortic arch. There was one operative death. Cerebral cyanosis developed in 3 of the 4 patients with transposition of the great arteries and necessitated additional operations within the first year postoperatively. Therefore, palliation with a pulmonary artery to descending aorta conduit and banding was abandoned in such patients. All 14 surviving patients with normally related great arteries had successful palliation and growth of left heart structures. Subsequent procedures included a Fontan/Damus procedure in 1 patient, patch aortoplasty and pulmonary arterioplasty in 5 patients, arch reconstruction with aortic valvulotomy and pulmonary arterioplasty in 2, double arch reconstruction in 2, and simple patch aortoplasty with plans for subsequent pulmonary arterioplasty and ventricular septal defect closure in 1. Results demonstrate that in patients with a hypoplastic ascending aorta, this radical method of palliation can result in growth of right and left heart structures and thus provides the possibility of biventricular repair.

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Surgical treatment of hypoplasia of the ascending aorta presents a difficult and challenging problem. The spectrum of disease ranges from aortic atresia to simple valvular aortic stenosis. Evaluation requires attention to the nature of the lesion itself and the associated problems of hypoplastic inflow to the left ventricle, left ventricular size, and the nature of the outflow obstruction, namely, subaortic, annular, valvular, ascending aortic, or transverse arch hypoplasia or atresia. All of these factors are critical in evaluation of the potential treatment modalities in this spectrum from simple valvular aortic stenosis to hypoplasia of all ventricular and arch structures. Repair is possible in the former, and radical palliation or transplantation is necessary in the latter [1–4].

Simple valvular aortic stenosis was recently examined in a three-institution study [5] of neonates with isolated aortic stenosis with or without coarctation or patent ductus arteriosus. Although multiple methods of cardiopulmonary support were used, overall hospital survival was 87.5%, and late survival (1 to 6 years postoperatively) was 82.5%, with only five reoperations in this early to intermediate follow-up. The diameter of the aortic annulus ranged from 5 to 10 mm (mean diameter, 7 mm); only three diameters were less than 5 mm. However, an ascending aortic diameter less than 6 mm was uncommon, a factor reflecting the general approach of using alternative therapeutic regimens for patients with this hypoplastic arch syndrome.

In a 15-year period at the University of California, San Francisco, 43 neonates with isolated aortic stenosis were operated on using cardiopulmonary bypass for support. Survival was 88%. Over the long follow-up (1 month to 15 years), nine reoperations were performed; they ranged from repeat valvulotomy to valve replacement to a Konno procedure. The results confirm the long-term nature of the disease, even with isolated valvular aortic stenosis. However, substantial growth does occur in a large number of patients [6].

The most difficult group is that in which both valvular aortic stenosis and ascending aortic hypoplasia are evident. In the experience of my colleagues and myself, ascending aortic size is critical in these patients. A long segment of hypoplasia in a neonate who has an ascending aorta less than 6 mm in diameter and who weighs more than 3 kg is incapable of sustaining systemic output. Often in these patients, a small annulus is present with subvalvular or arch hypoplasia and systemic output provided through a ventricular septal defect (VSD), a large pulmonary artery, and a patent ductus arteriosus. Hypoplastic transverse arch, interrupted arch with hypoplastic ascending aorta, and transposition with hypoplastic aorta can also be seen as part of this complex.

Transplantation or radical first-stage palliation for a subsequent Fontan procedure can be used in these patients. However, because they have two adequate ventricles and distal great vessels that are adequate for development of normal anatomy, an approach involving primary arch reconstruction or, more commonly, distal

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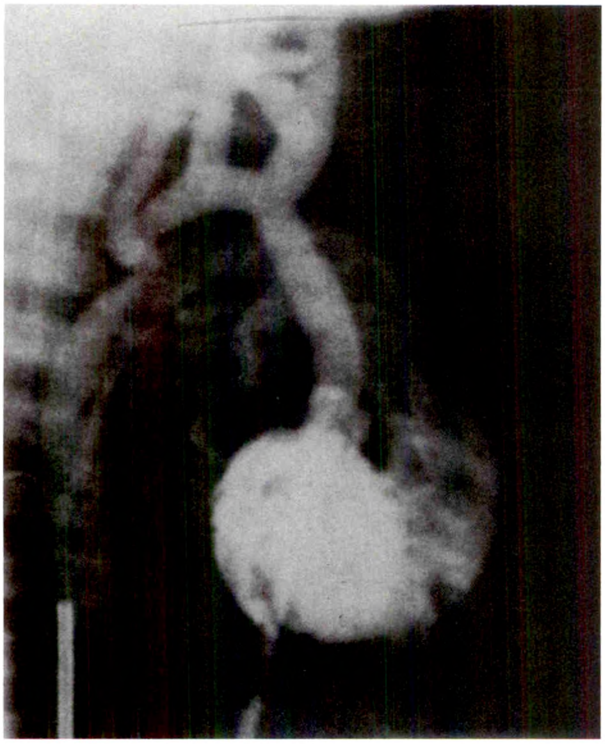

Fig 1. Anteroposterior cineangiogram of hypoplastic arch with ventricular septal defect.

palliation using a pulmonary artery to descending aorta conduit and band to stimulate growth has been used. Our experience with the latter approach is presented here.

Material and Methods

During the period 1981 to 1990, 19 patients with hypoplasia of the aortic arch underwent insertion of a pulmonary artery to descending aorta conduit and pulmonary artery banding (Figs 1, 2). Age ranged from 2 days to 2 months (median age, 5 days). There were 11 male and 8 female neonates and infants, and weight ranged from 2 to 4.2 kg (median weight, 3.5 kg). Hypoplasia of the ascending aorta, that is, a diameter less than 6 mm by echocardiographic assessment, was present in all 19 patients. Four patients had transposition of the great arteries, 12 had subaortic stenosis, and 5 had interrupted aortic arch.

A left thoracotomy was performed in each patient, providing access to the proximal pulmonary artery, ductus arteriosus, and distal aorta. A conduit was constructed from the proximal pulmonary artery to the distal aorta using a Gore-Tex graft. Graft size ranged from 5 to 8 mm (mean size, 6 mm) (Fig 3). Partial occlusion of the proximal and distal vessels was employed, and after completion of arch reconstruction, ligation of the ductus arteriosus was performed. The distal pulmonary artery was then banded to a saturation of 0.85, with care taken to suture the band proximal to the right pulmonary artery origin (Fig 4).

Results

There was one operative death. An infant weighing 2.4 kg died of sedation-induced hypotension and arrest at the completion of the procedure (Fig 5). The other 18 patients underwent successful proximal pulmonary artery to descending aorta bypass with distal pulmonary artery banding. Operation was performed in 12 patients with subaortic stenosis, 4 patients with transposition of the great arteries, and ony 5 patients with interruption of the aortic arch. The standard approach in this latter group is direct anastomosis and VSD closure through a median sternotomy when the ascending aorta is greater than 5 mm. A diminutive ascending aorta was encountered in 5 patients, 4 of whom had both subclavian arteries arising from the distal aorta. For patients with transposition, it was recognized early that cerebral cyanosis occurred with this approach. Three of the 4 required multiple reoperations in the first year of life, including a Senning procedure, an attempt at arterial switch, and atrial septectomy. Therefore, in 1985, this method of repair was abandoned in patients with transposition. Since then, arch reconstruction with a primary arterial switch procedure has been the method of choice for this group, as it allows reconstruction of the pulmonary artery before distal obstructions can occur [7].

Successful palliation with growth of the left heart struc-

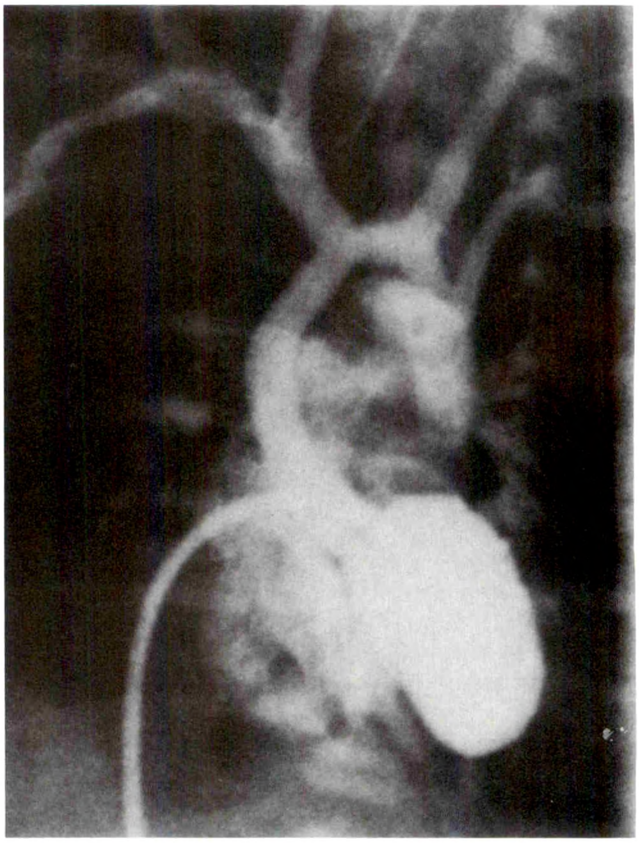

Fig 2. Lateral cineangiogram of hypoplastic arch with ventricular septal defect and large pulmonary artery.

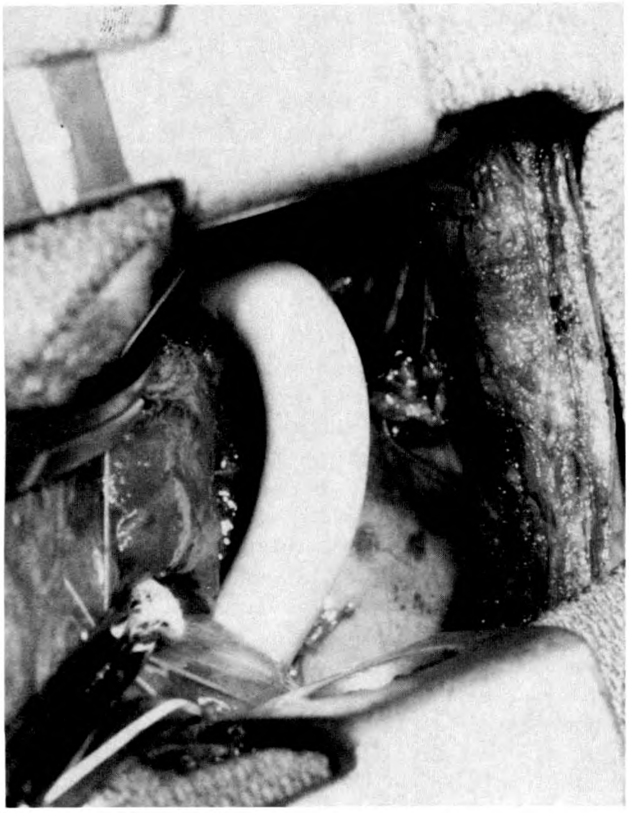

Fig 3. Pulmonary artery to descending aorta conduit.

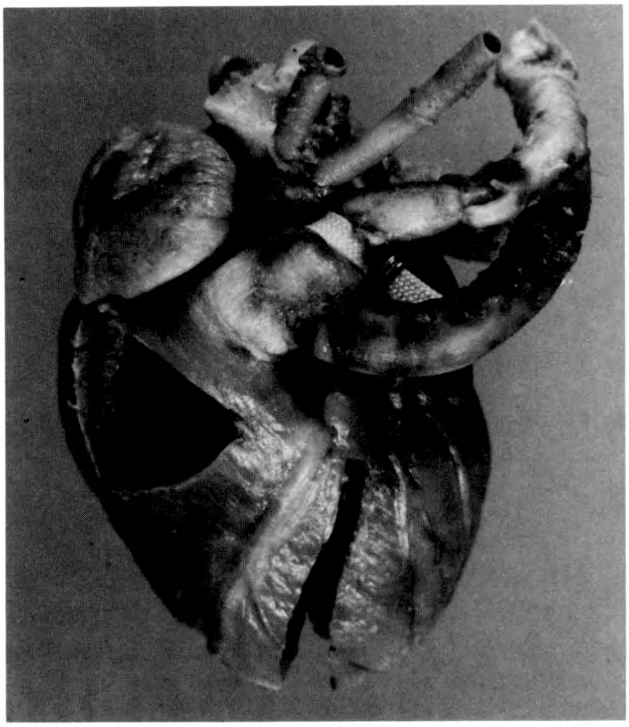

Fig 5. Autopsy specimen demonstrating proximal pulmonary artery to descending aorta graft, distal pulmonary artery band, and hypoplastic ascending aorta.

tures has been noted in all 14 of the surviving patients with normally related great arteries despite the fact that subaortic stenosis was present in 12 (Fig 6). Reconstructive procedures in 11 patients to date have included a Fontan/Damus procedure in 1 patient 3 years postoperatively; patch aortoplasty and pulmonary arterioplasty in 5 patients; arch reconstruction, aortic valvulotomy, and pulmonary arterioplasty in 2 patients; double arch reconstruction in 2; and simple patch aortoplasty with plans for subsequent pulmonary arterioplasty and VSD closure in 1

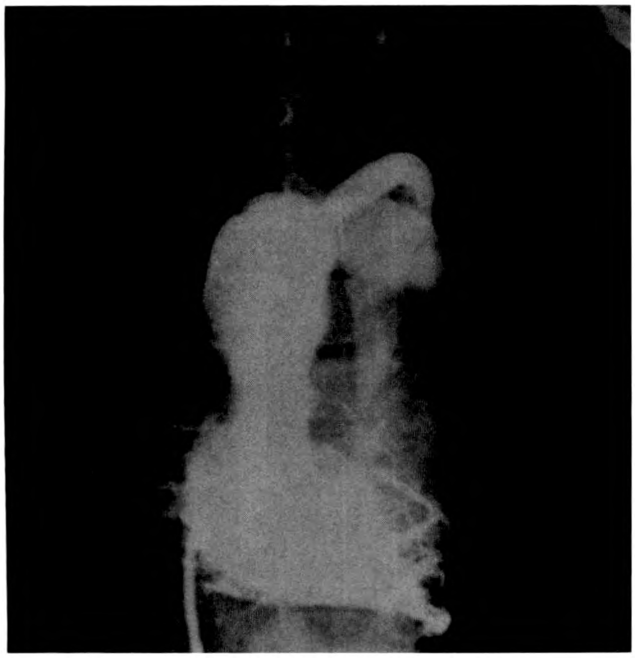

Fig 4. Cineangiogram demonstrating proximal pulmonary artery to descending aorta conduit.

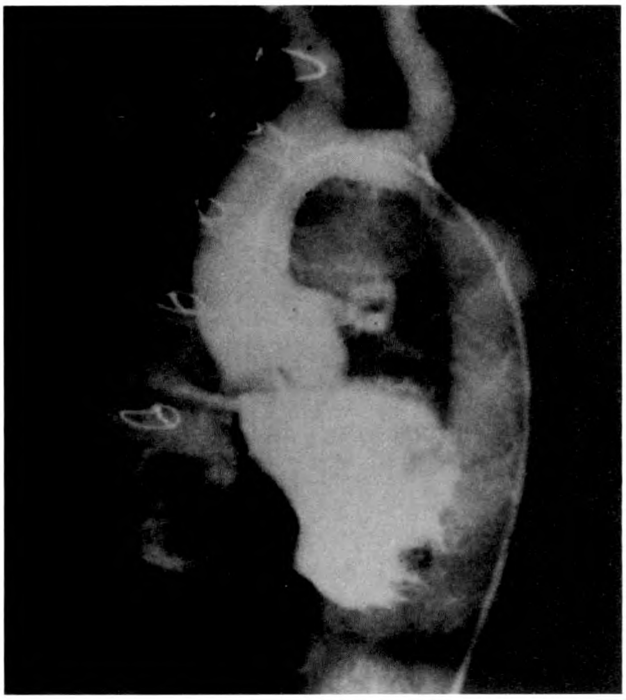

Fig 6. Late postoperative arch development with residual coarctation and small ventricular septal defect.

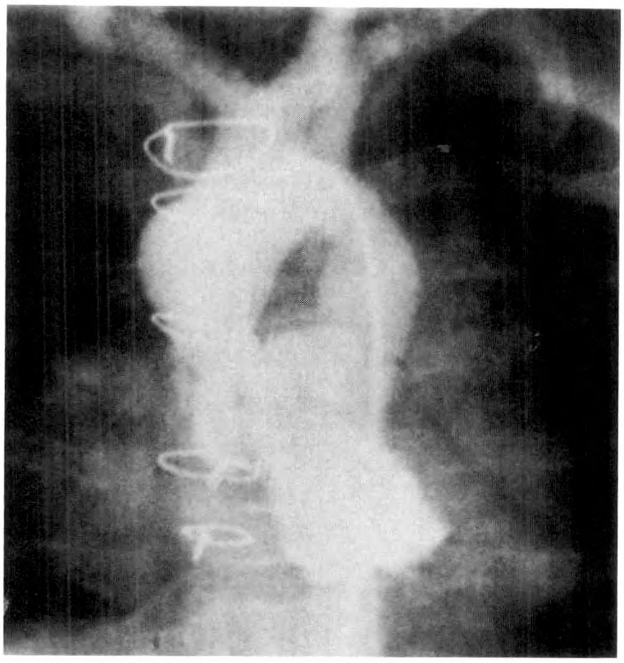

Fig 7. Double-arch configuration with growth of the ascending aorta as well as the native arch structures.

(Fig 7). In these patients, growth occurred due to progressive obstruction of the pulmonary artery outflow secondary to the fixed constriction in its size by the band as well as the conduit size and VSD closure (Fig 8). This result is consistent with the theoretical advantages of this approach. The regression of subaortic stenosis in this setting has been previously documented by Ilbawi and associates [8] and is corroborated by the current study.

Comment

Treatment of patients with hypoplasia of the ascending aorta and transverse arch is a difficult clinical challenge. Ascending aortic hypoplasia is part of a spectrum of disease characterized by multiple other defects. In many centers, these problems are combined, and patients with simple valvular aortic stenosis are included with all patients having variants of hypoplastic left heart syndrome. In fact, the two conditions represent the ends of the broad spectrum of this disease. A recent study [5] involving one center each from the West Coast, the Midwest, and the East Coast demonstrates that a low risk for surgical intervention in neonates with isolated aortic stenosis is possible. Conversely, it has been shown that patients with hypoplastic left heart syndrome are best treated by radical palliation or transplantation [3, 4]. If a major VSD exists, a biventricular repair using the arch reconstruction concepts of the Norwood procedure and a right ventricle to pulmonary artery conduit as described by Austin and co-workers [9] is possible. However, a substantial number of patients exist in the middle; they have an acceptable left ventricle, a small but adequate aortic annulus and adequate valve structure, a hypoplastic but potentially acceptable ascending aorta and transverse arch, and ductal dependence. In these patients, radical palliation or transplantation provides an unacceptable long-term operative approach, as they have the potential for normal biventricular anatomy. An approach that would facilitate aortic growth and subsequent biventricular reconstruction seems optimal. In patients with transposition of the great arteries, this can be achieved by an arterial switch and pulmonary artery reconstruction [7]. In patients with a large VSD and ventricular development, use of the pulmonary valve for aortic outflow with a Damus arch reconstruction and right ventricle to pulmonary artery conduit provides excellent results [9].

Proximal pulmonary artery to descending aorta bypass with distal pulmonary artery banding has, in our experience, provided stimulation to growth of both ventricular and arch structures, and has led to regression of subaortic stenosis in the other patients in this disease spectrum. Aortic size and transverse arch size have increased, making reconstruction possible and allowing maximal development of normal anatomy. The Achilles' heel of this approach has been the right pulmonary artery. In each case, relative obstruction has occurred and necessitated patching of the right and left pulmonary arteries as well as the banded area. However, development of the left heart structures has more than compensated for this deficiency in the approach. Late results demonstrate that normal right and left heart reconstruction is possible and that normal development of the subaortic region can occur [8]. In patients with hypoplasia of the ascending aorta in the setting of normal inflow and normal ventricular structure,

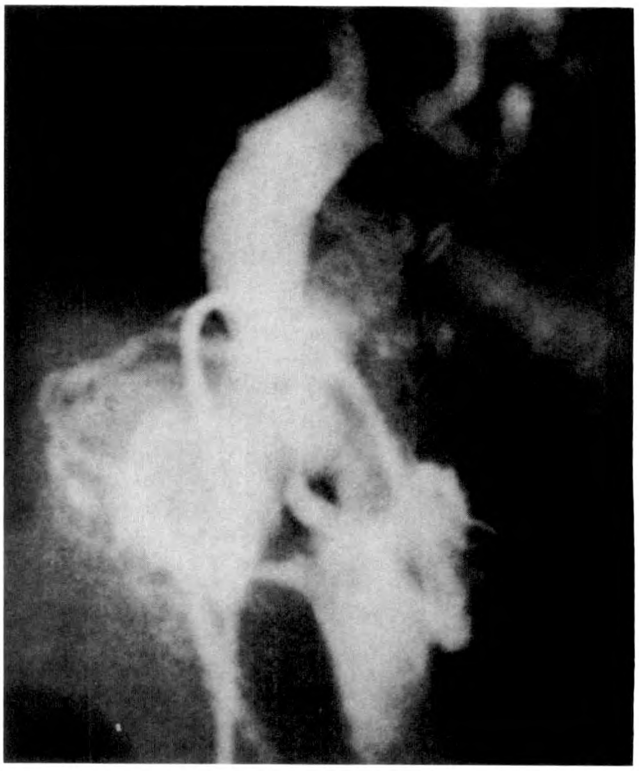

Fig 8. Closure of muscular ventricular septal defects with regression of subaortic stenosis.

treatment is possible using this aggressive approach, and normal biventricular development can occur.

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COMMENTARY: Aortic Valvotomy Using Inflow Occlusion

Critical aortic stenosis in the neonate continues to be a defect with substantial morbidity and mortality despite the improved ability to detect patients with this defect and despite improved resuscitative measures such as administration of prostaglandin E_1. A variety of methods have been used to enlarge the opening for the aortic valve, and it is my purpose here to briefly describe a technique for performing aortic valvotomy under inflow occlusion in the neonate. It is noteworthy, however, that this technique is rarely employed in our institution in the current era, as balloon dilation of the aortic valve in the catheterization laboratory has become the standard method of treatment of the neonate with critical aortic stenosis. The willingness on the part of the surgeons in our group to support the closed technique in the catheterization laboratory, however, was based in part on our experience with valvotomy under inflow occlusion and, in particular, with the remarkable improvement in clinical condition that was achieved with seemingly small increases in the size of the aortic valve orifice. Therefore, the technique as previously employed will be described, although it should also be noted that the discussion applies only to the neonate and not to the child beyond the first month of life.

Method

Preparation for Operation

Preoperative resuscitation to establish normal acid-base status and stable function of other organ systems including the kidney and liver is important in achieving a satisfactory outcome in these patients regardless of the operative approach. If patency of the ductus arteriosus can be established with prostaglandin E_1, resuscitation becomes remarkably simplified, as the right ventricle can contribute to the systemic output in the same way as in patients with hypoplastic left heart syndrome. Inotropic support with dopamine hydrochloride or dobutamine hydrochloride or both will also improve the perfusion of noncardiac organ systems, and it is generally possible to fully resuscitate the patient before operation.

Once resuscitated, the patient is taken to the operating room and prepared with a reliable arterial monitoring catheter in either the radial or femoral artery and with at least two large intravenous catheters for medication and fluid administration. Extremely close communication and cooperation between surgeon and anesthesiologist are fundamental to achieving satisfactory results with the closed technique, and reliable monitoring and intravenous access are crucial.

Operative Technique

After anesthesia is induced, a median sternotomy is carried out, and the thymus is resected to improve exposure. The pericardium is opened in the midline, and the superior vena cava is loosely encircled with a ligature passed through a Rumel tourniquet. The inferior vena cava is controlled in similar fashion. The manipulation of the heart necessary to encircle the inferior vena cava can produce ventricular arrhythmias (including fibrillation), and an alternative method to control the inferior vena cava is to use a vascular clamp. The aorta and pulmonary artery are separated, and traction sutures are placed in the ascending aorta at the proximal and distal ends of the proposed longitudinal aortotomy. Two separate suction devices are prepared, and paddles for defibrillation should be available on the operative field from the beginning of the procedure.

At this point, the proposed subsequent sequence of events is reviewed for all members of the surgical and anesthesia teams. The aorta is partially occluded with a spoon-shaped vascular clamp encompassing the previously placed traction sutures, and the aorta within the clamp is incised (Fig 1). Additional retraction sutures are placed on either side of the aortotomy. The superior vena cava is occluded with its tourniquet, and the inferior vena cava is occluded with its tourniquet or with an angled vascular clamp. Several rapid inflations of the lungs are given to empty blood from the pulmonary circulation, and when the heart appears to be empty and the arterial pressure trace is flat, the aorta is cross-clamped at the level of the innominate artery. The side-biting clamp on

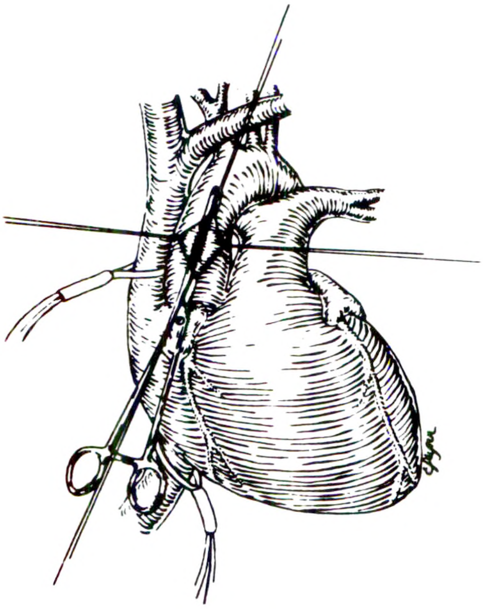

Fig 1. A partial occlusion clamp is put on the ascending aorta, the excluded segment is incised, and traction sutures are placed.

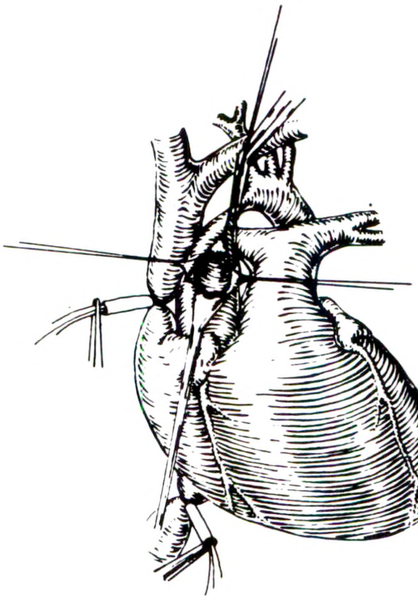

Fig 2. The aorta is cross-clamped after the cavae have been snared and the heart has been emptied. The valvotomy is quickly performed through the aortotomy.

the aorta is removed, and blood is suctioned from the aortic root (Fig 2).

The aortic valve is visualized and can be either dilated with a Jacobsen mosquito clamp or carefully incised along a line of commissural fusion. The stenotic valve in a neonate is frequently quite deformed with thickened dysplastic leaflets, and it is important not to attempt to open the valve too widely because of the risk of producing aortic valve insufficiency. (Because flow is a function of the fourth power of the radius, a relatively small improvement in orifice size results in a major improvement in flow and pressure gradient.) The clamp or tourniquet on the inferior vena cava is removed, allowing flow through the heart and lungs to refill the left ventricle and to evacuate air from the aortic root. The retraction sutures on the aortotomy are pulled up, the aortotomy is controlled with a spoon-shaped vascular clamp, and the aortic cross-clamp is removed. The aortotomy is then repaired with running monofilament suture (Fig 3).

As inflow occlusion is occurring, sodium bicarbonate, 1 mEq/kg of body weight, is given to offset the anticipated transient metabolic acidosis from systemic hypoperfusion. Intravenous inotropic support with calcium and epinephrine is sometimes necessary when perfusion is reestablished, and blood aspirated during the valvotomy must be rapidly replaced with warmed bank blood. It has generally been possible to carry out the valvotomy with 2 to 2½ minutes of inflow occlusion.

Results

As mentioned previously, this procedure has virtually disappeared from the logs of the cardiovascular surgical service at The Children's Hospital, Boston, despite a total of more than 950 cardiac procedures in 1990. The results of inflow occlusion valvotomy up to 1984, when the program of balloon valvotomy in the catheterization laboratory was begun, have been reported by Zeevi and colleagues [1]. Between 1978 and 1984, 14 neonates (mean age, 9 days) with critical aortic stenosis underwent valvotomy by the inflow occlusion technique. All survived the operation, but 4 died within 30 days, 2 of whom were determined at postmortem examination to have left ventricles less than 65% of normal volume. There were 2 late deaths, 1 patient had repeat surgical valvotomy, and 1 had late balloon dilation of recurrent aortic stenosis.

Comment

The techniques of inflow occlusion valvotomy, balloon dilation in the catheterization laboratory, and transven-

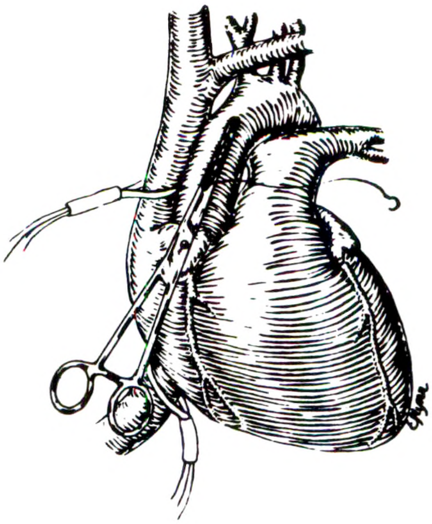

Fig 3. The partial occlusion clamp is reapplied and the cross-clamp and caval snares are released. The aortotomy is sutured.

tricular dilation are all more or less "blind" techniques in which the valve orifice is enlarged by dilation of the native orifice. In the neonate, these valves are generally quite dysplastic, and it seems that considerable "maturation" must occur postnatally. In patients seen late after neonatal valvotomy for what were termed dysplastic valves, the valves generally have "matured" into structures that are quite typical of congenital aortic stenosis. Therefore, as long as the valve is not made seriously regurgitant in the neonatal period, the precision of the technique by which its orifice is enlarged is less critical than minimizing the physiological insult of the operation or intervention. Follow-up data from the natural history study, which suggest that all aortic valve operations ultimately are palliative, further support this concept. Only in patients who cannot be resuscitated by establishing ductal patency or through moderate inotropic support does it seem that support on bypass offers any major advantages over one of the closed techniques.

Although it is beyond the scope of this presentation, perhaps the more important question is the determination of criteria by which the capacity of the left ventricle and aortic outflow tract to serve as the long-term pathway for systemic output can be predicted. This predictive ability would allow more appropriate selection of patients for a two-ventricle valvotomy approach versus the approach of creating a single-ventricle circulation leading to a Fontan repair. One such effort from this institution has been made [2], although the accuracy of the predictions remains at only the 90% level. Important echocardiographic variables appear to be mitral valve area (<4.75 cm^2/m^2), long-axis dimension of the left ventricle relative to long-axis dimension of the entire heart (<0.8), and diameter of the aortic root indexed to body surface area (<3.5 cm/m^2). None of the 12 patients with two or more measurements lower than these limits survived valvotomy.

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COMMENTARY: Transventricular Aortic Valve Dilation

Neonates with critical aortic valve stenosis are usually admitted with severe circulatory collapse. Fifty percent of these infants have major associated cardiac lesions, the most lethal of which is the triad of a small mitral valve, small left ventricle, and endocardial fibroelastosis.

Frustrated by the high operative mortality in these infants, my colleagues and I [1] undertook a trial of closed transventricular aortic dilation between 1980 and 1985. The closed technique described by Trinkle [2] offered a rapid, simple method of opening the aortic valve with minimal trauma to the infant. However in our experience with 18 infants in whom we used this technique, there was no improvement in operative survival (9 of 18). Further, the early reoperation rate (54%) was greater than in the preceding 22 infants treated by an open technique.

Consequently, since 1985 we have returned to an open aortic valvotomy with the child supported by normothermic cardiopulmonary bypass, a procedure in which survival has improved to 75% (15 to 20 neonates). Bypass can be very helpful in stabilizing these infants, in correcting acid-base imbalance, and in temporarily improving myocardial perfusion. We agree with Turley and associates' [3] conclusion that bypass is useful in these infants. At present, survival of neonates with critical aortic stenosis is clearly dependent on the presence or absence of major associated lesions, the most threatening of which is a small left heart. We believe that infants with isolated critical aortic stenosis can now be managed quite successfully by open valvotomy during a short period of cardiopulmonary bypass. However, those infants with major associated cardiac lesions should be considered for either heart transplantation or the Norwood operation. The impressive results described by Turley (*vide supra*) in a small but important subset of infants with two ventricles, a hypoplastic ascending aorta (less than 6 mm), and frequently subaortic stenosis provide an additional method of managing these critically ill newborns.

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Surgical Management of Diffuse Subaortic Stenosis: An Integrated Approach

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An integrated approach to the surgical management of diffuse subaortic stenosis has been designed to provide adequate relief of left ventricular outflow tract obstruction whatever the anatomical features encountered at operation. This approach was used in 22 patients with tunnel subaortic stenosis (19 patients) or diffuse hypertrophic obstructive cardiomyopathy (3 patients). The obstructive tissue was resected through an aortoseptal approach. In 18 patients, associated hypoplasia of the aortic orifice necessitated aortic valve replacement using the Konno procedure; in 4 patients with a normal-sized aortic orifice, the native aortic valve was preserved. There were two early deaths and one late death (all after a Konno operation). Long-term adequate relief of left ventricular outflow tract obstruction was achieved in all survivors. Operation for diffuse subaortic stenosis should be performed with two main goals: (1) to obtain complete relief of the left ventricular outflow tract obstruction by the appropriate procedure and (2) to preserve the native aortic valve whenever possible, particularly in young patients.

(Ann Thorac Surg 1991;52:654–62)

The surgical management of diffuse subvalvar aortic stenosis remains a therapeutic challenge. In the localized form of subaortic stenosis, resection of the stenotic segment (with or without associated myectomy) through the aortic orifice usually provides adequate and long-lasting relief of the left ventricular outflow tract (LVOT) obstruction [1–6]. In diffuse subvalvar stenosis, on the other hand, the conventional procedures often provide incomplete resection of the stenotic segment and offer less than satisfactory long-term results [1, 7–10]. A more aggressive surgical approach is mandatory.

The Spectrum of Diffuse Subaortic Stenosis

It is usual to differentiate the fixed or fibromuscular form of diffuse subaortic stenosis (tunnel subaortic stenosis) from the purely muscular form or hypertrophic obstructive cardiomyopathy (HOCM) (Fig 1). There is some evidence, however, that suggests that these two forms of diffuse subaortic stenosis may represent two clinical manifestations of the same genetically transmitted disease. Some patients with fixed subaortic stenosis have excessive hypertrophy of the septum (in contrast with thickening of the posterior left ventricular wall) and muscle fiber disorientation [7], features typical of HOCM [11, 12]. Clinically, in tunnel subaortic stenosis, there may be abnormal systolic anterior mitral leaflet motion and an abnormal postectopic response, again typical findings in HOCM [1]. Hypertrophic obstructive cardiomyopathy is a genetically determined disorder that is transmitted as an autosomal dominant trait [13], but a familial incidence of fixed subaortic stenosis has been reported as well [7, 14].

Even if nosologically relevant, the differentiation between tunnel subaortic stenosis and diffuse HOCM does not seem justified from a purely surgical point of view. All forms of diffuse subaortic stenosis require complete relief of the obstruction, and the same surgical problems must be solved whatever the anatomical nature of the obstacle.

In most instances, diffuse subaortic stenosis is easy to distinguish preoperatively and intraoperatively from the more common localized form of the disease. However, in some cases, the differentiation is not as evident. The anatomical variability in patients with tunnel subaortic stenosis has been clearly emphasized [7]. Typically, these patients have a diffuse fibromuscular narrowing of the LVOT, an abnormally small aortic annulus (even though the aortic valve cusps are normally formed), concentric thickening of the ventricular wall, and a mitral valve normally positioned in the left ventricular cavity. Other patients with a typical tunnel at the subvalvar level may demonstrate disproportionate ventricular septal thickening, abnormal systolic anterior mitral leaflet motion, a mitral valve positioned anteriorly in the left ventricular cavity, and a normal-sized aortic annulus. These features fall between typical tunnel stenosis and typical HOCM. In other forms of fixed subaortic stenosis, the stenotic segment can be shorter and the aortic annulus, of normal size. Then the morphology resembles that of the most severe forms of localized discrete subvalvar aortic stenosis [15].

In purely muscular HOCM, the obstructing muscle mass can be localized at the subvalvar level or can be very large and extend well down into the body of the left ventricle. It can even be largest at a site below the anterior mitral leaflet, producing a so-called midcavity obstruction [16]. Most patients with HOCM are currently successfully managed with medical treatment: β-blockers and calcium-

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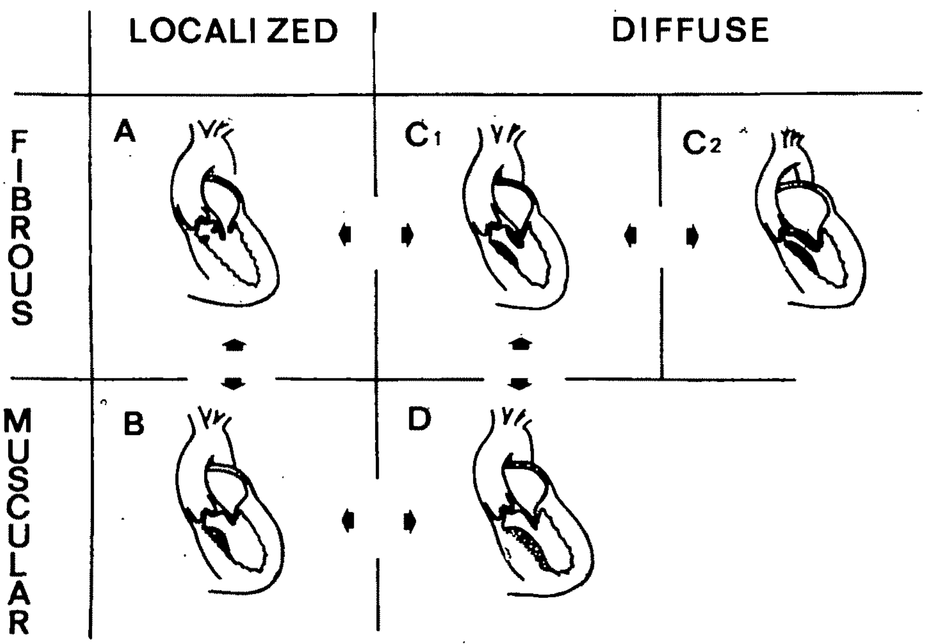

Fig 1. The spectrum of subaortic stenosis: (A) localized (discrete) fibrous stenosis; (B) localized hypertrophic obstructive cardiomyopathy (HOCM); (C) tunnel subaortic stenosis with (C1) normal-sized aortic annulus and (C2) hypoplastic aortic annulus; and (D) diffuse HOCM. Arrows indicate the presence of transitional forms between two subsets.

channel blockers. Surgical intervention is advocated only for patients who have persistent symptoms or a marked left ventricular–aortic gradient at rest under optimal medical therapy. It is therefore suggested that operation be performed in the most anatomically severe cases of HOCM.

Some patients who have had surgical excision of a discrete subvalvar stenosis require subsequent reoperation for what is often described as a tunnel stenosis [10, 17, 18]. The rate of "recurrence" is high in some series [18]. Controversy exists regarding whether the obstruction is recurrent or residual. However, it is likely that, in most cases, the initial procedure was inadequate and the initial obstruction was incompletely relieved. This finding emphasizes the difficulty in differentiating, even intraoperatively, a localized obstruction from a more diffuse one and points out the need to obtain complete relief of the subaortic obstruction to lessen the risk of recurrence.

Finally, it should be remembered that a subaortic obstruction can coexist with obstruction at the valvar or supravalvar level.

In summary, before and during an operation for subaortic stenosis, two points must be kept in mind.

1. The anatomical features of the LVOT obstruction must be perfectly delineated so as to answer several questions. Is the subaortic stenosis localized or diffuse? Is the stenotic segment limited to the subvalvar area, or does it extend to the valvar or supravalvar level? Is the aortic annulus of normal size or hypoplastic? Are the aortic valve cusps normal or abnormal?
2. Adequate relief of the LVOT obstruction must be obtained by an appropriate surgical procedure to lessen the possibility of a subsequent reoperation.

The Surgical Management of Diffuse Subaortic Stenosis: An Integrated Approach

The basic concept of this approach is to provide an adequate surgical procedure in all instances based on the intraoperative findings (Fig 2). The operation is performed through a median sternotomy. The left ventricular and aortic pressures are measured to evaluate the importance of the left ventricular–aortic systolic gradient. This measurement serves as a reference to assess the effectiveness of the procedure at the end of the operation. Cardiopulmonary bypass is established using two caval cannulas and caval snares, as the right ventricle may be opened. A sump sucker is placed in the left atrium through the right superior pulmonary vein.

During core cooling to 25°C, the ascending aorta and the pulmonary trunk are dissected apart as close as possible to the aortic annulus, thus exposing the upper part of the left anterior fibrous trigone (the small triangle of fibrous tissue located between the left and right aortic annuli and the muscular septum [19]). The aorta is cross-clamped, and cold cardioplegic solution is given. A longitudinal incision in the aorta is carried obliquely down toward the top of the commissure between the right and left aortic cusps (top of the left anterior fibrous trigone), with care taken to stay away from the left and right coronary ostia (one of which may be abnormally located). The aortic cusps are retracted, and the subvalvar area is fully examined.

Situation 1: Localized Stenotic Segment

In this situation, the stenotic segment can be resected through the aortic orifice whether it is fibrous, fibromuscular [1], or purely muscular [11, 20].

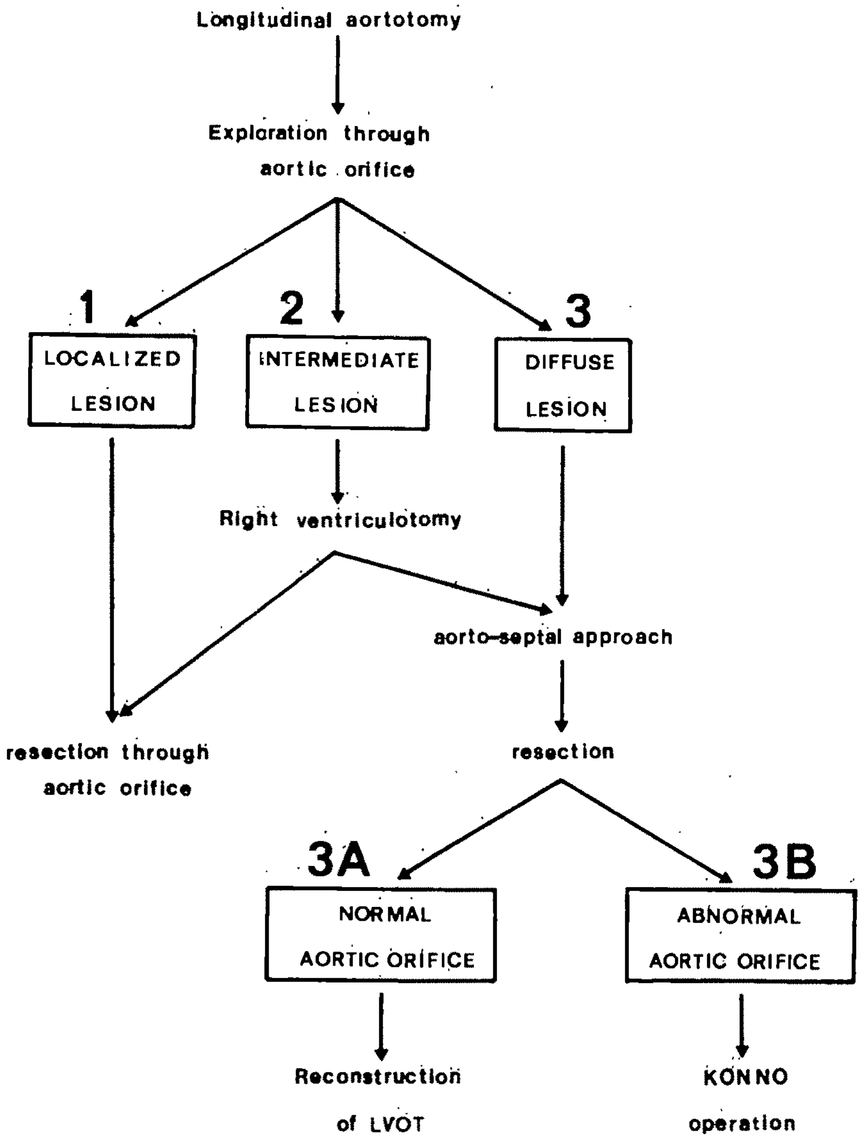

Fig 2. The integrated approach to the surgical management of subaortic stenosis. (LVOT = left ventricular outflow tract.)

Situation 2: Intermediate Stenotic Segment

In this case, the stenotic segment is longer or extends more deeply into the LVOT. The right ventricular infundibulum is opened transversely below the pulmonary valve, care being taken to avoid damaging the pulmonary valve cusps. A finger is placed into the right ventricular cavity and pressed against the right aspect of the interventricular septum, which brings the subvalvar area into better view through the aortic orifice. If complete resection of the obstruction is possible, it is done; if not, the procedure proceeds to situation 3.

Situation 3: Diffuse Stenotic Segment

The aortoseptal approach is completed (Figs 3, 4) [21, 22]. The aortic and infundibular incisions are joined together at the top of the left anterior fibrous trigone (which is made easier by the previous dissection of the roots of both the aorta and the pulmonary trunk).

The aortic ring is divided through a vertical incision of the left anterior fibrous trigone. Great care is taken to avoid injury to the adjacent aortic leaflets. The anatomy of the commissure and the shape of the left anterior fibrous trigone are variable (see Fig 4). In some cases, the trigone is wide and not very high (see Fig 4A), and the cusps move away from each other rapidly. In these instances, division of the trigone is easy. More often, the trigone is higher and narrower (see Fig 4B), and the cusps are in contact with each other through a distance of a few millimeters. However, it has been shown experimentally and clinically that it is always possible to incise the aortic annulus without injury to the adjacent aortic valve leaflets [21, 22].

Stay sutures are placed at the top and at the bottom of the left anterior fibrous trigone; they will serve as guidelines during the reconstruction of the aortic orifice. The muscular septum is incised, first transversely below the pulmonary valve and then obliquely toward the left ventricular apex, with the incision kept away from the

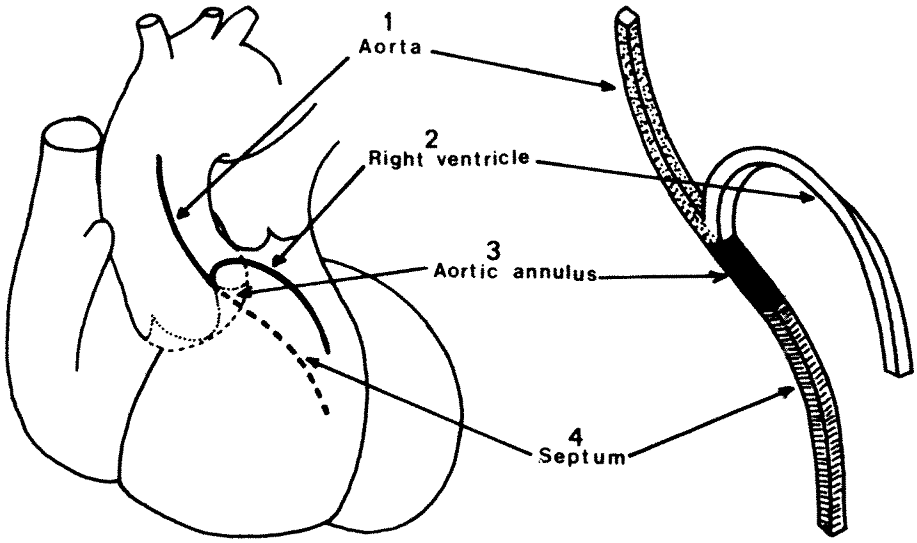

Fig 3. The incisions performed during the aortoseptal approach: (1) longitudinal aortotomy; (2) right ventricular infundibulotomy; (3) division of aortic annulus (left anterior fibrous trigone); and (4) septotomy. See text for details.

conduction pathways (see Fig 4). The septotomy must be carried far enough toward the apex to get below (or upstream to) the stenotic subaortic segment. The subvalvar area is thus exposed widely, and the obstructing tissue (fibrous or muscular or both) can be easily and adequately resected from each edge of the septal incision, providing important widening of the LVOT (Fig 5). If the bundle of His follows its normal course, atrioventricular block should be avoided by not removing muscle too deeply from the inferior edge of the septal incision. At this point, two distinct situations exist (see Fig 5).

SITUATION 3A: NORMAL-SIZED AORTIC ANNULUS AND NORMAL AORTIC VALVE CUSPS. This is the case in patients with diffuse HOCM and in some patients with tunnel subaortic stenosis. The LVOT can be reconstructed without implantation of foreign material.

The muscular septum is closed using interrupted pledgeted mattress sutures. The bites in the septum are large to obtain a strong septal closing, but they are not very deep to preserve the widening of the subaortic area that has been obtained by the resection of the obstructing tissue (see Fig 5). The left anterior fibrous trigone is carefully reconstructed with a running suture. Fine bites are taken to obtain a normal commissural anatomy without any narrowing of the aortic orifice. The right ventricular and aortic incisions are closed.

SITUATION 3B: HYPOPLASIA OF AORTIC ANNULUS OR LESIONS OF AORTIC VALVE CUSPS (CONGENITAL STENOSIS OR SEVERE AORTIC INCOMPETENCE) OR ASSOCIATED SUPRAVALVAR STENOSIS. This is the case in most patients with a tunnel form of diffuse subaortic stenosis. The operation is completed using the aortoventriculoplasty concept (Konno-Rastan procedure).

The aortic valve leaflets are resected. The newly enlarged aortic annulus is sized, and an oval-shaped patch is appropriately sized and trimmed. We use a double-velour woven Dacron patch lined with preserved heterologous pericardium, the pericardium being placed on the left ventricular side. Beginning at the inferior angle of the septal incision, the patch is sewn in place onto the edges of the septotomy. Interrupted mattress sutures are passed through the patch, through the depth of the interventricular septum, and then through Teflon felt buttresses and are tied on the right ventricular side. The patch is then sutured to the level of the aortic annulus. An appropriately sized valve prosthesis is chosen and sewn in simple interrupted fashion to the posterior native portion of the annulus, which makes up 50% to 65% of the enlarged annulus. Horizontal mattress sutures are used to secure the remainder of the prosthetic valve sewing ring to the septal patch. The upper portion of the patch is sutured in place to enlarge and close the aortotomy.

The defect remaining in the right ventricular outflow tract is closed using an appropriately trimmed triangle-shaped Dacron patch lined with pericardium on the right ventricular side. This patch is secured to the septal patch a little above the line suturing the prosthetic valve to that septal patch. Particular care is taken in suturing at the junction between the right ventricle and the aorta. The second patch is then secured to the right ventricular wall with a running over-and-over suture. Any postoperative oozing from the left ventricular cavity is therefore contained as part of the right ventricular cavity.

Before the aortic cross-clamp is released, all sutures are sealed using autologous fibrin glue. The remainder of the operation is carried out in the usual manner.

Whatever the anatomy or the surgical procedure, the left ventricular and aortic pressures are measured at the end of operation. The persistence of a major left ventricular–aortic gradient (which should be evaluated with reference to its preoperative value) may indicate the need to reestablish cardiopulmonary bypass and to use a more aggressive procedure based on the integrated approach.

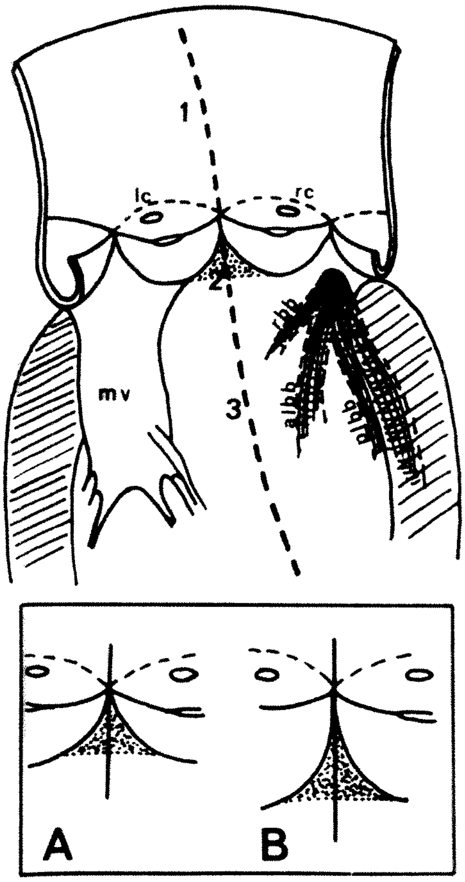

Fig 4. The left ventricular outflow tract. The aortoseptal incision involves the aorta (1), *the left anterior fibrous trigone (aortic annulus)* (2), *and the interventricular septum* (3). *(A) The left anterior fibrous trigone is wide, and its division is easy. (B) The trigone is narrow and high, and its division is more difficult. All transitional forms can exist.* (albb = *anterior left bundle branch;* lc = *left coronary ostium;* mv = *mitral valve;* plbb = *posterior left bundle branch;* rbb = *right bundle branch;* rc = *right coronary ostium.)*

Clinical Experience

Patients

Between 1982 and 1990, the protocol presented here was used in 22 patients with diffuse subaortic stenosis. Three patients 25, 30, and 46 years old had diffuse HOCM with a high left ventricular–aortic gradient at rest (100 to 120 mm Hg). Nineteen patients, 10 male and 9 female, had tunnel subaortic stenosis. The mean age at operation was 12.8 years (range, 4 to 35 years). Thirteen (68%) of the 19 patients had had a previous open heart operation (Table 1). The mean preoperative gradient was 125 mm Hg (range, 60 to 180 mm Hg). The aortic annulus was of normal size in 1 patient, moderately hypoplastic in 2 patients, and hypoplastic in 16 patients. Eleven patients had associated lesions (Table 2).

Surgical Management

The aortoseptal approach was used in all patients, and the obstructing tissue was resected from each edge of the septotomy.

In 4 patients (1 with tunnel subaortic stenosis and the 3 with HOCM), the LVOT was reconstructed. The mean immediate postoperative left ventricular–aortic systolic gradient was 14 mm Hg (range, 0 to 30 mm Hg).

Sixteen patients (all with tunnel stenosis) had a hypoplastic aortic annulus, and 11 had associated aortic valve lesions. A Konno procedure was performed. The mean immediate postoperative left ventricular–aortic systolic gradient was 5 mm Hg (range, 0 to 15 mm Hg).

In 2 patients with tunnel subaortic stenosis, the aortic annulus was moderately hypoplastic and the aortic valve cusps were normal. After resection of the tunnel, the LVOT was reconstructed. At the end of cardiopulmonary bypass, a pullback left ventricular–aortic pressure tracing showed that the subaortic stenosis had been adequately relieved but that there was residual obstruction at the annular level (left ventricular–aortic systolic gradients, 40 and 35 mm Hg). In both patients, the procedure was converted immediately to a Konno operation.

Results

Aortoseptal Approach + LVOT Reconstruction (4 patients)

There were no operative deaths. One patient required reoperation 8 months postoperatively for a residual ventricular septal defect at the suture line of the septal incision. All 4 patients were alive and symptom free after a mean follow-up of 7.6 years (range, 4 to 9.2 years). None had a major left ventricular–aortic gradient at last Doppler echocardiographic examination.

Aortoventriculoplasty (18 Patients)

There were two early deaths (11%; 70% confidence limits, 4% to 24%). Postmortem examination of 1 patient showed a massive septal myocardial infarction, probably due to the cutting of a dominant septal artery. A coronary angiogram had not been obtained before operation. The second death was related to early bacterial endocarditis.

There was no postoperative heart block. Three early reoperations were required: two for a residual ventricular septal defect and one for a right ventricular infundibular aneurysm.

Severe bacterial endocarditis occurred in 3 patients (17%; 70% confidence limits, 8% to 30%). One of them died early despite urgent reoperation. In the case of the other 2 patients, sterilization was achieved under appropriate antibiotic treatment, but aortic incompetence developed and necessitated reoperation 3 months and 6 months postoperatively. One patient died at reoperation. The other survived but underwent a third operation 2 months later for residual aortic regurgitation. Complete heart block occurred postoperatively. At last follow-up 8.5 years after the initial procedure, the patient was in New York Heart Association class II and had persistent mild aortic regurgitation.

The other 14 survivors were symptom free with a mean follow-up of 7.1 years.

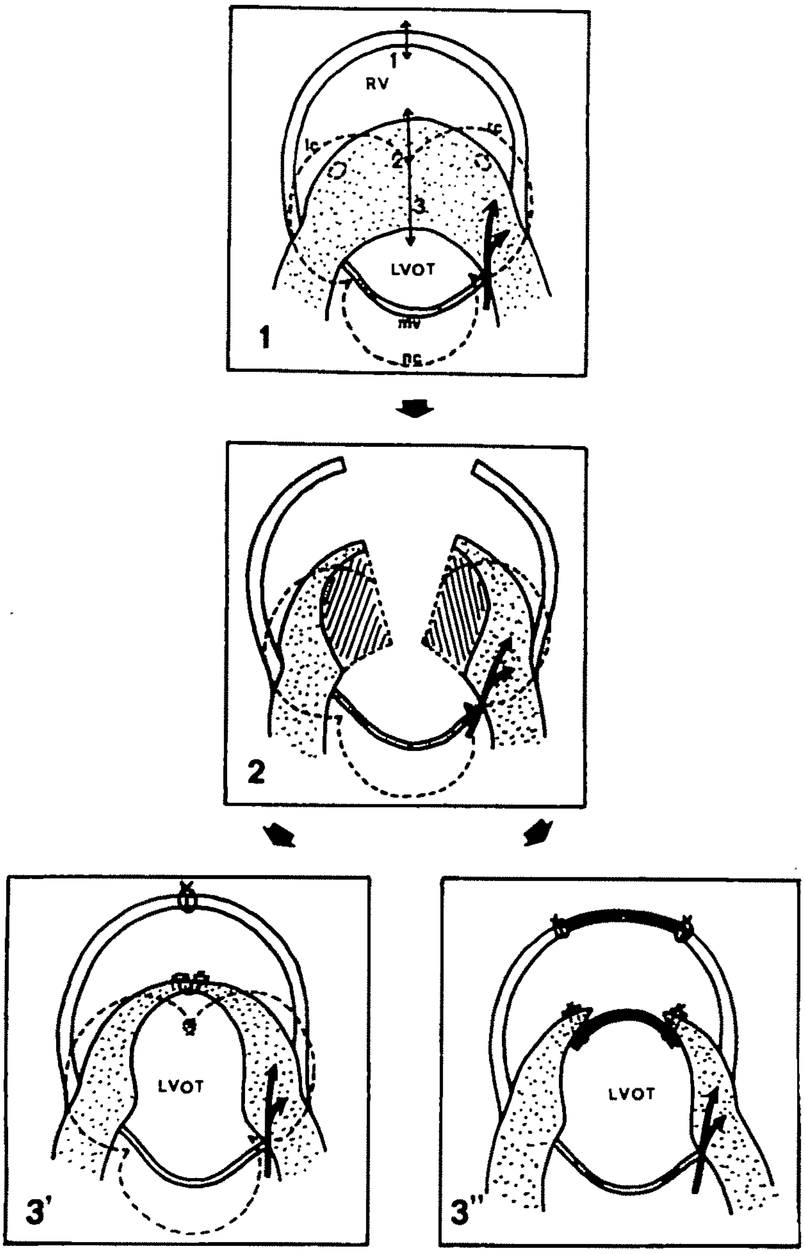

Fig 5. The concept of the integrated approach: (1) the incisions of the aortoseptal approach: (1) *right ventricle* (RV), (2) *aortic annulus,* (3) *interventricular septum; (2) the resection from each edge of the septotomy; (3') the reconstruction of the widened left ventricular outflow tract* (LVOT) *(the aortic valve is preserved); and (3") aortoventriculoplasty (Konno procedure). See text for details.* (lc = *left coronary aortic annulus;* mv = *anterior mitral valve leaflet;* nc = *noncoronary aortic annulus;* rc = *right coronary aortic annulus;* black arrows = *conduction pathways.)*

Comments

The integrated approach to the surgical management of subaortic stenosis as described here was designed to provide an adequate surgical procedure whatever the anatomical features encountered at operation. The main goal is to obtain complete relief of the LVOT obstruction. All forms of subaortic stenosis can be successfully managed by one of the procedures presented. In most cases, it is possible to assign the procedure to the form of obstruction preoperatively. However, in some patients, an obstruction more severe than expected can be discovered at operation, and the surgeon should be prepared to change his or her mind so as to use the right procedure. The

Table 1. Previous Operations in Patients With Tunnel Subaortic Stenosis

Operation	No. of Operations
Resection of subaortic stenosis	6
Valvotomy (congenital valvar stenosis)	4
Aortoplasty (congenital supravalvar stenosis)	1
Aortic valve replacement (ventricular septal defect + aortic insufficiency)	1
Apicoaortic valved conduit	2
Total	14

Table 2. Associated Lesions in Patients With Tunnel Subaortic Stenosis

Lesion	No. of Patients
Valvar lesions	
Aortic stenosis	8
Aortic regurgitation	3
Supravalvar stenosis	1
Tetralogy of Fallot	1

integrated approach makes this necessary adaptability easier. In the case of diffuse subaortic stenosis, the aortoseptal approach followed by either aortoventriculoplasty or reconstruction of the LVOT should be used.

There is one absolute contraindication to use of these procedures: the presence of a dominant septal coronary artery, which could be cut by the septal incision and might cause a massive septal myocardial infarction [23]. This was probably the cause of one early death in our series. A coronary angiogram should be obtained and studied carefully before operation. In the presence of a dominant septal coronary artery, a diffuse LVOT obstruction can be relieved by the implantation of a valved conduit between the left ventricle and aorta [24–26].

It has been clearly shown that the Konno procedure is very effective in the treatment of complex forms of LVOT obstruction [1, 23, 27, 28]. Usually the aortic annulus is divided slightly to the left of the nadir of the right coronary cusp. We favor the division of the left anterior fibrous trigone because the incision of the septum remains farther away from the conduction pathways and because the reconstruction of the LVOT is symmetrical and therefore easier. The use of Dacron patches lined with pericardium and of fibrin glue to seal the sutures makes the bleeding problem trivial. The operative risk of the Konno operation is relatively low [27, 28], and satisfactory long-term results can be anticipated [27]. In our series, there was a high incidence of bacterial endocarditis. This risk is probably increased by the quantity of prosthetic material implanted. Strict prophylactic antibiotic therapy is mandatory not only postoperatively but also for the long term.

The Konno procedure is indicated in patients with tunnel subaortic stenosis and hypoplastic aortic annulus, in patients with diffuse subaortic stenosis and associated valvar lesions (congenital valvar stenosis or aortic regurgitation usually secondary to bacterial endocarditis), and in patients with multiple levels of LVOT obstruction.

Use of the Konno operation becomes questionable in a patient with diffuse subaortic stenosis but a normal-sized annulus and a normal aortic valve, ie, in most patients with diffuse HOCM and in some patients with tunnel subaortic stenosis. Under these conditions, aortoventriculoplasty necessitates the excision of the normal aortic valve and its replacement with a prosthesis. This is an obvious drawback, particularly in young patients. Some solutions have been proposed: resection by a combined aortic and left ventricular approach in diffuse HOCM [29] and a modified Konno operation (ie, aortoseptal approach without division of the aortic annulus) in complex forms of subaortic stenosis [1, 30].

As pointed out by Kirklin and Barratt-Boyes [1], "the modified Konno procedure provides only limited exposure of the subvalvar area." The aortoseptal approach, which we favor, on the other hand, offers very good exposure of this area. It has been shown in animals as well as in humans that it is possible to divide the aortic annulus without damage to the cusps and to reconstruct a normal aortic valve afterward. Consequently, if properly done, the aortoseptal approach is not deleterious and provides good exposure for adequate surgical resection. Excision of fibrous or muscular tissue or both from each edge of the septal incision provides substantial widening of the LVOT and should adequately relieve any form of diffuse subaortic stenosis. In our experience, although small, it has never been necessary to complete the septal excision by implanting a widening septal patch, as suggested by Kirklin and Barratt-Boyes [1] and Cooley and Garrett [30]. Besides, such a patch may compromise the reconstruction of the aortic valve architecture and induce some valvar dysfunction (mainly aortic regurgitation).

Compared with the combined aortic and left ventricular approach [29], the aortoseptal approach is probably less deleterious to left ventricular function and has the advantage of being easily converted to an aortoventriculoplasty if necessary.

Finally, in several patients with tunnel subaortic stenosis, the aortic annulus is moderately hypoplastic. It is sure that the division and reconstruction of the left anterior fibrous trigone makes the annulus a little more narrow. Thus, the subaortic obstruction can be adequately relieved, but a mild obstruction may remain at the annular level. This was the case in 2 of our patients. As it was the second operation for both children, the procedure was immediately converted to a Konno operation in an attempt to avoid a third open heart operation. However, this raises the question whether in a child it is better to accept mild valvar aortic stenosis with a normal native aortic valve or to obtain complete relief of the LVOT obstruction at the expense of a prosthetic valve replacement. Further experience with these procedures is necessary before this question can be answered.

In conclusion, the current surgical armamentarium can provide adequate relief of all forms of subaortic stenosis. Each situation should be clearly analyzed and the right procedure should be used. The surgeon should keep in mind two main goals: first, to obtain adequate relief of the LVOT obstruction, and second, to preserve the native aortic valve whenever possible, particularly in young patients.

We thank Ms Corinne Pasquet for secretarial assistance.

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COMMENTARY: Reoperations After Konno Aortoventriculoplasty

Between 1976 and 1986, 16 Konno-type aortoventriculoplasty procedures were performed at the University of Nebraska Medical Center. There was one early death (6%), which occurred in 1977. One patient died suddenly at 51 months of unknown causes. Three patients have been lost to follow-up, at least 1 of whom is known to be on another continent. He was alive and well 3 years postoperatively.

Eleven patients have been followed up in the past 4 months after 67 to 168 months (mean follow-up, 116 months). Three of them had initial placement of St. Jude valves. After 5, 6, and 6 years, all are in New York Heart Association class I. Two are taking aspirin and Persantine (dipyridamole), and 1 is on a regimen of Coumadin (crystalline warfarin sodium). Eight patients had initial implantation of tissue valves: seven Carpentier-Edwards valves and one Ionescu-Shiley valve. The Ionescu-Shiley valve was replaced at 33 months for tissue failure, and all seven Carpentier-Edwards valves were replaced at 49 to 110 months (mean time, 81 months) after the original implantation. All of the valve re-replacements were with St. Jude valves, usually of the same size as the tissue valve removed. At a mean of 57 months (ranges, 33 to 98 months) after reoperation, all 8 patients are in New York Heart Association class I. Five are on a regimen of Coumadin, and 3 take aspirin and Persantine.

Six of the re-replacements were performed by the surgeon who originally did the Konno procedure. They were not regarded as difficult procedures; all went well, and there were no complications. Two were performed by surgeons not familiar with the Konno procedure, and problems did arise, including one perioperative cerebrovascular accident.

For those who are unfamiliar with the Konno procedure and are doing a secondary valve replacement, the problems seem to focus primarily on knowledge of the location of the suture lines and patches in the aortoventriculoplasty. The first concern is where to put the aortotomy. My colleagues and I have been happy with placing it obliquely in the aortic patch but above the point at which the right ventricular outflow tract patch joins it. This is easily visualized provided the surgeon understands how the original operation was done. The second question is the location of the pledgets from the first operation. Pledgets on some of the sutures brought through the ventricular septal patch are simply not accessible from within the aorta. However, they seem to be sufficiently endothelialized on the right ventricular side that they can be left in place without danger. The third concern is how to suture in the new prosthesis. It has been our experience that a St. Jude valve of the same nominal size as the removed tissue valve will go in with no particular difficulty. Nevertheless, always check the proposed site with a valve sizer to be sure there will not be a problem. The suturing technique used is identical to that used in a native aortic annulus, that is, pledgeted mattress sutures of 2-0 Ethibond. Tying the valve sutures on the side where the aortoventriculoplasty patch is may be minimally more difficult, as the cloth is not distensible, but it has not posed any real problem in our experience.

We continue to believe that aortic valve replacement with a Konno aortoventriculoplasty offers excellent palliation for children with aortic valve disease and a small aortic annulus. We were initially pleased with tissue valves, but we have experienced the usual problems of calcification and tissue degeneration. We have now switched to St. Jude valves in the primary procedure and have had very satisfactory results. Experience with a small number of re-replacements suggests that these procedures are best done by a surgeon familiar with the Konno procedure. Because the re-replacements are often performed after the patient is a young adult, this point should probably be raised with the patients and families at the time of the original procedure.

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Extended Aortic Valvuloplasty: A New Approach for the Management of Congenital Valvar Aortic Stenosis

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A new technique for the treatment of congenital valvar aortic stenosis is described. It consists of augmenting the aortic cusp by extending the commissurotomy incision into the aortic wall around the leaflet insertion, mobilizing the valve cusp attachment at the commissures, and freeing the aortic insertion of the rudimentary commissure. The results of standard valvotomy performed on 48 patients (group 1) were compared with those of the new extended valvuloplasty carried out on 16 patients (group 2). The two groups were comparable in age at operation (2.7 ± 2.1 years for group 1 versus 2.1 ± 1.7 years for group 2; p = not significant) and in preoperative pressure gradient (58 ± 25 mm Hg for group 1 versus 61 ± 36 mm Hg for group 2; p = not significant). There was no operative mortality in either group. Follow-up is available on all patients, with a mean of 4.3 ± 2.6 years for group 1 versus 1.7 ± 0.5 years for group 2 ($p = 0.05$). There was one late death in group 1. Postoperative gradient was 47 ± 13 mm Hg in group 1 versus 19 ± 13 mm Hg in group 2 ($p = 0.05$). Moderate or severe regurgitation was present in 18 patients (38%) in group 1 and 2 patients (13%) in group 2 (p = not significant). Reoperation was needed in 8 patients (17%) in group 1 versus 2 patients (13%) in group 2 (p = not significant). The described valvuloplasty procedure addresses the unique pathological features of valvar aortic stenosis and provides better relief of the obstruction than the presently available techniques. Longer follow-up is needed to determine the late results of this approach.

(Ann Thorac Surg 1991;52:663–8)

Surgical treatment of congenital valvar aortic stenosis in infants and children has been associated with a fairly high incidence of residual stenosis or regurgitation [1–4], with about 35% of patients undergoing repeat valve operations, mostly replacement, within 15 to 20 years of the original procedure [5–8]. Introduction of various technical modifications, such as transventricular dilation of the aortic valve, partial opening of the fused rudimentary commissure, or the use of balloon dilation, has not improved the long-term outcome [1, 9–12]. The recent trend toward the use of valvuloplasty procedures rather than valve replacement in the treatment of valve abnormalities in pediatric patients and the encouraging results obtained when such techniques are used for mitral valve disease have prompted the search for comparable procedures applicable to the management of aortic stenosis.

This report describes a new valvuloplasty technique for the treatment of congenital aortic stenosis. The proposed approach addresses the unique pathological features of the aortic valve in this lesion and attempts to improve the long-term surgical results. Comparison is made with previously employed procedures to demonstrate the feasibility and advantages of this new technique.

Material and Methods

Patients

Records of all infants and children who underwent open valvotomy for isolated congenital aortic valvar stenosis at the Children's Memorial Hospital and the Heart Institute for Children between January 1980 and July 1990 were reviewed. Cases with associated intracardiac lesions or aortic arch anomalies were excluded. Also excluded were patients less than 3 months old to eliminate the influence of variables unique to newborn infants with critical aortic stenosis.

The study group comprised 64 patients. All patients had two-dimensional echocardiograms with Doppler studies and 40 patients (62%) had cardiac catheterization with angiography before operation. Indications for operation included a pressure gradient across the aortic valve of 50 mm Hg or more, or a lesser gradient associated with symptoms, strain pattern on electrocardiogram, or abnormal exercise test results. Gradient across the aortic valve was estimated by measuring peak systolic velocity from Doppler studies or by measuring pressures at cardiac catheterization. Based on echocardiographic or angiographic findings, aortic insufficiency was defined as mild when the regurgitant jet reached 25% of the distance from valve to apex, moderate when the jet reached 50% of the distance, and severe when the jet reached more than 50%

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of the distance. In addition, the insufficiency was considered at least moderate if there was evidence of retrograde diastolic flow in the descending aorta at the level of the diaphragm.

Two groups were identified based on the surgical procedure used. In group 1 (48 patients) the standard aortic commissurotomy procedure was performed, whereas in group 2 (16 patients), the new proposed extended valvuloplasty technique was used.

Statistics

All data were expressed as mean ± standard deviation. The differences between the two groups were compared by the unpaired Student's *t* test. A two-tailed probability level less than 0.05 was considered significant. Planimetry was used to estimate the aortic valve orifice from two-dimensional echocardiographic views.

Techniques

STANDARD VALVOTOMY (GROUP 1). The fused commissures were opened to the valve annulus. An additional partial incision in the rudimentary commissure with everting mattress sutures to prevent prolapse of the valve leaflet edges was performed in 21 patients (44%) [1]. Augmentation of the supravalvar area with a polytetrafluoroethylene patch was done in 16 patients (33%) to prevent leaflet crowding and to allow maximal space for valve opening.

NEW EXTENDED VALVOTOMY (GROUP 2). Cardiopulmonary bypass, routine cannulation, moderate hypothermia (28°C), and cold blood cardioplegia were used. An oblique aortotomy extending into the noncoronary sinus of Valsalva was made (Fig 1A). The fused commissures were opened. The incision was extended in a circumferential fashion beyond the commissure into the aortic wall around the insertion of the aortic leaflet for a distance of 1 to 3 mm on both sides, splitting the aortic wall into two layers. The inner layer, made of intima and part of the media, remains as part of the cusp and maintains its configuration and insertion to the aortic wall. The outer layer, made of media and adventitia, preserves the integrity of the aortic wall (Fig 1B). The cusps of the valve were then mobilized by undermining their insertion at the commissural level using a similar aortic wall-splitting incision (Fig 1C). Finally, the rudimentary commissure was released from its attachment by splitting the aortic wall in that area for a distance of 3 to 5 mm (Fig 1C). Areas of leaflet thickening or myxomatous nodularities were thinned out. The aortotomy was closed either primarily or with a small synthetic patch.

Results

Age at operation was 2.7 ± 2.1 years for group 1 and 2.1 ± 1.7 years for group 2 (p = not significant [NS]). Preoperative pressure gradient across the aortic valve was 48 ± 25 mm Hg in group 1 and 61 ± 36 mm Hg in group 2 (p = NS). At operation, the valve was bicuspid in 88% and 86%, tricuspid in 10% and 14%, and monocuspid in 2% and 0% in groups 1 and 2, respectively.

There was no operative mortality. Thirteen patients in group 1 (27%) and 6 patients in group 2 (38%) needed temporary inotropic support in the immediate postoperative period (p = NS).

Follow-up is available on all patients in both groups, being 4.3 ± 2.6 years in group 1 and 1.7 ± 0.5 years in group 2 ($p = 0.05$). All patients had serial postoperative two-dimensional echocardiograms and Doppler studies (Fig 2). Eight patients in group 1 (17%) and 3 patients in group 2 (19%) had postoperative cardiac catheterizations. There was one late death in group 1 (2%) in a patient with serious aortic insufficiency 5 years after operation. Gradient across the aortic valve, measured at the time of most recent evaluation, was 47 ± 13 mm Hg in group 1 and 19 ± 13 mm Hg in group 2 ($p = 0.05$) (Tables 1, 2). Mild valvar insufficiency was present in 10 patients (20%) in group 1 and 4 patients (25%) in group 2 (p = NS). Moderate or severe aortic insufficiency was present in 18 patients (38%) in group 1, being most common among patients who had partial incision of the rudimentary commissure (13/21). In contrast, serious insufficiency occurred in only 2 patients in group 2 (13%) (p = NS) (see Tables 1, 2). Both patients had serious preoperative regurgitation caused by previous open valvotomies in both and by an additional balloon valvuloplasty with resultant tearing and extensive scarring of the valve leaflets in 1. Reoperation for severe residual stenosis or regurgitation was performed in 8 patients (17%) in group 1 and in 2 patients (13%) in group 2 (p = NS).

Comment

The unique pathological features of congenital aortic valve stenosis dictate the surgical outcome. The bicommissural valve, the most common form, has an eccentric opening and commissural attachments that substantially limit the degree to which the valve can open even when the commissures are not fused. The free edges of both bicuspid leaflets are thickened, taut, and shorter than the diameter of the aortic valve annulus, further limiting their mobility. The leaflets are also suspended below the level of the sinotabular bar, with the length of the free edge of the fused leaflet shorter than the length of the sinus and the depth of the cusp shallower than normal (Fig 3) [13]. Moreover, the insertion of the valve leaflet at the commissural level is acute and is associated with a bulging valvular ring and with subcommissural fibrosis resulting in crowding of the area and restriction of cusp displacement away from the blood stream during systole [14].

Failure of presently available surgical techniques to provide good long-term results in patients with aortic valvar stenosis stems from their inability to provide solutions to the underlying pathology. After standard valvotomy, the valve opening remains eccentric with a shortened diameter and serious residual gradient. Incising the rudimentary commissure, recommended by some, provides a larger central opening and a better blood flow pattern but is frequently associated with serious regurgitation [1]. Excessive residual turbulence and hemodynamic trauma result in progressive scarring, calcification, and restenosis [15].

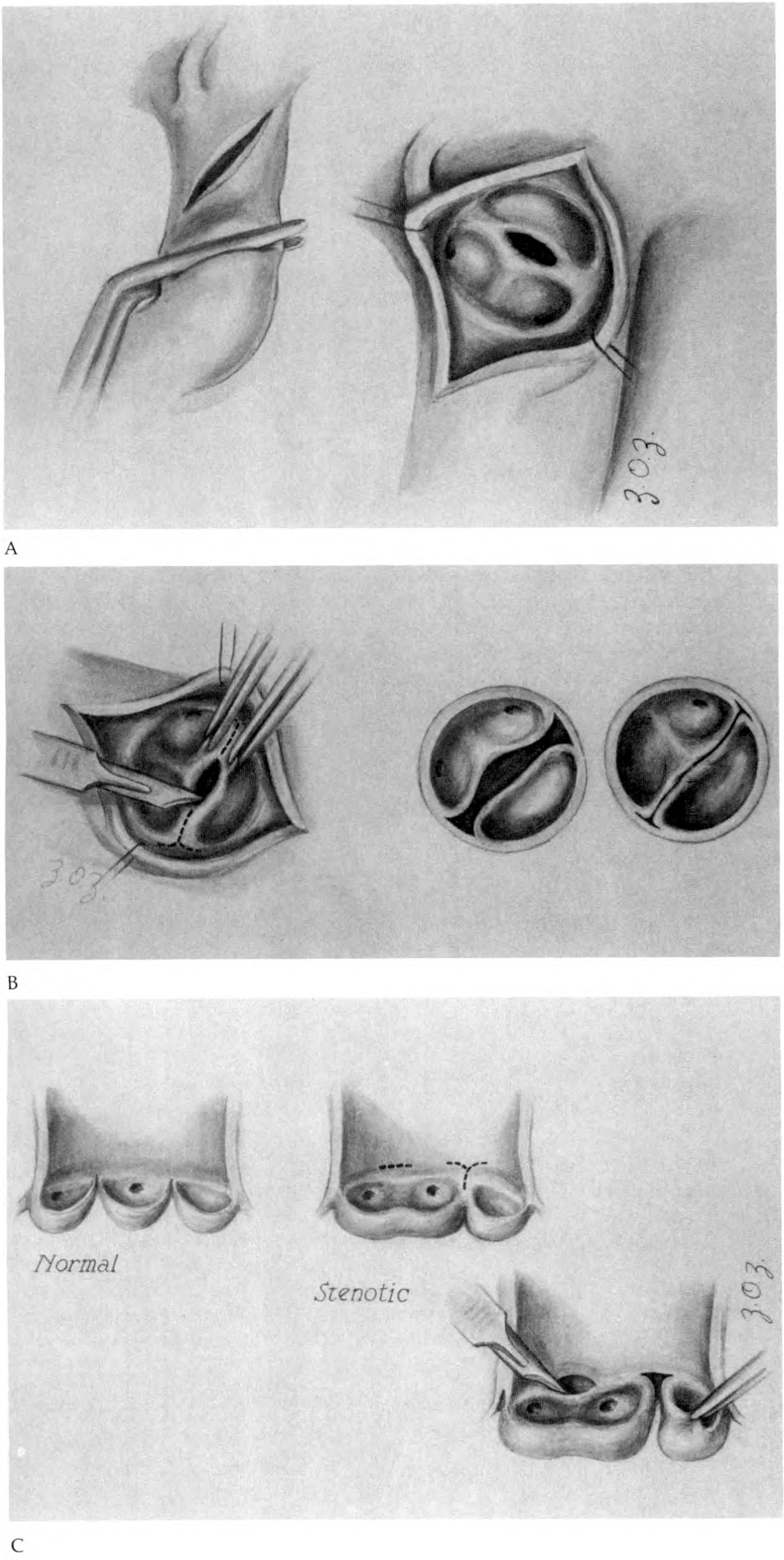

Fig 1. Details of extended valvuloplasty techniques. (A) An aortotomy incision is made, exposing the stenotic valve with fused commissure. (B) The incision is carried out through the commissural fusion into the aortic wall. The incision splits the aortic wall an equal distance on both sides of the commissure. (C) Longitudinal view of the open aorta to illustrate the extent and technique of leaflet mobilization. The incision is extended superior to the commissure to provide additional valve tissue. Note also the crowding of the subvalvar area at the commissural level and the acute angle of leaflet insertion. Normal aorta is included for comparison. At right is shown the rudimentary commissure releasing incision.

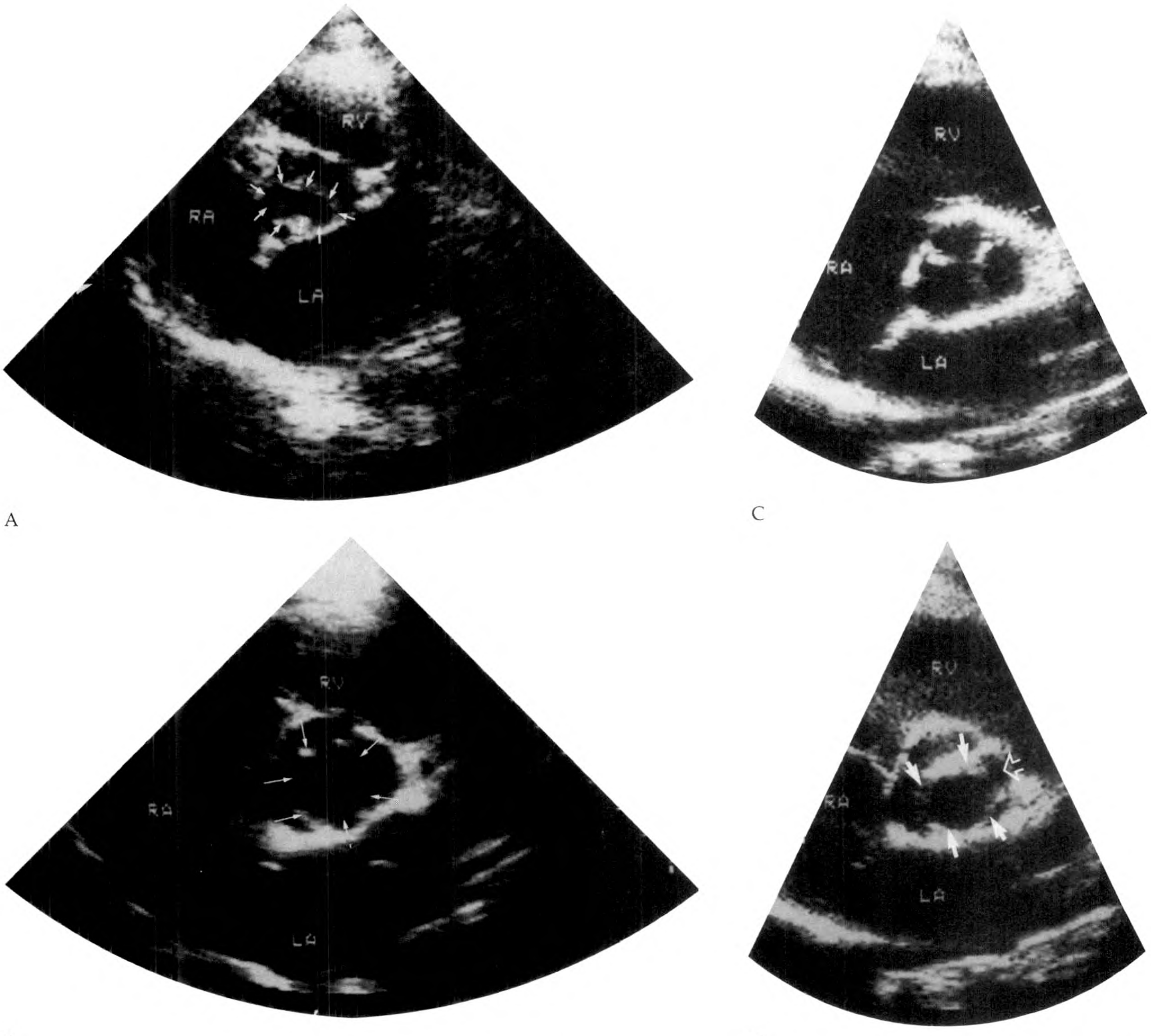

Fig 2. Postoperative echocardiogram showing the open aortic valve. (A) After standard valvotomy. (B) After extended valvuloplasty in the same patient. (C, D) Views of aortic valve in systole and diastole in another patient after extended valvuloplasty. Arrows *outline the aortic valve orifice.* Hollow arrow *outlines the site of configuration of extended valvotomy. Note the marked increase in orifice opening of the aortic valve.* (LA = *left atrium;* RA = *right atrium;* RV = *right ventricle.)*

The approach described here provides an alternative to the surgical management of aortic stenosis and addresses the problems imposed by the valve pathology. The extension of the commissurotomy into the aortic wall and the mobilization of the cusp insertion at the area of the commissures increases the mobility of the valve and

Table 1. Postoperative Results of Traditional Valvotomy (group 1: 48 patients)

Aortic Insufficiency	No. of Patients	Gradient (mm Hg)	Follow-up (y)
Absent or mild	30	52 ± 16	5.7 ± 2.3
Moderate or severe	18	32 ± 22	3.9 ± 2.6
Total	48	47 ± 13	4.3 ± 2.6

Table 2. Postoperative Results of Extended Valvuloplasty (group 2: 16 patients)

Aortic Insufficiency	No. of Patients	Gradient (mm Hg)	Follow-up (y)
Absent or mild	14	17 ± 3	1.9 ± 0.7
Moderate or severe	2	23 ± 6	1.6 ± 0.3
Total	16	19 ± 13	1.7 ± 0.5

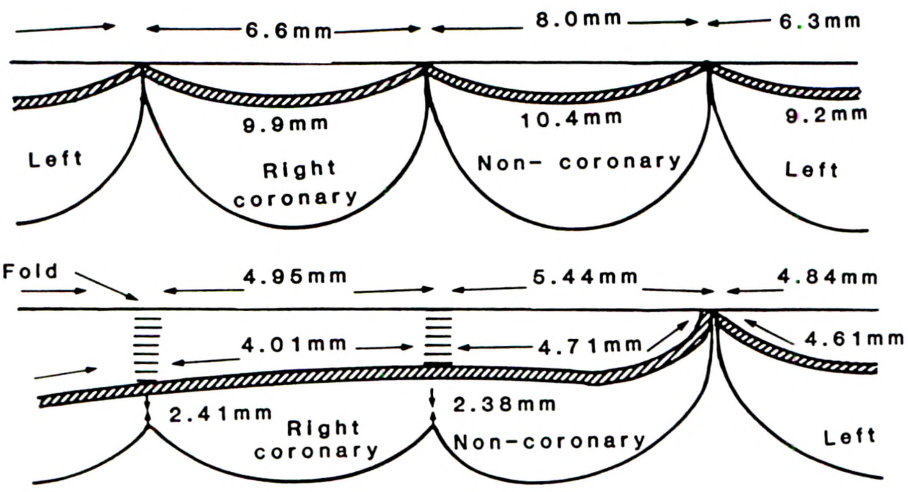

Fig 3. Anatomical differences between normal and congenitally stenotic aortic valve. The free edges of the bicuspid leaflet are shorter than the diameter of the "annulus" where normal leaflets are inserted. As a result, the cusps are shallower than normal. (Reprinted from Leung MP, McKay R, Smith A, Anderson RH, Arnold R. Critical aortic stenosis in early infancy—anatomical and echocardiographic substrates of successful open valvotomy. J Thorac Cardiovasc Surg [in press], by permission of Mosby-Year Book, Inc.)

allows a larger and more central opening. It maintains the cusp configuration and its attachment to the aortic wall. Partial release of the rudimentary commissure from the aorta without perforation or detachment of the leaflet gives an additional depth to the cusp and hence better approximation with the other leaflet.

It is essential that the commissural incisions extend superior to and to both sides of the commissures to provide ample tissue for enlarging the cusp and increasing its depth without loss of coaptation. The length of the horizontal incision on both sides of the commissure varies with the diameter of the aortic annulus. Presently, available preliminary data from planimetric studies of the special preoperative and postoperative echocardiographic views indicate that a 1-mm extension on each side for infants, and 2 to 3 mm for older children, increases the central opening up to 85% of normal (Fig 4; see Fig 2). Care must be taken not to extend the valvuloplasty too far on both sides to avoid postoperative regurgitation. Similarly, it is important to have the incisions symmetrical and equal on both sides to maintain leaflet coaptation.

The results of the new technique are encouraging. Serious residual stenosis and regurgitation were less in patients who underwent the extended valvuloplasty. However, the follow-up period is short and longer periods of observation are necessary before the technique is judged to be superior to existing approaches. On one hand, there is a remote possibility that with time the aortic wall tissue used to augment the cusps might become scarred and retracted, resulting in lack of coaptation of the leaflets. Also, aneurysmal dilatation of the aorta at the area of the valvuloplasty might occur. On the other hand, if the procedure withstands the test of time, it might encourage a more aggressive approach and earlier surgical intervention for valvar stenosis or restenosis. Such an early relief of left ventricular hypertension protects the myocardium from late onset of fibrosis and dysfunction. In addition, it decreases turbulence and might minimize the degree of rescarring and the need for reoperation.

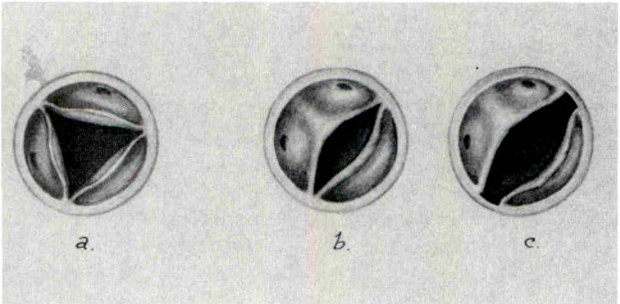

Fig 4. Comparative orifices of the aortic valve. (a) Normal tricuspid valve. (b) Bicuspid valve after standard valvotomy. (c) Bicuspid valve after extended valvuloplasty. The more central orifice opening in (c) is 40% larger than in (b).

In the two patients in group 2 with postoperative persistence of aortic insufficiency, regurgitation was centrally located and was caused by the stiffening and scarring of the aortic leaflets from previous surgical interventions. This indicates that the procedure might not be optimal in patients with severely dysplastic and markedly thickened aortic valve leaflets.

The new approach was applied to a few newborns with critical aortic stenosis. No attempt was made to include these patients in the present study to eliminate the influence of additional variables not infrequently present in newborns, such as hypoplasia of the ascending aorta, left ventricle, or the valve annulus, on the outcome of the procedure. The initial results of the technique in newborns are comparable with those of older patients, with no evidence of serious aortic regurgitation or stenosis postoperatively. It seems that the new approach has added advantages in this age group. The short incision into the wall of the aorta might contribute to the enlargement of the frequently hypoplastic annulus [16]. Conservative mobilization of the leaflets decreases crowding and could result in a larger central opening and less turbulence. The relatively more pliable leaflet and aortic wall structures in this age group maintain coaptation better than the stiffened structures in older infants and children. Such postoperative improvements in valve function have been shown to be among the most significant determinants of postoperative survival in newborn patients [9]. Although the data are not complete yet, it is hoped that the new approach might also improve the outcome of newborns with critical stenosis.

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A triangular patch cut from the remaining distal allograft aorta is used to repair the right ventricular outflow tract. The base of the patch is sutured to the donor aortic root annulus; tailoring of the corners is done with special care to avoid bleeding (Fig 5). The right ventricular suture line is then completed with the heart beating as rewarming progresses. Temporary pacemaker wires are placed for postoperative management. After the patient is weaned from cardiopulmonary bypass, the operation is completed in standard manner.

Results

Aortic valve allografts with an internal diameter of 11 to 25 mm (mean diameter, 19 mm) were implanted. Children weighing as little as 15 kg were able to receive adult-sized allografts.

Three patients (9%) died intraoperatively. A 24-year-old man had undergone three previous cardiac surgical procedures. At 4 years of age, he required aortic valvotomy. Eight years later, the aortic valve was replaced, and at age 15 years, a ventricular apex–descending aorta conduit was placed. He was seen for his fourth cardiac procedure with severe aortic stenosis and apicoaortic conduit valve insufficiency. On completion of EARR, there was excessive blood loss during takedown of the apicoaortic conduit, and this caused hypotension with subsequent LV failure and death.

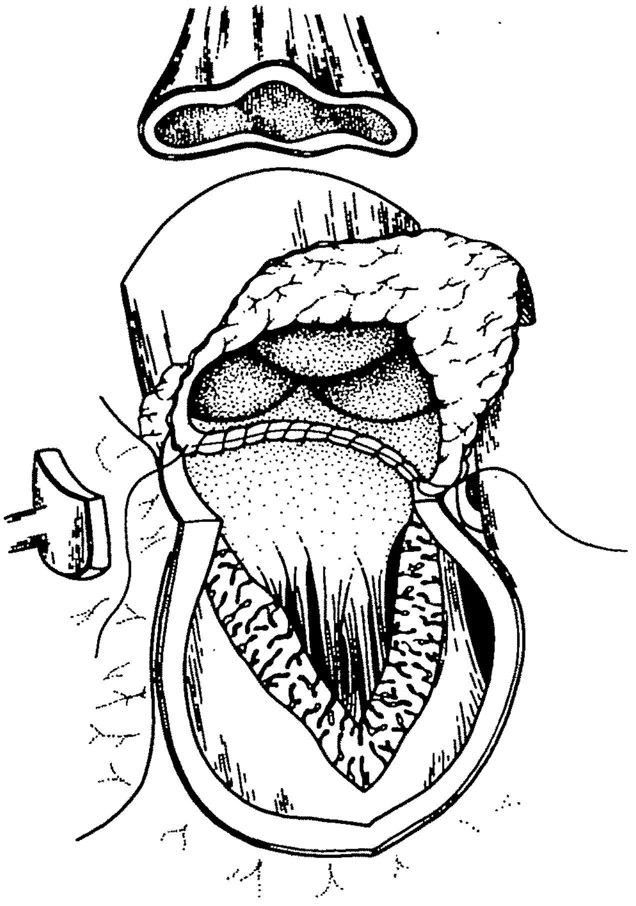

Fig 3. Allograft and recipient annuli are approximated posteriorly. Suture lines continue right and left to the interventricular septum. (Reprinted from Clarke DR. Extended aortic root replacement for treatment of left ventricular outflow tract obstruction. J Cardiac Surg 1987;2[Suppl]:121–8; by permission of Futura Publishing Company, Inc.)

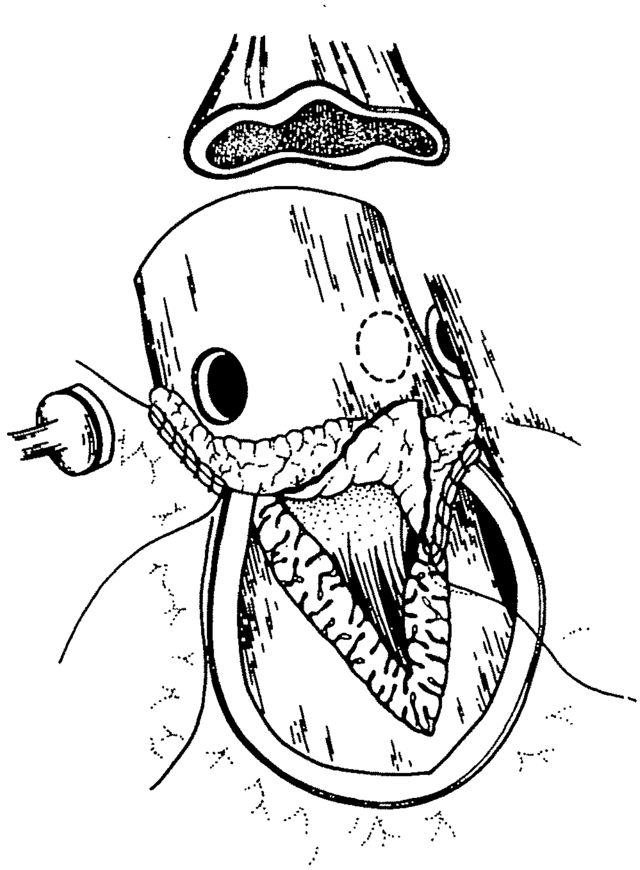

Fig 4. The anterior leaflet of the donor mitral valve is used to repair the septal incision and to enlarge the outflow tract. This portion of the suture line is usually reinforced with pledgeted sutures. (Reprinted from Clarke DR. Extended aortic root replacement for treatment of left ventricular outflow tract obstruction. J Cardiac Surg 1987;2[Suppl]: 121–8; by permission of Futura Publishing Company, Inc.)

The second intraoperative death occurred in a 3-year-old girl with a transvalvar aortic gradient of 80 mm Hg, moderate insufficiency, and progressive LV hypertrophy. She had undergone aortic valvotomy at the age of 20 months. Problems were encountered with reimplantation of the left coronary artery, and the child could not be weaned from cardiopulmonary bypass secondary to ischemic contracture.

An 11-day-old female neonate had undergone aortic valvotomy on the first day of life. Severe aortic stenosis with LV dysfunction persisted. At the time of EARR, severe (3 to 4 mm thick) endocardial fibroelastosis of the left ventricle was discovered. The LV dysfunction caused her death.

One hospital death occurred in a 9-year-old boy. He had had prior aortic coarctation repair as well as an LV

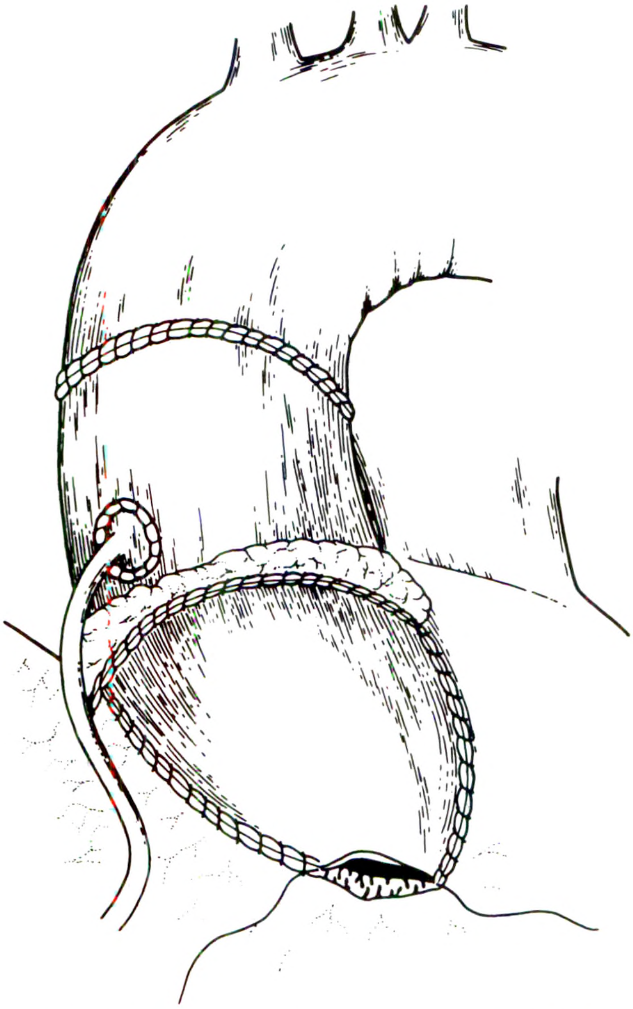

Fig 5. Buttons of coronary ostia have been reimplanted, and the distal anastomosis is complete. A triangular patch of distal allograft is used to repair the right ventricular outflow tract. (Reprinted from Clarke DR. Extended aortic root replacement for treatment of left ventricular outflow tract obstruction. J Cardiac Surg 1987;2[Suppl]:121–8; by permission of Futura Publishing Company, Inc.)

outflow tract myectomy. Reoperation was performed when recurrent subvalvar aortic stenosis in addition to aortic and mitral valve stenosis and pulmonary hypertension developed. At the time of EARR, mitral subvalvar chordal fusion was fenestrated. The postoperative course was marked by hemodynamic deterioration with intermittent stabilization. He died of biventricular failure on the seventh postoperative day.

Cardiac transplantation was ultimately done in a 5-year-old child with multilevel aortic stenosis and congenital mitral stenosis. Prior cardiac operations included repair of coarctation of the aorta at 4 days of age and aortic subvalvar membrane resection at 8 months. Mitral valve replacement was performed concurrently with EARR. Intraoperatively, LV failure necessitated the use of an LV assist device. With no improvement in LV function and deteriorating right ventricular function, the child underwent cardiac transplantation 30 hours after the allograft procedure and is alive and well at 6 months' follow-up.

Of the other patients, 9 (33%) experienced a benign hospitalization. Although the list of postoperative complications is extensive, the majority were temporary and had resolved by discharge (Table 1). Noteworthy was the development of *Serratia marcescens* mediastinitis and bacteremia in 1 child, which required debridement and irrigation. A 6-week course of antibiotics was administered. Allograft function was unaffected.

The surviving EARR recipients have been followed up clinically for 6 months to 4.8 years (mean follow-up, 2.5 years). Coronary artery bypass grafting was necessary in a 14-year-old boy with familial hyperlipidemia. Six months after allograft placement secondary to a hypoplastic aortic annulus, the child was readmitted with angina pectoris. On cardiac catheterization, a narrowed distal allograft–native aorta anastomosis was evidenced as well as 80% obstruction of the left main coronary artery. Surgical inspection revealed a normal allograft valve with lipid deposits on the intima. The coronary artery lesion was attributed to progression of the hyperlipidemia, as the stenosis was 2 cm from the ostia. The allograft was not replaced.

Three infants aged 3, 7, and 12 months experienced allograft calcification, stenosis, and progressive insufficiency, which eventually required reoperation. One child underwent redo EARR 8 months after the initial procedure. Intraoperative findings were compatible with rejection. Calcification involved the allograft conduit wall and right ventricular outflow tract patch; the aortic valve leaflets were fibrotic and retracted. The donor mitral leaflet in the septum was unaffected. Reoperative techniques were identical to those of the first EARR including replacement of the calcified right ventricular outflow tract patch to allow implantation of a larger conduit. Administration of cyclosporine was initiated concurrent with the implantation of a second allograft. Twenty-one months later, the child is normally active and asymptomatic.

Table 1. Early Postoperative Complications of Extended Aortic Root Replacement

Complication	No. of Patients
Transient heart block	4
Bleeding leading to reoperation	3
Postpericardiotomy syndrome	3
Arrhythmias	3
Pleural effusion	2
Serratia marcescens mediastinitis	1
Superficial wound infection	1
Right phrenic nerve palsy	1
Incisional ventral hernia	1
Pericardial effusion	1
Dialysis and ventilatory support	1
Mucous secretions leading to bronchoscopy	1
Atelectasis	1
No complications	9

Table 2. Most Recent Echocardiographic Evaluation After Extended Aortic Root Replacement

Variable	No. of Patients
Left ventricular function	
Good	21
Depressed	2
Aortic stenosis	
None	10
Mild	12
Moderate	1
Aortic insufficiency	
None	6
Mild	15
Moderate	2

Of the 2 other patients who required allograft replacement, 1 infant received a mechanical aortic valve prosthesis through an aortoventriculoplasty technique 37 months after EARR. The other underwent a pulmonary autograft procedure [7, 8] 39 months after allograft insertion. In each case, the calcified right ventricular outflow tract patch was replaced.

Twenty-three survivors continue to lead clinically asymptomatic lives. Most recent echocardiographic evaluations at 6 months to 3.9 years (mean duration, 1.9 years) after EARR reveal mild aortic stenosis or insufficiency or both to be common. Left ventricular function is good in 21 (91%) of these patients (Table 2).

Comment

Cryopreserved aortic valve allografts have enhanced the surgeon's ability to correct extremely complex congenital cardiac anomalies. At times, they are the only available surgical option. The EARR technique is straightforward, prolonged or severe early postoperative complications are not excessive, and permanent complete heart block has not occurred.

Of primary concern are the early allograft failures. Seven children less than 3 years old had EARR with one hospital death. Three of the other 6 children have ultimately required allograft replacement. This trend will be monitored intensively. In young patients, immunosuppressive therapy with low-dose cyclosporine or use of the pulmonary autograft procedure [8] are alternatives worthy of consideration.

After nearly 5 years of follow-up, it is impossible to adequately answer the question posed by the title of this article. The long-term fate of allografts used in EARR procedures remains undetermined. Improved techniques of cryopreservation provide superior tissue viability, which may produce increased allograft durability [9]. Intermediate follow-up of aortic allograft valved conduits in the LV outflow tract is generally favorable. This observation in conjunction with the immediate advantages such as their relative ease of insertion, their versatility and resistance to infection, the avoidance of anticoagulation in the pediatric population, and the ability to implant adult sizes in children certainly justifies their continued use for LV outflow tract reconstruction, particularly in the older child.

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COMMENTARY: Homograft Aortic Root Replacement

I read with interest Dr Clarke's paper on extended aortic root replacement and have heard him talk on the subject on a number of occasions. We are in complete agreement on the advantages of using an adult-sized homograft root that the child will grow into rather than a small mechanical valve that the child will outgrow and therefore be seen later with an obstructive prosthesis. The homograft is the only biological valve (apart from the pulmonary autograft) applicable to children, and because it is inserted as a complete functional entity and is not distorted by surrounding tissues, it is invariably competent. For that reason alone, I believe it will last longer than the conventional orthotopic homografts.

Dr Clarke and I differ in regard to technical details only. In 37 patients with tunnel-type left ventricular outflow tract, most of whom had had one to three previous operations, like the patients of Dr Clarke, my colleagues and I have never had occasion to split the septum or do a combined Konno procedure and root replacement such as he describes. Simply excising the root and all the hypoplastic fibrous tissue above and below the valve usually results in dramatic opening of the outflow. Any subvalvular thickening can be removed from above or a myotomy can be carried out. Again, an adult-sized homograft root is used.

Also, we always insert orthotopic homograft valves and roots in an anatomical orientation. The aortic valve and cusps are not of equal size and depth, and the shallower right cusp should, I believe, be in its anatomical position.

Finally, I now quite often use the modification of Elkins in root replacements in patients without infection. In this technique, the left coronary orifice is retained on a tongue of aorta, an extension of the aorta separated just below the left coronary orifice. This simplifies the operation and avoids distortion and tension on the left coronary artery.

I have also been asked to comment on the use of fresh homografts. We have both frozen valves and fresh antibiotic-preserved valves at 4°C. The latter have a number of advantages: two or three valves can be visible and readily available at the operation, and there is no delay in their reconstitution.

Studies on viability that we carried out years ago [1, 2] indicated that a significant proportion of viable fibroblasts persist for up to 3 weeks in nutrient solution, and it has been our policy to use valves up to 18 storage days old. Apart from the surgical convenience of "fresh" valves, we question the advantages of fully viable or homovital homografts with antigenically active tissues, particularly in children and especially if the endothelium persists. Our chief aim is to maintain structural integrity, and viability is simply an indication that the tissue has not been denatured or damaged substantially. In our experience, the cusps will become acellular in any case, probably as a result of low-grade antibody reaction.

Fresh valves are convenient only when the supply and turnover of valves is at a predictable level. Otherwise, immediate freezing is necessary to preserve stocks of homografts in a nondeteriorating state. It is, however, no guarantee of long-term viability within the patient.

I congratulate Dr Clarke on his results and initiative. If I see the need for extended root replacement, I shall not hesitate to follow his example.

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COMMENTARY: Experience With Pulmonary Autografts for Aortic Stenosis

The pulmonary autograft is unique among aortic valve substitutes. Its three-cusp design offers a nearly perfect replication of the normal aortic valve. The tissue is undoubtedly alive and immunologically compatible at the time of implantation. Both experimental studies [1] and clinical follow-up [2] suggest that the pulmonary autograft is capable of growth in its new environment. However, this procedure also is probably among the more technically demanding aortic valve replacements, and the pulmonary autograft itself is clearly a "limited edition." Evaluation of the early and late results is thus essential to identify optimal circumstances for transfer of a patient's own pulmonary valve to the aortic position.

The autograft operation is a long surgical procedure. Cardiopulmonary bypass time is usually more than 2 hours and may exceed 3, and the average aortic cross-clamp time among our 34 youngest patients was 133 minutes [2]. Although associated procedures have been carried out in about 15% of patients [3], such procedures generally have been related to the left ventricular outflow tract. In addition to the obvious prerequisites of a normal pulmonary valve and the possibility of excising it without damage, patient selection must take into consideration any major associated lesions that would prolong the operative procedure.

The choice between implantation of the autograft as a valve or a complete "aortic" root is made intraoperatively. When the native aortic root is sufficiently large to accept the pulmonary valve without distortion, it can be inserted with two suture lines, like a homograft valve [3]. This is usually the situation in older patients with aortic regurgitation. The normal pulmonary valve, however, is marginally larger than the normal aortic valve, a difference that may be accentuated in congenital obstruction of the left ventricular outflow tract. In these instances, root replacement is desirable, both to accommodate the pulmonary autograft and to relieve any subaortic obstruction.

Early mortality has resulted from myocardial damage

[2, 3], infection [4], or hemorrhage [5]. The first appears to be related to injury of the first septal branch of the anterior descending coronary artery during removal of the autograft and can be avoided by preservation of this important vessel [6]. The etiology of infection remains uncertain but may be a consequence of previous endocarditis, the length of the operation, or the homograft implanted in the right ventricular outflow. Although we have not had major hemorrhage as the sole cause of an operative death, this has been reported by others [5] and may reflect technical factors. It is our policy to tie the sutures securing the ventricle and autograft over a buttress of Teflon felt or pericardium to avoid this complication, but complete relief of left ventricular outflow obstruction and careful management of arterial pressure after bypass may also be contributory factors.

In theory, the properly implanted autograft valve should function indefinitely, but late endocarditis has occurred among these patients at a rate of 1.2% to 1.8% per patient-year [3]. Again, the reasons for this complication are uncertain, but it may mean that young patients should have prophylactic antibiotics as do patients with recently affected rheumatic valves. Patient selection may be important, and we would now be reluctant to use a pulmonary autograft after recent endocarditis. Reoperation has also been necessary to replace the homograft valve in the right ventricular outflow tract and to repair or replace the autograft after technical errors, particularly during the early part of the learning curve [3]. Overall actuarial freedom from reoperation (repair or replacement) on the pulmonary autograft was 40.5% ± 11.9% at 19 years after implantation [3]. In most of the patients who required autograft removal and wished to have a biological heart valve, it was possible to implant a homograft aortic valve or root at the subsequent operation.

Perhaps the most compelling recommendations for the autograft operation, however, are the patients themselves. Virtually all survivors have remained in New York Heart Association functional class I without anticoagulation or other cardiac medication, and, although trivial regurgitation can be detected by Doppler echocardiography, this has not become hemodynamically significant or progressive in the absence of endocarditis. Left ventricular function has been good and has shown no deterioration with time. If the problems of infection on the autograft and degeneration of the homograft in the pulmonary position can be solved, the pulmonary autograft may become the ideal "prosthesis" for aortic valve or root replacement.

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The Damus-Fontan Procedure

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The Damus-Kaye-Stansel operation is a useful technique for the treatment of complex cyanotic congenital heart disease when there is obstruction between the systemic ventricle and the aorta. Modifications of the technique include transection of the aorta and the pulmonary artery, anastomosis of the contiguous aortic and pulmonary walls, and connection of the distal aorta to the perimeter of the new bivalved proximal great artery. In addition, the bidirectional cavopulmonary shunt technique can be used with or without the Fontan procedure. Six patients underwent a Damus-Fontan operation, and all survived. Two patients underwent the Damus–cavopulmonary shunt (hemi-Fontan) procedure, and 1 survived. The postoperative status of the 7 survivors is good to excellent. Follow-up ranges from 2 months to 7½ years.

(Ann Thorac Surg 1991;52:676–9)

Anastomosis of the proximal main pulmonary artery to the side of the aorta to achieve unobstructed blood flow from the left ventricle to the aorta was suggested by Dr Paul Damus in 1973 [1]. Stansel [2] and Kaye [3] also described this approach for the treatment of complex cyanotic congenital heart disease. We [4] have previously reported our experience with the Damus-Kaye-Stansel procedure, here referred to as the Damus procedure. This report provides long-term follow-up of the original patients with single ventricle and expands the discussion of the technical modifications used in our institution. In addition, the bidirectional cavopulmonary shunt [5, 6] can be used with the Damus procedure to provide excellent palliation for patients with single ventricle.

Material and Methods

Patient Population

Six patients with univentricular hearts underwent the Damus-Fontan procedure [4]. Three patients had tricuspid atresia with transposition of the great arteries and a restrictive ventricular septal defect. One patient had double-outlet right ventricle, mitral atresia, and subaortic stenosis. Two patients had double-inlet left ventricle and transposition of the great arteries with restrictive ventricular septal defect. All patients had had prior pulmonary artery banding.

Two patients underwent the Damus–cavopulmonary shunt operation for palliation of complex congenital heart defects [7]. A patient born with a variant of the hypoplastic left heart syndrome had undergone repair of coarctation and pulmonary artery banding as an infant. He was seen at 1 year of age with severe right ventricular hypertension, elevation of pulmonary artery pressure, and a small, poorly functioning left ventricle. The other patient was seen with tricuspid atresia, transposition of the great arteries, and a restrictive ventricular septal defect at 11 months of age. Initially, pulmonary artery banding and repair of coarctation of the aorta were performed. The Damus–cavopulmonary shunt procedure was performed 2 months later.

Operative Technique

In older infants and children undergoing the Damus-Fontan procedure, continuous hypothermic cardiopulmonary bypass is used. It is usually possible to free up and mobilize the aortic arch beyond the level of the innominate vein. Cannulation on the inner curvature of the aortic arch allows placement of the cross-clamp just proximal to the innominate artery; thus continuous perfusion of the innominate artery and other arch vessels is possible while the repair is accomplished. In infants, deep hypothermia and circulatory arrest may be necessary to provide adequate access to the side of the ascending aorta. If hypoplasia of the proximal arch is present, a gusset of homograft can be used, as in the Norwood procedure [8]. Venous cannulation is adapted to the planned operation. We usually place a right-angled, metal-tipped cannula in the superior vena cava, particularly when transection of the vena cava is planned. The inferior vena cava can be cannulated with an angled cannula or a straight, basket-tipped cannula.

In the original reports [1–3], transection of the pulmonary artery with anastomosis to the side of the aorta was recommended. We believe that the operation should be adjusted to provide the optimal connection between the proximal pulmonary artery and the aorta. When dense adhesions exist or if the band site is proximal, the main pulmonary artery may not easily reach the side of the

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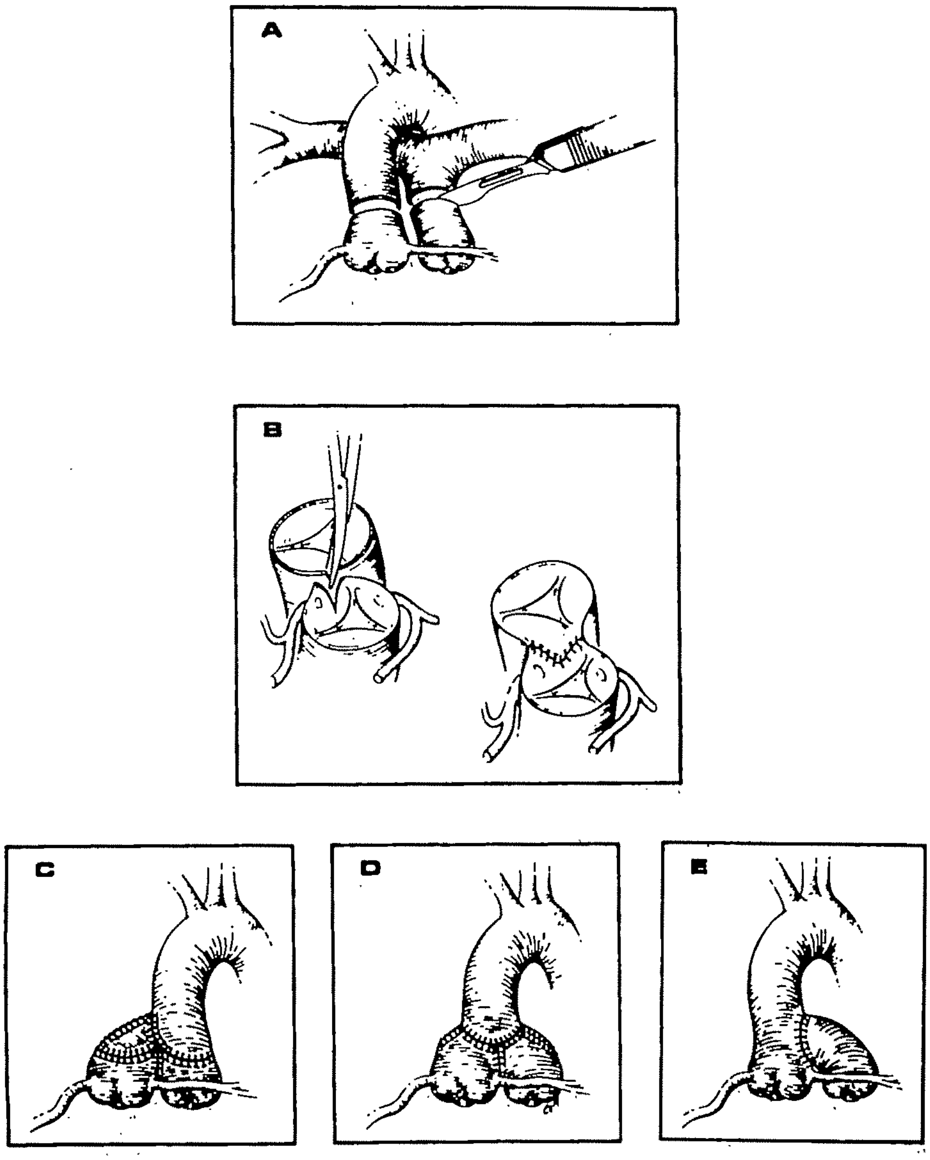

Fig 1. Modifications of the Damus procedure. (A) The aorta and the pulmonary artery are transected at the same level, the cephalad end of the aortic sinuses of Valsalva. (B) Incisions are made in the contiguous pulmonary and aortic walls, with special attention paid to the coronary ostia and the valve commissures. These walls are sewn together. (C) Early modification of the Damus operation to allow unobstructed flow into the ascending aorta without distortion of the pulmonary annulus (causing regurgitation). (D) Current adaptation of the Damus approach allows rapid anastomosis of the new (bivalved) single "artery" (see B) to the ascending aorta. (E) Original Damus concept for comparison: end-to-side anastomosis of the pulmonary artery to the aorta. (Reprinted from Waldman JD, Lamberti JJ, George L, et al. Experience with Damus procedure. Circulation 1988;78(Suppl 3):32–9; by permission of the American Heart Association, Inc.)

ascending aorta. A gusset of Dacron, pericardium, or homograft may be necessary to provide adequate egress for blood from the left ventricle to the aorta. Transection of the main pulmonary artery at the level of the band site allows complete removal of scar tissue and repair of the pulmonary arteries when necessary. The aorta can also be transected. This approach allows optimal exposure of the pulmonary artery confluence and of the semilunar valves (Fig 1).

An incision is made in the adjacent sinuses of Valsalva, and the aorta and pulmonary artery are connected with continuous Prolene (Ethicon, Somerville, NJ) or absorbable suture. The distal aorta is then positioned over the new, bivalved "artery" arising from the ventricular mass. The distal aorta is sutured to the bivalve with Prolene or absorbable suture in an alignment that minimizes obstruction to blood flow. The pulmonary artery can be tailored if necessary, and a gusset of patch material may be useful to complete the anastomosis.

Three of the 6 patients undergoing the Damus-Fontan procedure underwent transection of the aorta and main pulmonary artery. In the other 3 patients, the pulmonary artery was transected and sutured to the side of the ascending aorta.

Patients undergoing the Damus–cavopulmonary shunt procedure underwent transection of the main pulmonary artery with patch reconstruction of the distal pulmonary artery. The proximal pulmonary artery was then sutured to the side of the ascending aorta. In 1 of our 2 infant patients, deep hypothermia and circulatory arrest were employed, and a gusset of homograft was used to complete the aortopulmonary connection.

Figure 2 depicts the exposure when the aorta, the main pulmonary artery, and the superior vena cava are transected. After transection, the aorta may be mobilized away from the distal pulmonary arteries. Distal pulmonary artery stenoses can be easily repaired with an appropriate patch, and a capacious anastomosis between the

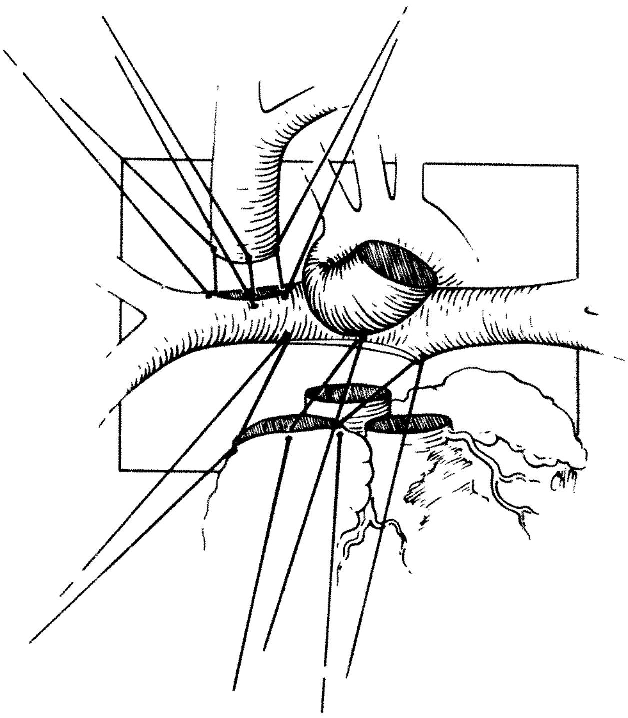

Fig 2. Operative technique for combined Damus-Fontan-cavopulmonary connection. The transected main pulmonary artery and aorta allow excellent exposure of the distal pulmonary artery confluence. Unobstructed connections can be achieved between the right atrium and the pulmonary artery and the combined proximal aorta and pulmonary artery. For clarity of representation, cannulas are not shown. This operation has always been done using continuous hypothermic cardiopulmonary bypass with direct cannulation of the superior vena cava at the innominate junction.

superior aspect of the right atrium and the undersurface of the pulmonary artery can be performed. Then the Damus connection can be completed. Finally, the Fontan connection can be accomplished with performance of a cavopulmonary anastomosis if appropriate.

Myocardial preservation includes the use of multidose blood cardioplegia and core cooling. A cooling jacket is placed around the heart in all patients, and the anterior myocardial temperature is monitored continuously. Cardioplegia is repeated at 20- to 30-minute intervals with direct cannulation of the coronary ostia if necessary.

Results

All 6 patients undergoing the Damus-Fontan procedure survived. Follow up ranges from 4 to 7½ years. Each patient has satisfactory exercise tolerance, and no patient experiences fluid retention. Two patients have had recurrent episodes of supraventricular tachycardia. One is on a regimen of digoxin (Lanoxin; Burroughs Wellcome, Research Triangle Park, NC). The other has an epicardial DDD pacemaker for late-onset sick sinus syndrome association with amiodarone hydrochloride therapy. Four patients are arrhythmia free. None of the 6 patients has required reoperation for the Damus-Fontan connection.

Five of the 6 patients have undergone recatheterization. There are no gradients in the systemic venous circuit. One patient had an 8-mm gradient from the left ventricle to the ascending aorta. Three patients have trace to 1+ semilunar valve regurgitation. In 1 patient, a left ventricular apical conduit was placed to the descending aorta during infancy. This was left in position at the time of the Damus-Fontan connection. Late-onset regurgitation through the porcine valved conduit developed. At the age of 7 years, 3 years after the Damus-Fontan connection, the patient underwent percutaneous umbrella occlusion of the conduit. Complete occlusion of the apical conduit was achieved, and the patient has remained well since that time.

Two patients underwent the Damus–cavopulmonary shunt operation [7]. The 1 with a variant of hypoplastic left heart syndrome died on the operating table. The preoperative pulmonary artery pressure was 28/20 mm Hg in the right pulmonary artery and 28/16 mm Hg in the left pulmonary artery. Average pulmonary artery pressure was about 22 mm Hg. Adequate oxygenation could not be achieved at the completion of the operation despite elevation of the superior vena cava/pulmonary artery pressure to 25 mm Hg. We concluded that the patient had elevated pulmonary vascular resistance and that adequate pulmonary blood flow could not be achieved with a cavopulmonary connection. The cavopulmonary shunt was taken down, and a systemic pulmonary artery shunt was implanted. The patient still could not be weaned from bypass because of poor myocardial function.

The other patient, who had tricuspid atresia with transposition of the great arteries, is doing well 2 months postoperatively. The resting oxygen saturation is 0.80. The patient is on a regimen of maintenance digoxin and Aldactazide (spironolactone with hydrochlorothiazide; G.D. Searle & Co, Chicago, IL) therapy.

Comment

The Damus-Kaye-Stansel procedure, here referred to as the Damus procedure, is a useful technique for repair or palliation of infants and children born with complex congenital heart defects. The operation provides a means to bypass obstruction to systemic blood flow without the need of complex intracardiac baffles or extensive intracardiac muscle resection. The risk of heart block associated with this connection should be negligible. Despite this, prior reports [9–11] have indicated a relatively high mortality for this operation. In addition, the development of the coronary switch procedure (anatomical repair of transposition) has reduced the total number of candidates for the Damus operation.

Nonetheless, patients born with obstruction to systemic output because of a single ventricle and obstructive bulboventricular foramen, patients born with two ventricles and transposition of the great arteries with subaortic stenosis, or patients born with hypoplastic left heart syndrome with a small but inadequate left ventricle are still candidates for the Damus procedure. Although an obstructive bulboventricular foramen can be enlarged surgically [12], this procedure carries some risk of complete heart block, and there is always the possibility of inadequate resection.

The Damus procedure is especially applicable for patients in whom the Fontan connection is planned, because it assures a nonobstructed systemic outlet with minimal possibility of myocardial damage. Intracardiac repair in patients born with transposition of the great arteries and subaortic stenosis is possible, although the risk is increased. When complex baffles are used for the intraventricular repair, anatomical repair with concomitant resection of obstructing muscle or tissue may not be as safe as the Damus-Fontan procedure.

Some authors [10, 13] have emphasized that pulmonary artery banding exaggerates subaortic stenosis and have recommended an early Damus shunt type of procedure. We [4] believe that pulmonary artery banding carries lower risk than a primary Damus shunt operation during the neonatal period. Early debanding after the pulmonary resistance has fallen to normal levels permits use of the cavopulmonary shunt to provide pulmonary blood flow after the Damus procedure. Thus, pulmonary artery banding followed by the Damus–cavopulmonary shunt operation should convert patients with a single ventricle to the optimal physiological status during the first year of life. The Damus procedure eliminates afterload, and the cavopulmonary shunt provides increased pulmonary blood flow without volume load of the single ventricle.

In summary, the Damus procedure provides a straightforward, safe method for relieving obstruction to systemic blood flow in complex forms of congenital heart disease. For patients with single ventricle, the Damus procedure seems ideal. For patients born with two functional ventricles, alternative surgical procedures may be equally attractive. The surgeon can determine the appropriate surgical procedure at the time of operation when the anatomy is clearly defined. Medium-term follow-up of our patients indicates satisfactory growth of the anastomosis and minimal progression of semilunar valve insufficiency. Long-term follow-up will demonstrate whether patients undergoing the Damus-Fontan procedure have the same natural history as patients undergoing the Fontan procedure for single ventricle with unobstructed systemic ventricular outlet.

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The Damus-Stansel-Kaye Procedure: Anatomical Determinants and Modifications

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Seven of 119 patients undergoing anatomical correction for transposition of the great arteries and Taussig-Bing anomalies without pulmonary stenosis had the Damus-Stansel-Kaye procedure and the rest, the arterial switch. The age of the patients having the Damus-Stansel-Kaye procedure ranged from 0.5 year to 5 years (mean age, 2.2 ± 1 years). Four patients had transposition, 2 had Taussig-Bing anomaly, and 1 had corrected transposition. Indications for the Damus-Stansel-Kaye procedure were side-by-side great arteries associated with difficult coronary anatomy (5 patients), single coronary system (1 patient), and subaortic stenosis (1). A graft between the ascending and descending aorta for interrupted aortic arch made mobilization and posterior displacement of the ascending aorta for the arterial switch difficult. Subaortic stenosis (1 patient), subpulmonary ventricular septal defect (2 patients), and restrictive ventricular septal defect (4) precluded the Rastelli procedure. In 6 patients, the main pulmonary artery was transected at the band, a proximal main pulmonary artery to aorta anastomosis was complemented with a synthetic patch, and a right ventricle to distal main pulmonary artery valved conduit was inserted. Four patients had closure of the aortic outflow. Two patients had postoperative bleeding and 2, heart block. The only patient who did not have transection of the main pulmonary artery, an omission that led to an obstructed conduit at the distal anastomosis, died late. Two patients subsequently needed aortic outflow closure for critical aortic insufficiency. The Damus-Stansel-Kaye procedure has a definite role and can be safely performed in patients with transposition of the great arteries and Taussig-Bing anomalies. Aortic outflow closure minimizes the risk of aortic insufficiency. Transection of the distal main pulmonary artery allows a wide open conduit to main pulmonary artery anastomosis.

(Ann Thorac Surg 1991;52:680–7)

The use of the proximal main pulmonary artery as an outflow to the aorta was applied successfully by Neches and colleagues [1] in 1973 as palliation for a form of subaortic stenosis from a restrictive ventricular septal defect in a patient with tricuspid atresia and transposition of the great arteries. In 1975, Damus [2], Stansel [3], Kaye [4], and Diaz and associates [5] independently proposed correction of transposition of the great arteries by applying a similar principle of directing the left ventricular output to the aorta through a proximal main pulmonary artery–aortic root anastomosis and interposing a valved conduit between the right ventricle and distal main pulmonary artery. Subsequently, Norwood and colleagues [6] extended its application to the first-stage repair of hypoplastic left heart syndrome.

The need for a valved conduit in anatomical correction without coronary artery translocation for transposition of the great arteries, henceforth called the Damus-Stansel-Kaye procedure, is a major disadvantage compared with the arterial switch with coronary artery transfer for simple transposition. However, the Damus-Stansel-Kaye procedure has a definite role in patients with transposition of the great arteries and Taussig-Bing malformations with coronary artery anatomy unsuitable for transfer. In addition, patients with associated ventricular septal defect and without pulmonary stenosis who have subaortic stenosis or restrictive ventricular septal defect might be better candidates for the Damus-Stansel-Kaye procedure than for the Rastelli operation [7]. We reviewed our experience to define anatomical variations appropriate for the Damus-Stansel-Kaye procedure and to determine surgical modifications that might improve results.

Material and Methods

Patient Population

One hundred nineteen patients with transposition of the great arteries and Taussig-Bing anomalies without pulmonary stenosis underwent anatomical correction in our institutions in the past 7 years. One hundred twelve patients had an arterial switch with coronary artery transfer, whereas 7 patients had the Damus-Stansel-Kaye procedure for various reasons.

A summary of the clinical data on the 7 patients who underwent the Damus-Stansel-Kaye procedure is outlined in Table 1. Four patients who had transposition of the great arteries, ventricular septal defect, and previous pulmonary artery banding underwent exploration to de-

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Table 1. Summary of Clinical Data on Patients Undergoing Damus-Stansel-Kaye Procedure

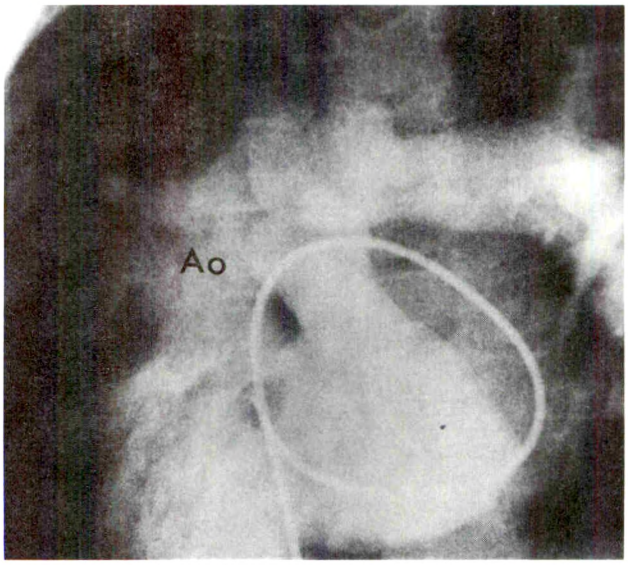
Patient No.	Age at Repair (y)	Diagnosis	Previous Operations	Reason for Damus-Stansel-Kaye Procedure	Aortic Valve or Subvalvar Region Closed	Size of Conduit	Complications	Outcome
1	3	TGA, VSD, dextrocardia, juxtaposed atrial appendages	PAB, SP shunt	Side-by-side great arteries, restrictive VSD	No	#22	Postop bleeding	Aortic valve closure for AI, 10 mo postop Well, 7 y postop
2	2	TGA, VSD, PAPVD, polysplenia	Atrial septectomy, PAB	Side-by-side great arteries, muscular VSD	Yes	#20	None	Well, 5 y postop
3	2	TGA, VSD, "criss-cross" heart	PAB	Side-by-side great arteries, levoposition of aorta crowded VSD	Yes	#20	None	Well, 4 y postop
4	0.9	TGA, VSD, coarctation	PAB, coarctation repair	Single coronary artery, restrictive VSD	Yes	#16	None	Well, 5 mo postop
5	0.5	Taussig-Bing anomaly, coarctation	PAB, coarctation repair	Side-by-side great arteries, SAS	No	#20	None	Closure of subaortic region for AI, 22 mo postop; well, 6 y postop
6	2	Taussig-Bing anomaly, IAA	PAB, repair of IAA	Side-by-side great arteries	Yes	#18	Postop bleeding, heart block	Well, 5 y postop
7	5	Corrected TGA, VSD, SAS, coarctation	S_1 PAB, coarctation repair; S_2 MPA–Aorta anastomosis (age 1 y); S_3 Glenn shunt, left SP shunt, ligation of MPA (age 2 y)	SAS	No	#15	Heart block	Died of homograft obstruction, 2 mo postop

* Side-by-side great arteries means that the right or left coronary artery is far from the main pulmonary artery.

AI = aortic insufficiency; IAA = interrupted aortic arch; MPA = main pulmonary artery; PAB = pulmonary artery banding; PAPVD = partial anomalous pulmonary venous drainage; SAS = subaortic stenosis; SP = systemic–pulmonary artery; TGA = transposition of great arteries; VSD = ventricular septal defect.

termine the possibility of performing an arterial switch procedure. At operation, the Damus-Stansel-Kaye procedure was done because the great arteries were found to have a side-by-side relationship that resulted in the right coronary artery being far from the main pulmonary artery in 2 patients with dextroposition of the aorta (Fig 1), and the left coronary artery far from the main pulmonary artery in 1 patient with levoposition of the aorta (Fig 2). In the fourth patient, who also had side-by-side relationship of the great arteries, the right and left coronary arteries originated as a single trunk from the posterior sinus of the aorta. Although they were in close proximity to the main pulmonary artery, transfer might have resulted in kinking of one of them. In addition, there was a right marginal coronary artery originating from the right aortic sinus that was far from the main pulmonary artery. Although the preoperative angiograms showed the side-by-side relationship of the great arteries, the feasibility of transferring the coronary arteries could be determined only at operation. The presence of restrictive ventricular septal defects (muscular in 3 patients, posterior and covered by endocardial tissue in 1 patient) made the Rastelli procedure not feasible.

Two patients who had Taussig-Bing anomalies and previous pulmonary artery banding underwent exploration for anatomical correction. The Damus-Stansel-Kaye procedure was performed because a side-by-side relationship of the great arteries made the distance between the right coronary artery and main pulmonary artery great, thereby precluding an arterial switch procedure (Fig 3). One patient also had major subaortic stenosis. In the second patient, the presence of a graft interposed be-



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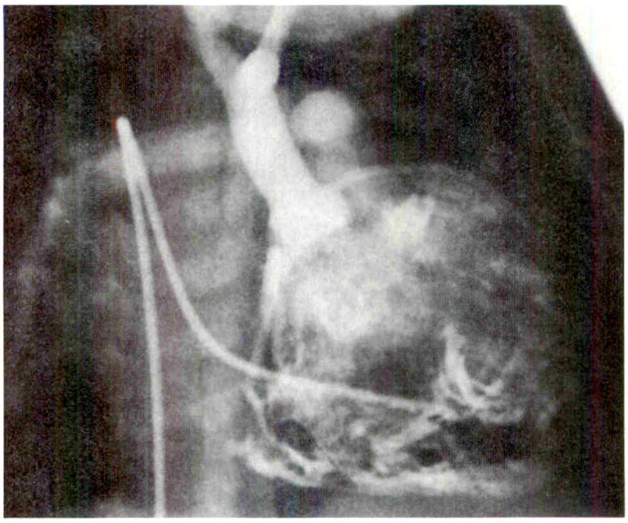


B

Fig 1. Angiograms of (A) patient 1 and (B) patient 2 with dextro-transposition of the great arteries showing side-by-side relationship of the aorta (Ao) *and pulmonary artery.*

tween the ascending and descending aorta for correction of an interrupted aortic arch early in infancy made mobilization of the ascending aorta difficult for an arterial switch procedure (Fig 4). Additionally, the necessity of displacing the ascending aorta posteriorly with the Lecompte procedure caused concern about the possibility of kinking the ascending–descending aorta graft in this patient [8]. The subpulmonary location of the ventricular septal defect in both patients also made the Rastelli procedure not an ideal choice.

The seventh patient had an essentially corrected transposition anatomy except that the aorta and the pulmonary artery were both related to the left-sided right ventricle and a large subaortic ventricular septal defect was present. The aorta was to the right of the main pulmonary artery. The patient initially underwent pulmonary artery banding and coarctation repair, followed by a main pulmonary artery–ascending aorta anastomosis for severe subaortic stenosis a year later. When the patient was 2 years of age, because of substantial pulmonary blood flow and obliteration of the right ventricle by hypertrophied muscles, the main pulmonary artery was ligated at the band area, a Glenn shunt was created, and a left subclavian–pulmonary artery shunt was established. When the patient was 5 years of age, cardiac catheterization showed moderate tricuspid valve insufficiency and adequate size of the ventricles. There was no gradient across the main pulmonary artery–ascending aorta anastomosis.

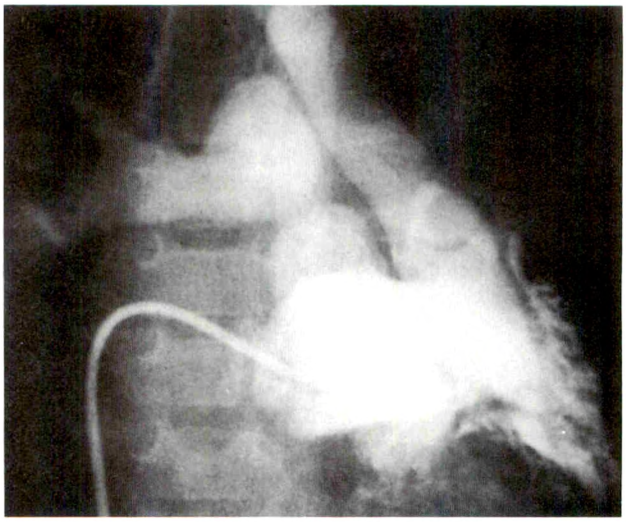


Fig 2. Angiogram of patient 3 with "criss-cross" heart and transposition showing side-by-side relationship of the great arteries and levoposition of the aorta.

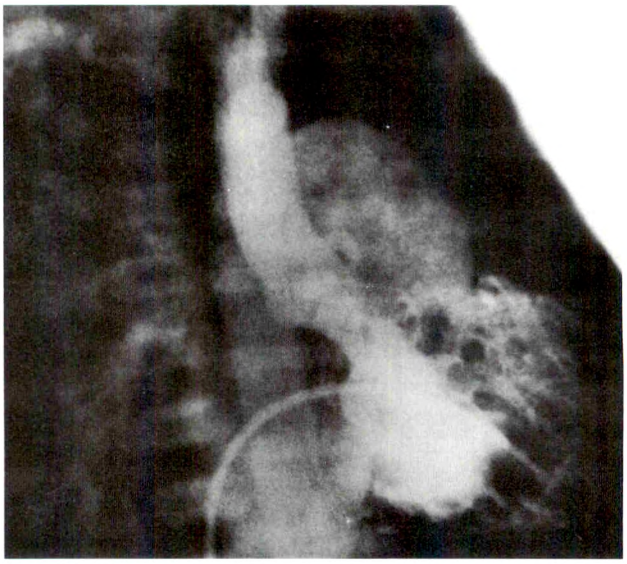


Fig 3. Angiogram of patient 5 with Taussig-Bing anomaly showing side-by-side relationship of the great arteries.

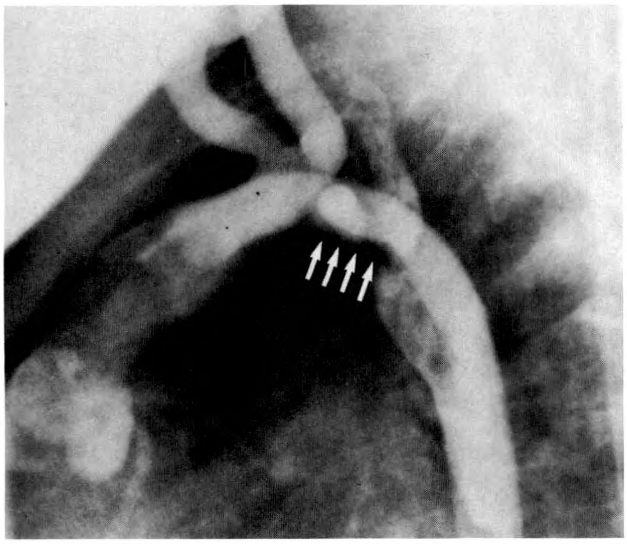


Fig 4. Aortogram of patient 6 with a graft (arrows) *for interrupted aortic arch. Mobilization and posterior displacement of the ascending aorta would have been difficult and probably would have caused kinking of the graft.*

Surgical Technique

The procedure was carried out through a median sternotomy incision. Cardiopulmonary bypass with moderate hypothermia was instituted through an aortic cannula placed high in the ascending aorta and caval cannulas inserted through the right atrial wall. In 1 patient (patient 1) with juxtaposition of the atrial appendages and dextrocardia that resulted in the right atrium being posterior to and covered by the right ventricle, venous drainage was accomplished by inserting the inferior vena caval cannula through the left-sided right atrial appendage followed by the superior vena caval cannula through the right atrial wall once cardiopulmonary bypass was established. The patient with the Glenn shunt (patient 7) had one venous cannula directed to the inferior vena cava, leaving the Glenn shunt undisturbed. A left ventricular sump was placed through the apex.

In 6 patients, after the aorta was cross-clamped and blood cardioplegia had been given, the ventricular septal defect was closed or directed to the main pulmonary artery through a right ventriculotomy. The main pulmonary artery was transected at the area of banding and the proximal main pulmonary artery–ascending aorta anastomosis was performed, complemented by a polytetrafluoroethylene patch (Fig 5). One patient had direct closure of the aortic valve before completion of the anastomosis. Once the proximal main pulmonary artery–ascending aorta anastomosis was finished, the aortic cross-clamp was removed. In 3 patients, the subaortic valve region was closed with a Dacron patch through the right ventriculotomy. Finally, the valved conduit was inserted between the right ventricle and distal main pulmonary artery.

In the patient with corrected transposition (patient 7) who had already had the main pulmonary artery–ascending aorta anastomosis created for subaortic stenosis, the left ventricular flow was directed with a baffle through the ventricular septal defect to the aorta and pulmonary artery. The main pulmonary artery was not detached at the banded area, as it had been completely ligated at the band area 3 years earlier. A homograft was interposed between the right ventricle and the distal main pulmonary artery. A Mustard procedure was performed to direct the inferior vena caval flow to the right ventricle and the pulmonary venous flow to the left ventricle [9]. The Glenn shunt was left alone.

Results

Early Results

There were no early deaths. Two patients had postoperative bleeding requiring reexploration. Two patients had complete heart block requiring permanent pacemaker implantation.

Late Results

One patient (patient 7) who had heart block and severe ascites postoperatively, which improved somewhat with diuresis, returned moribund 2 months postoperatively and subsequently died. Postmortem examination showed severe stenosis at the homograft–distal main pulmonary artery anastomosis.

The initial 2 patients who did not have closure of the aortic valve or the subvalvar region at the time of the Damus-Stansel-Kaye procedure had major aortic insufficiency postoperatively and underwent closure at 10 months and at 22 months postoperatively. The last 4 patients in the series had closure of either the aortic valve or the subvalvar region at the time of the Damus-Stansel-Kaye procedure. All 6 patients are doing well at up to 7 years of follow-up.

Comment

Because of concerns regarding late right ventricular dysfunction and arrhythmias in patients with transposition of the great arteries who had either a Senning or Mustard procedure, there is now a trend toward anatomical correction, eg, arterial switch with coronary artery transfer [10, 11], Rastelli procedure [7], and Damus-Stansel-Kaye procedure. Arterial switch with coronary artery transfer is the preferred choice, as use of a valved conduit or homograft that will eventually require replacement is not necessary. However, because some patients with transposition of the great arteries and Taussig-Bing anomalies have anatomical variations not suitable for an arterial switch, there is a definite role for either the Rastelli or Damus-Stansel-Kaye procedure in the correction of these lesions.

A side-by-side relationship of the great arteries associated with difficult coronary anatomy was the most common major reason for not attempting the arterial switch in our patients. Although we [11] have successfully performed the arterial switch in patients with Taussig-Bing anomalies, there are patients with side-by-side relation-

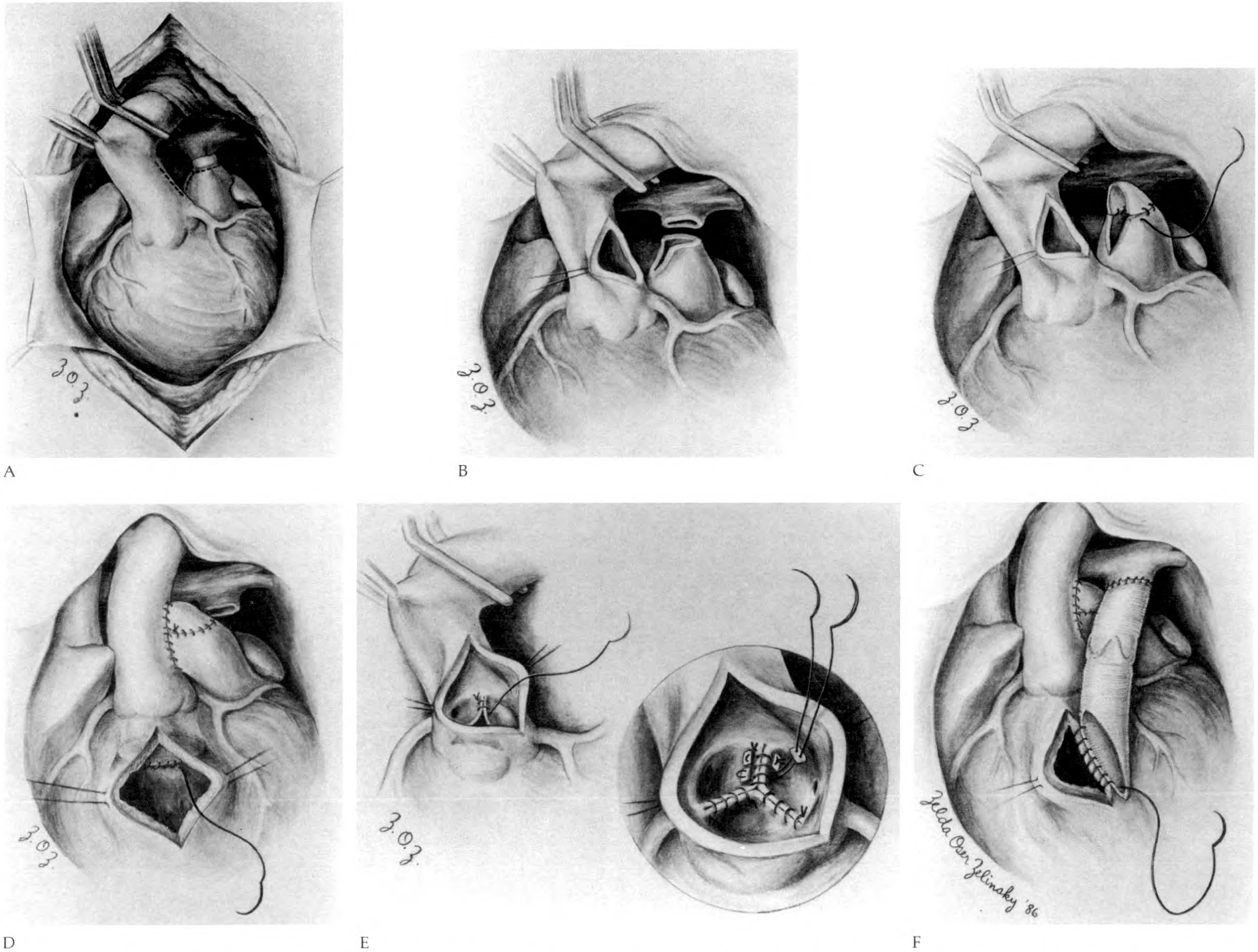


Fig 5. (A) Lines of incisions and (B) incisions in aorta and pulmonary artery. (C) Polytetrafluoroethylene patch for pulmonary artery–aorta anastomosis. (D) Closure of subaortic valve area. (E) As an alternative, closure of aortic valve through aortotomy used for pulmonary artery–aorta anastomosis. (F) Completion of procedure with insertion of valved conduit. See text for complete description.

Table 2. Collected Patients Undergoing Damus-Stansel-Kaye Procedure

	Year	No. of Patients	Diagnosis	Closure of Aortic Valve or Subvalvar Region: Initial	Closure of Aortic Valve or Subvalvar Region: Later	Outcome
Damus et al [13]	1982	1	TGA	Not closed	. . .	Alive
Smith et al [16]	1982	1	Taussig-Bing anomaly	Closed	. . .	Alive
Binet et al [17]	1983	1	Taussig-Bing anomaly	Closed	. . .	Alive
Glaser and Castaneda [18]	1983	1	Taussig-Bing anomaly	Not closed	. . .	Alive
Crupi and Parenzan [19]	1983	1	Taussig-Bing anomaly	Closed	. . .	Alive
Ceithaml et al [20]	1984	24	TGA (20); Taussig-Bing anomaly (4)	Closed, 1	Closed for PH, 1	Alive, 9
Trusler and Freedom [21]	1985	4	Taussig-Bing anomaly	None closed	. . .	Alive, 3
Lin et al [22]	1986	1	TGA	Closed	. . .	Alive
Oldham[a]	1986	1	Taussig-Bing anomaly	Not closed	Closed for AI	Alive
Waldham et al [15]	1988	6	TGA (5), DORV (1)	Closed, 3	. . .	Alive, 4; mild AI, 2
DeLeon et al [this report]	1991	7	TGA (5), Taussig-Bing anomaly (2)	Closed, 4	Closed for AI, 2	Alive, 6

[a] This was a personal communication from H. N. Oldham, Jr.

AI = aortic insufficiency; DORV = double-outlet right ventricle with subaortic stenosis; PH = pulmonary hypertension; TGA = transposition of great arteries.

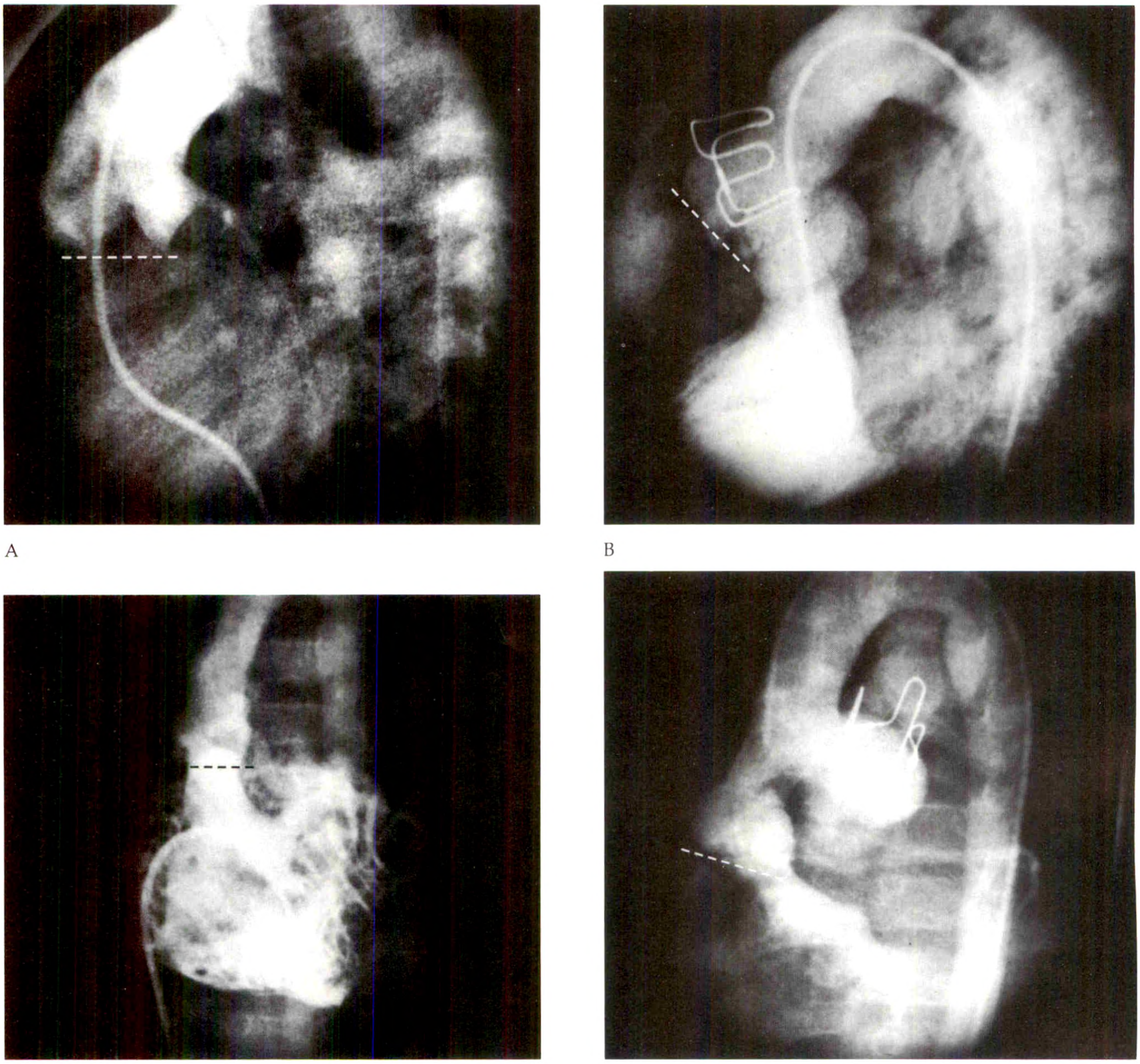
ship of the great arteries in whom there is quite a distance between the right or left coronary artery and the main pulmonary artery. The presence of subaortic stenosis and subpulmonary and restrictive ventricular septal defects would also make the Rastelli procedure not an ideal choice.

The patient with corrected transposition (patient 7) had essentially a combination of a Rastelli and a Damus-Stansel-Kaye procedure similar to that described by Kawashima and colleagues [12]. In this patient, we connected the homograft to the side of the distal main pulmonary artery, which was not detached at the banded area. This led to a severely narrowed and crowded distal anastomosis for the homograft.

The question of whether or not to close the aortic valve or the subvalvar region at the time of the Damus-Stansel-Kaye procedure remains unresolved. Because of concern about possible future aortic valve incompetence, Kaye [4] suggested closure at the time of correction. Diaz and colleagues [5] also suggested closing the aortic valve. Stansel [3] recommended closure of the subaortic valve region in patients with elevated pulmonary vascular resistance to avoid possible right-to-left shunting through the aortic valve. Damus and colleagues [13] and Danielson and co-workers [14] suggested leaving the aortic valve open so that, should conduit obstruction occur, cyanosis rather than right ventricular failure would result.

Including our patients, we collected 48 patients in whom the Damus-Stansel-Kaye procedure had been performed (Table 2) [13, 15–22; Oldham HN Jr: Personal communication]. Twelve patients (25%) had closure of the aortic valve or the subvalvar region at the time of operation. There were 29 survivors (60%), 19 of whom did not have closure of the aortic valve or the subvalvar region at the time of correction. Of these 19 patients, 4 (21%) subsequently required closure because of critical aortic insufficiency in 3 and cyanosis from pulmonary hypertension in 1. An additional 2 patients have mild aortic valve insufficiency [15]. We thought that the reason for the aortic valve insufficiency in our patients was because the aortic valve faced a large, low-pressure right ventricle, and this allowed prolapse of the aortic valve structures, which were subjected to high pressures during systole. The orientation of the aortic valve vis-à-vis the right ventricle changed postoperatively in these patients (Fig 6) [23]. In contrast, our experience [23] and that of others [22] showed no major aortic valve insufficiency developing in patients with univentricular hearts undergoing a similar proximal main pulmonary artery–ascending aorta anastomosis for varied forms of subaortic stenosis. In these patients, the aortic valve was facing a small, thick-walled chamber, making the occurrence of aortic valve prolapse unlikely.

In summary, the Damus-Stansel-Kaye procedure has a definite role in anatomical correction and can be safely performed in patients with transposition of the great arteries and Taussig-Bing anomalies. The aortic valve or the subvalvar region should probably be closed at the time of correction to minimize the risks of aortic insufficiency. In addition, the distal main pulmonary artery should be detached to allow a wide open conduit–pulmonary artery anastomosis.



A B C D

Fig 6. (A, B) Preoperative and (C, D) postoperative angiograms of patients 1 (A, C) and 5 (B, D) showing change in orientation of aortic valve (broken lines) *vis-à-vis right ventricle with resulting aortic insufficiency.*

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III. Hypoplastic Left Heart Syndrome

Hypoplastic Left Heart Syndrome

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The evolution of the present approach to the newborn with hypoplastic left heart is outlined. Preoperatively, maintenance of ductal patency with prostaglandin E_1 and balancing of systemic and pulmonary blood flow are essential. Operative details of the first-stage palliation and the definitive second-stage procedure are described. The more recent adoption of an intermediate-stage hemi-Fontan procedure is also described. Since January 1989, 151 patients have been treated using this three-stage approach, with 109 early survivors. Seventy-eight have undergone the hemi-Fontan operation with nine deaths (5 of whom came to this stage early nonelectively because of shunt failure or ventricular dysfunction). Twenty-seven of the 78 patients undergoing hemi-Fontan operation have subsequently undergone definitive Fontan procedures with no deaths.

(Ann Thorac Surg 1991;52:688–95)

Hypoplastic left heart syndrome is the most common form of congenital cardiac malformation where there is only one fully developed ventricle. It represents fully 9% of those heart defects of infants with critical heart disease [1]. Untreated, it is universally fatal and accounts for 25% of deaths in the first few weeks of life. Although lethal if left untreated, this malformation is eminently treatable with conventional reconstructive surgical techniques or, as advocated by some, heart replacement, with results comparable with other forms of complex congenital cardiac disease [2–5]. This report outlines one means of approaching hypoplastic left heart syndrome and its contemporary results.

The central anatomical feature of hypoplastic left heart syndrome is severe aortic valve hypoplasia or, more often, aortic valve atresia. As a consequence of limited outflow, the left ventricle develops abnormally and is hypoplastic or virtually absent. There is associated mitral valve hypoplasia or atresia. Twenty-five percent of children have either double-outlet right ventricle with aortic valve atresia and left ventricular hypoplasia or complete common atrioventricular canal malaligned over the right ventricle and associated left ventricular hypoplasia and aortic valve atresia with or without double-outlet right ventricle.

The most obvious physiological consequence of this anatomical constellation is that systemic blood flow is provided virtually in its entirety from the right ventricle through a patent ductus arteriosus. The fetus is generally well and unaffected by the anatomical abnormality, but two of the natural changes from the fetal to the newborn physiology are life threatening. As the ductus arteriosus closes, systemic perfusion is impaired. In addition, a natural decrease in pulmonary vascular resistance shortly after birth results in a volume shift from the systemic to the pulmonary circulation, also impairing systemic perfusion. Satisfactory systemic perfusion and oxygenation can be maintained in the newborn by assuring patency of the ductus arteriosus and adequate intravascular volume and by avoiding large differences between systemic and pulmonary vascular resistance. As will be discussed, the last two principles are critically important in the management after palliative operation for hypoplastic left heart syndrome as well.

Preoperative Management

In virtually all neonates with hypoplastic left heart syndrome, continuous infusion of prostaglandin E_1 satisfactorily maintains patency of the ductus arteriosus. Only rarely, when there is virtual absence of an interatrial communication, does hypoxemia as a consequence of a low pulmonary to systemic blood flow ratio occur. Rather, the baby is most often threatened not by metabolically important hypoxemia but by decreased systemic perfusion as a consequence of low pulmonary vascular resistance or increased systemic vascular resistance. In this circumstance, hypoventilation to increase carbon dioxide tension and pulmonary vascular resistance, and occasionally continuous infusion of sodium nitroprusside to decrease abnormally elevated systemic vascular resistance, can approximate the satisfactory physiological state of the fetus with hypoplastic left heart syndrome. The use of catecholamines is almost never necessary or appropriate. As with single ventricle complex, babies with hypoplastic left heart syndrome are ultimately treatable by some modification of the Fontan procedure. However, at birth the lungs are immature and the vascular resistance is naturally high, precluding a Fontan procedure in the neonatal period.

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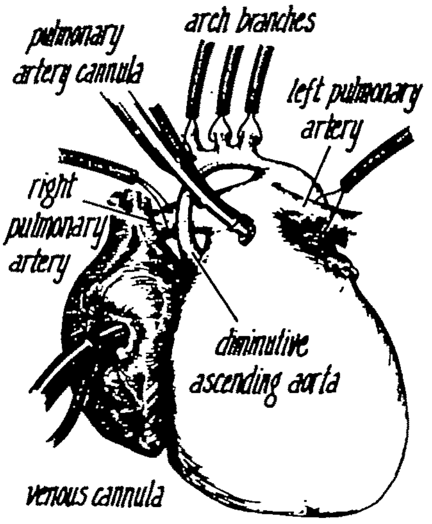


Fig 1. Exposure and cannulation for first-stage palliation. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

Yet, these infants are dependent on patency of the ductus arteriosus. Although the ductus arteriosus can be opened and maintained with continuous infusion of prostaglandins, prolonged association of the pulmonary circulation with the systemic circulation will result in either intractable congestive heart failure or, ultimately, development of pulmonary vascular obstructive disease. Therefore, it was clear even from the beginning of our surgical development that staged management is necessary.

The initial palliation is embodied by three basic principles. First, the aorta must be associated directly with the right ventricle in a fashion guaranteeing unobstructed flow from right ventricle to the systemic circulation, with growth potential obviating further aortic operation. Second, pulmonary blood flow must be regulated for proper growth and development and maturation of the pulmonary vasculature to avoid the development of pulmonary vascular obstructive disease and to minimize the volume load on the right ventricle. Finally, a large interatrial communication is necessary to avoid pulmonary venous hypertension and its consequences. The surgical details of initial palliation have evolved over the last decade. It is a personal prejudice that the intricacies that have been inserted have an important impact on the physiological stability of these patients, subsequent suitability for a Fontan procedure, and survival with a good long-term functional result after the Fontan procedure.

Operative Procedure

Through a conventional midline sternotomy incision, the thymus gland is excised, exposing the diminutive aortic arch and its branch vessels (Fig 1). Cannulation for arterial infusion is conveniently achieved in the proximal main pulmonary artery just above the sinuses of Valsalva. The tip of a 10F aortic cannula is introduced 3 to 4 mm into the artery. Threading the cannula through the ductus arteriosus is to be avoided; it is unnecessary and requires excessive manipulation of the cardiovascular structures. A minimal amount of distortion, manipulation, and trauma to the delicate neonatal cardiovascular structures is essential to preserve anatomy and function. A single venous cannula is placed through the right atrial appendage, and cardiopulmonary bypass is instituted. The right and left pulmonary artery branches are rapidly occluded to ensure systemic perfusion through the ductus arteriosus.

The baby is cooled on bypass while esophageal, nasopharyngeal, and rectal temperatures are monitored. The rectal temperature reaches 20°C after approximately 15 minutes of cardiopulmonary bypass. During this time, the branch vessels of the aortic arch are exposed and looped with suture tourniquets in preparation for circulatory arrest. Dissection is carried around the aortic arch onto the thoracic aorta in the posterior mediastinum. At this point, the branch vessels of the aortic arch are occluded, the circulation is discontinued, and the blood is drained into the venous reservoir.

After removal of the arterial and venous cannulas, the atrial septum is excised to allow unimpeded pulmonary venous return to the right atrium (Fig 2). The main pulmonary artery, which has been separated from the diminutive ascending aorta during the cooling phase of cardiopulmonary bypass, is transected adjacent to the takeoff of the right pulmonary artery, and the distal stump of the main pulmonary artery is closed with a patch (Fig 3). Patch closure is recommended to ensure continuity between the right and left pulmonary artery branches. The ductus arteriosus is then exposed, ligated, and transected at its entrance to the thoracic aorta. An incision

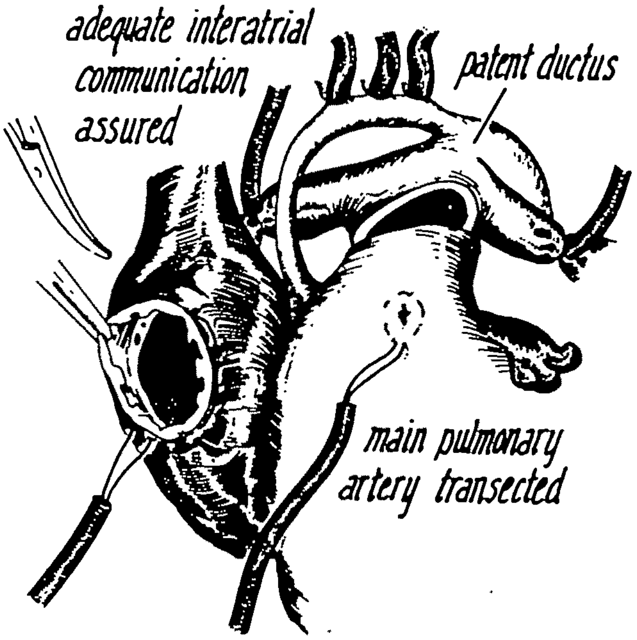

adequate interatrial communication assured
patent ductus
main pulmonary artery transected

Fig 2. First-stage palliation: the atrial septum is excised and the main pulmonary artery is transected. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

in the aorta is carried distally 1 to 2 cm into the thoracic aorta (Fig 4) and also proximally into the aortic arch and ascending aorta to the level of the rim of the transected proximal main pulmonary artery. Because the isthmus of the aorta and the aortic arch actually function as a branch of the main pulmonary artery-ductus-thoracic aorta continuum, the junction of the isthmus and thoracic aorta is gusseted with a patch to avoid subsequent development of distal aortic arch obstruction (Fig 5). A piece of pulmonary homograft is used for this patch because it is thin, pliable, and hemostatic.

At this point, we favor the construction of a short central shunt of 4-mm tube graft between the inferior aspect of the augmented aortic arch and the confluence of the branch pulmonary arteries (Fig 6). The rationale for a central shunt is to obtain more even distribution of flow and thus uniform growth of the right and left pulmonary arteries. The remaining pulmonary homograft gusset is then carried to 5 mm above the end of the most proximal incision in the ascending aorta (Fig 7). The proximal transected main pulmonary artery is then anastomosed to the ascending aorta and homograft gusset, thus creating outflow from the right ventricle to the augmented aorta through the pulmonary valve (Figs 8, 9).

Cardiopulmonary bypass is reinstituted and the patient is rewarmed to 37°C. The tourniquets on the branch vessels of the aortic arch are removed, but those on the branch pulmonary arteries remain until the time of weaning from cardiopulmonary bypass. After weaning, the cannulas are removed and a pressure monitoring line is placed through the right atrial appendage cannulation site.

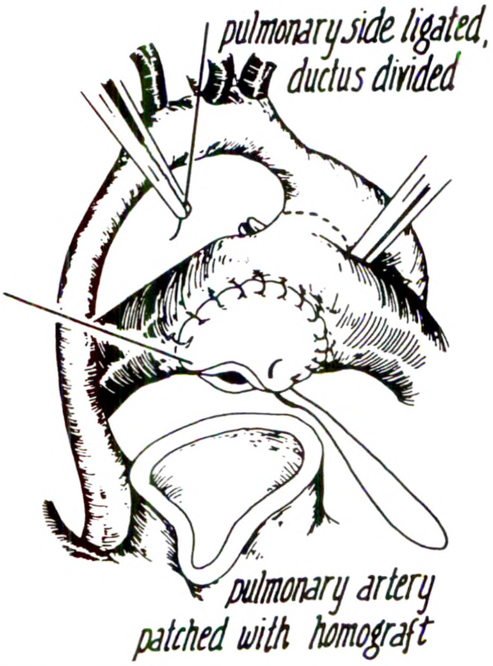


Fig 3. First-stage palliation: closure of main pulmonary artery stump. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

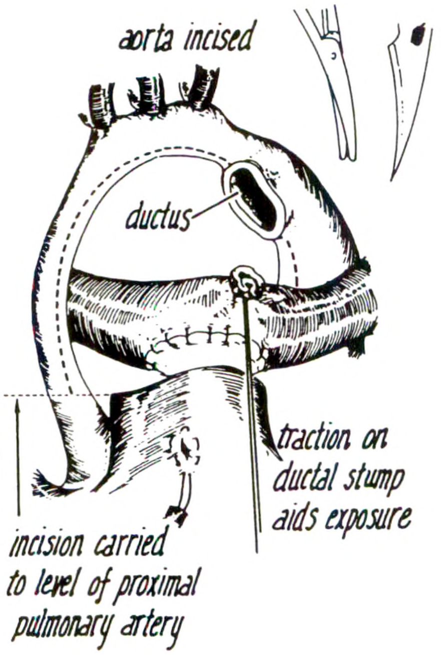


Fig 4. First-stage palliation: incision in aorta. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

Postoperative Management

Using the lowest possible mean airway pressure, ventilation is adjusted to maintain the carbon dioxide tension between 20 and 30 mm Hg to minimize pulmonary vascular resistance. Generally, the oxygen tension ranges from 40 to 45 mm Hg. However, an arterial oxygen tension of 50 mm Hg or greater suggests a large pulmonary to systemic flow ratio, which may result in inadequate systemic perfusion. When this occurs, the ventilator may be used to adjust the pulmonary to systemic flow ratio by rapidly decreasing the inspired oxygen fraction to 30 mm Hg and allowing carbon dioxide tension to increase to 40 mm Hg, with a concomitant increase in pulmonary vascular resistance. Pharmacologic support is rarely necessary in the postoperative period.

After the previously described palliative operation, the right ventricle is subjected to both volume and pressure load greater than normal for a right ventricle. With a view to long-term preservation of ventricular function, early assessment for suitability for the Fontan procedure is undertaken when the infant is 12 to 18 months of age. At this age, the pulmonary resistance generally has decreased to less than 2.5 Wood units and the ventricular end-diastolic pressure has remained normal (less than 7 to 8 mm Hg). The following operation may be planned.

Second-Stage Fontan Procedure

Again, the heart is exposed through a midline sternotomy, and cannulation for cardiopulmonary bypass is achieved by placement of an arterial cannula in the ascending aorta and a single venous cannula through the right atrial appendage (Fig 10). The systemic to pulmo-

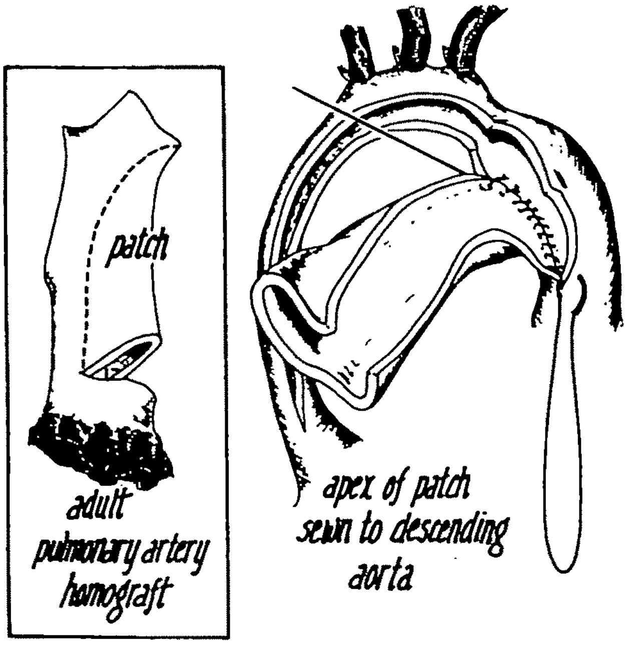

patch
adult pulmonary artery homograft
apex of patch sewn to descending aorta

Fig 5. First-stage palliation: gusseting of isthmus–thoracic aorta junction. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

nary artery shunt is exposed and occluded as cardiopulmonary bypass is begun, and the patient's core temperature is reduced to 20°C.

During this cooling phase on cardiopulmonary bypass, the right and left pulmonary artery branches are exposed from pericardial reflection to pericardial reflection. This exposure prepares for widely augmenting the pulmonary arteries to avoid proximal pulmonary arterial obstruction from unrecognized irregularities in size (Figs 11, 12).

During a period of circulatory arrest, the pulmonary arteries are opened and an incision in the right atrium is made from the sulcus terminalus superiorly to the right lateral insertion of the eustachian valve inferiorly. The interatrial communication is inspected and enlarged if possible. A final incision is then made in the superior right atrium adjacent to the right pulmonary artery and carried into the posterior aspect of the right superior vena cava immediately adjacent to the most rightward aspect of the incision in the right pulmonary artery (Fig 13). A suture line is begun between the inferior lip of the incised right pulmonary artery and the posterior lip of the right superior vena caval–right atrial incision (Fig 14). This will provide the floor for the anastomosis of the systemic venous return to the pulmonary arterial tree.

A piece of tube graft 10 mm in diameter, of sufficient length to extend from the inferior vena caval–right atrial junction to the right superior vena caval–right atrial junction, is cut in half lengthwise (Fig 15). This is for use as a baffle to channel inferior vena caval flow along the right lateral aspect of the right atrium to the anastomosis between the right atrium and the pulmonary arterial tree superiorly. The baffle is sutured around the orifice of the inferior vena cava along the right lateral floor and free wall of the right atrium and around the patulous orifice in the superior dome of the right atrium (Figs 16, 17). This particular baffling technique was introduced to minimize problems associated with tricuspid prolapse or regurgitation, or with obstruction of pulmonary venous return to

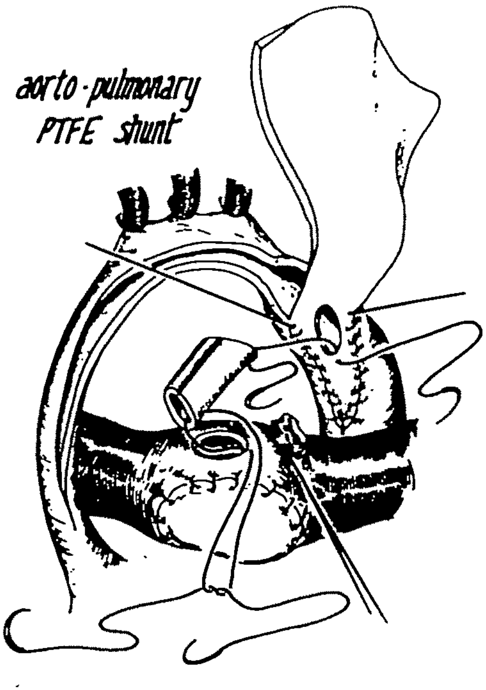


Fig 6. First-stage palliation: construction of central shunt. (PTFE = polytetrafluoroethylene.) (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

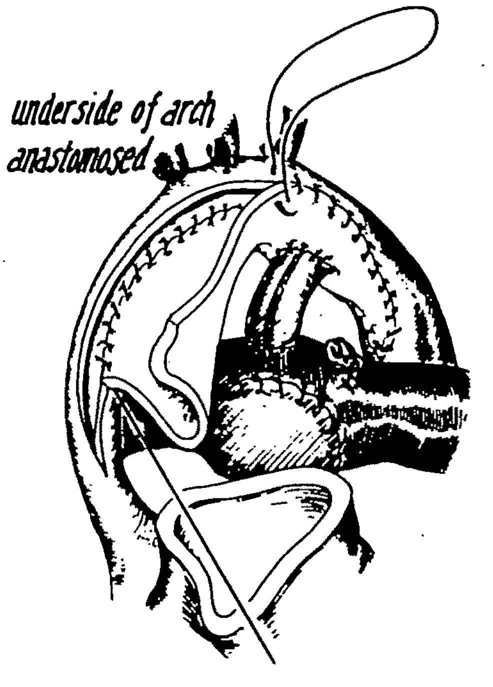


Fig 7. First-stage palliation: completion of homograft gusset. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

the right ventricle, which was experienced early in this series with an alternative baffling technique.

The construction of the systemic venous pulmonary arterial system is completed by gusseting the pulmonary arterial incision with an elongated triangular patch, beginning on the left pulmonary artery. As the right pulmonary artery and adjacent right superior vena cava are approached, the base of the triangular patch is sutured onto the anterior lip of the right superior vena caval–right atrial incision, providing a roof for the anastomosis (Figs 18, 19). The patient is placed back on cardiopulmonary bypass after closure of the initial right atriotomy and rewarmed to 37°C.

Hemi-Fontan Procedure

In the last 2 years, it has become apparent that mortality and morbidity may be reduced by separating the Fontan operation into two procedures: initial anastomosis of the superior vena cava to the pulmonary arteries, followed in 6 months by channeling of the inferior vena cava to the pulmonary arteries. The rationale is that diminution in end-diastolic volume develops rapidly in some patients after removal of the volume demands associated with a systemic to pulmonary artery shunt. Moreover, preoperative physiological information does not predict those patients in whom rapid contraction of end-diastolic volume will develop. The anatomical and physiological consequences of this process are an apparent increase in wall thickness, because muscle mass does not change as rapidly, and the diastolic function of the ventricle is impaired resulting in an increased end-diastolic pressure, increased central venous pressure, and decreased cardiac output. To minimize the physiological significance of such a rapid geometrical change, dividing the Fontan procedure into two parts is postulated to allow a decrease in end-diastolic volume and a commensurate decrease in muscle mass while only half of the systemic venous return is obligated to flow passively through the pulmonary vascular bed. When the end-diastolic volume and wall thickness ratio have normalized, completion of the Fontan procedure may be undertaken.

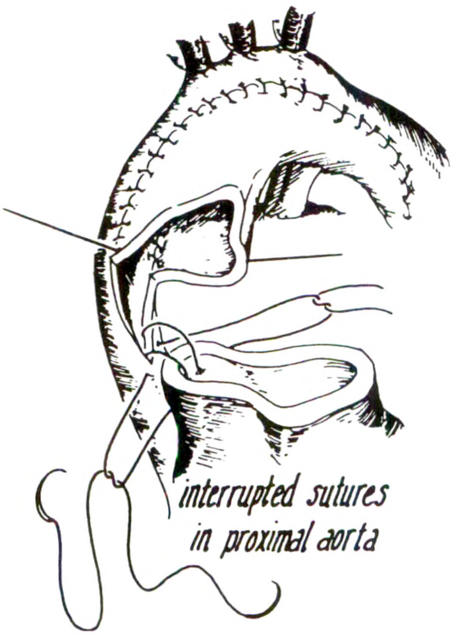
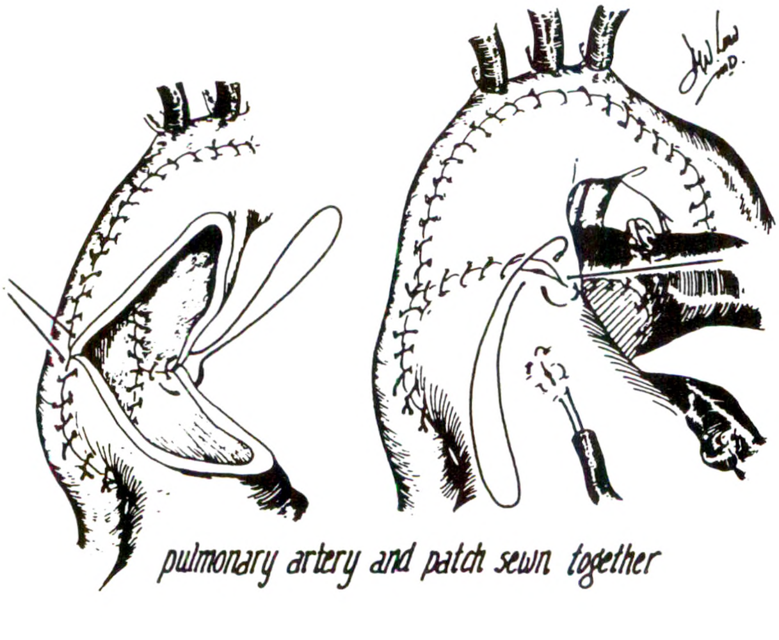


Fig 8. First-stage palliation: creation of outflow from right ventricle to augmented aorta. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

Results

The results of reconstructive operation for hypoplastic left heart syndrome continue to improve, for both initial palliation and later reconstructive operation, as we gain ever-increasing knowledge of the anatomy and physiology of this complex group of patients and of the surgical procedures required for them. In the 2-year period beginning January 1989, 151 newborns entered our institution for palliative operation. There were 42 (28%) early and 5

Fig 9. First-stage palliation: completion of right ventricle–augmented aorta outflow. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)



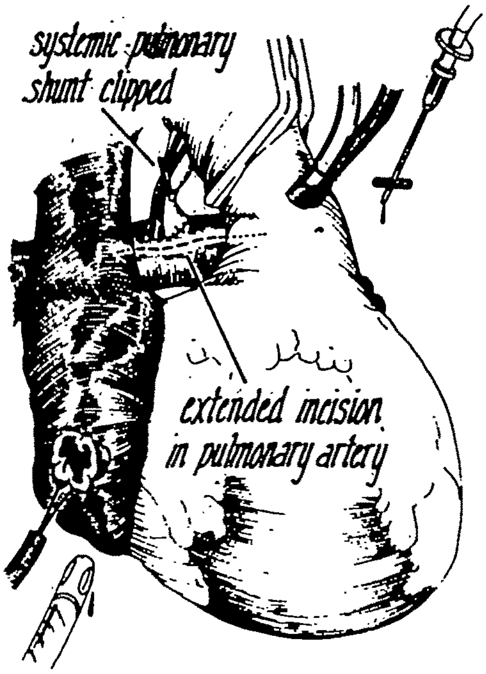


Fig 10. Exposure and cannulation for second-stage Fontan. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

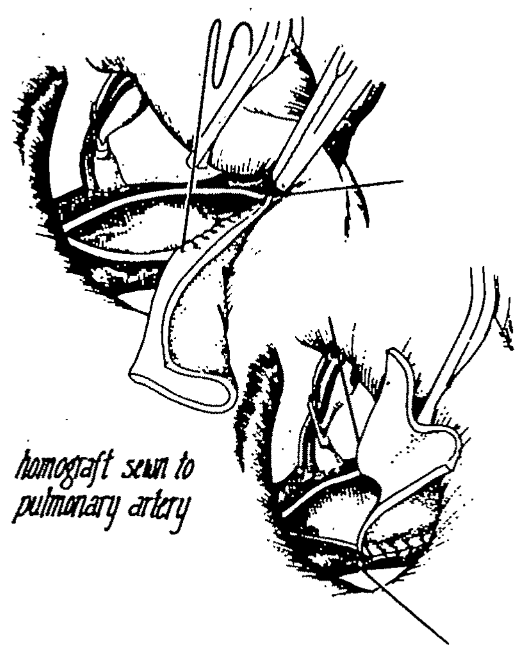


Fig 12. Second-stage Fontan: completion of pulmonary artery augmentation. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

(5%) late deaths among these patients. Seventy-eight of the 104 survivors have already undergone a hemi-Fontan operation (anastomosis of the superior vena cava to the branch pulmonary arteries). Among the 9 patients who died (12%), 5 came to hemi-Fontan operation earlier than 5 months of age because of ventricular dysfunction or shunt failure, which may have unfavorably affected the postoperative physiology. Of the 27 patients in this group

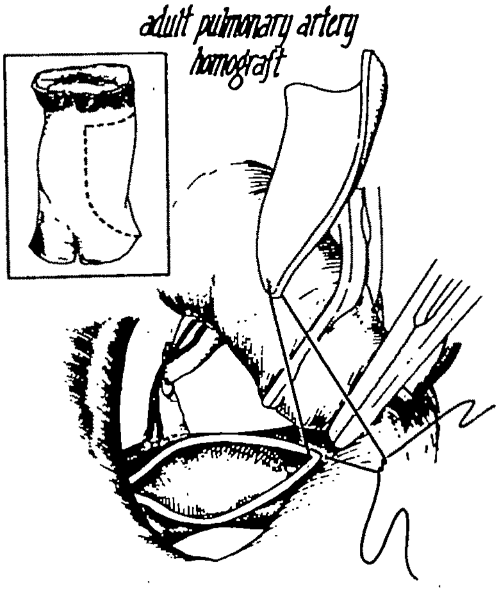


Fig 11. Second-stage Fontan: use of homograft for pulmonary artery augmentation. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

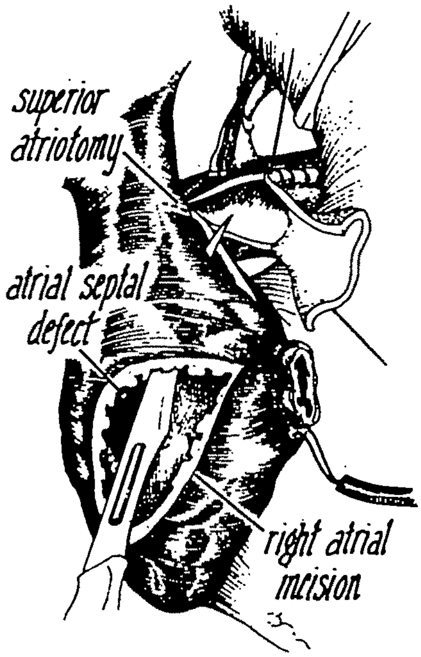


Fig 13. Second-stage Fontan: right atrial incision. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

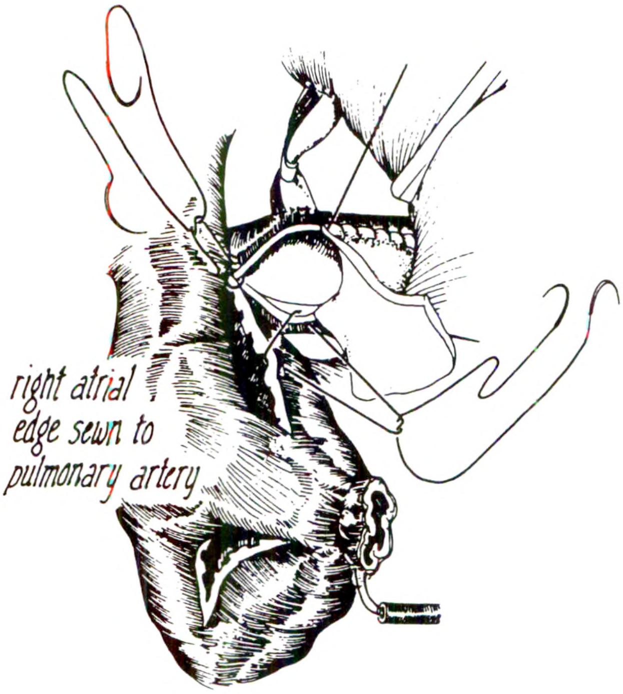


Fig 14. Second-stage Fontan: suture line between right pulmonary artery and right atrium. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

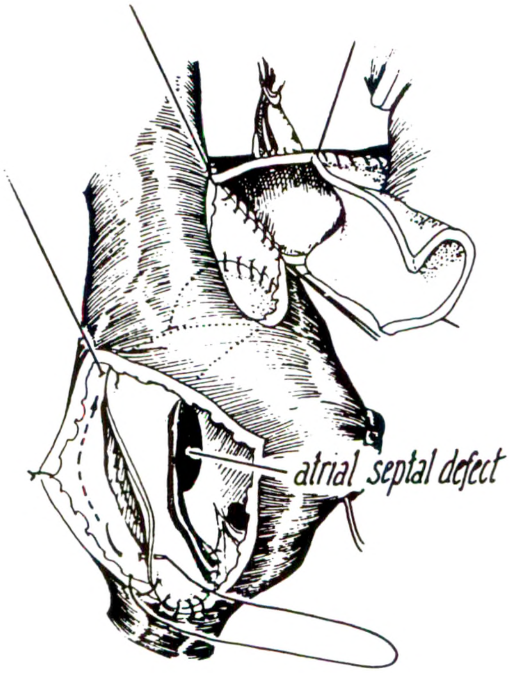


Fig 16. Second-stage Fontan: construction of baffle. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

who have undergone completion of the Fontan operation, there has been no mortality. At present, our management scheme is palliation of the newborn, hemi-Fontan procedure at 6 months of age, and completion of the Fontan procedure at 12 months of age. The approach outlined here for reconstructive therapy of this malformation will continue to evolve.

The challenge for the future is to characterize better this group of lesions so that the preoperative, operative, and postoperative management may be further improved.

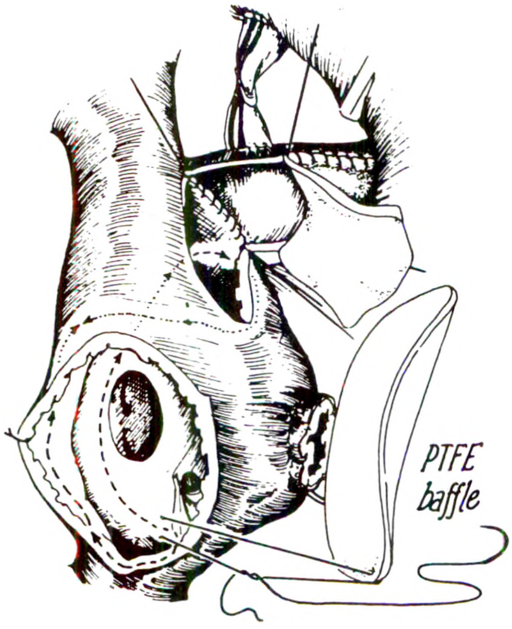


Fig 15. Second-stage Fontan: 10-mm polytetrafluoroethylene (PTFE) tube graft used as baffle. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

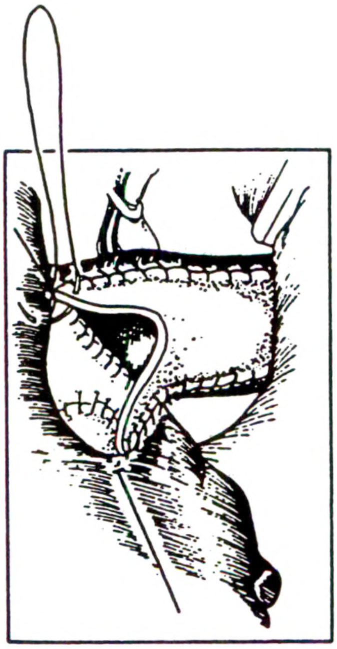


Fig 17. Second-stage Fontan: final suture line. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

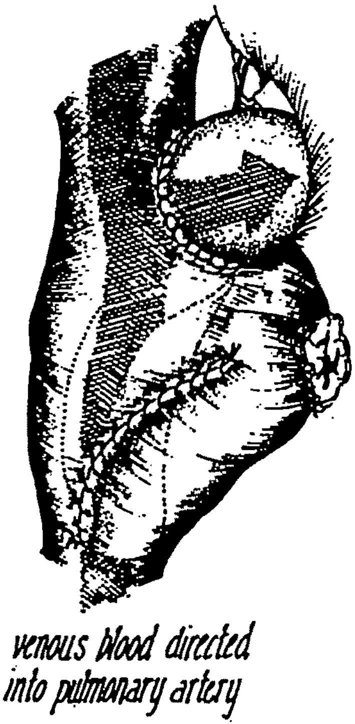


Fig 18. Second-stage Fontan: completed repair. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

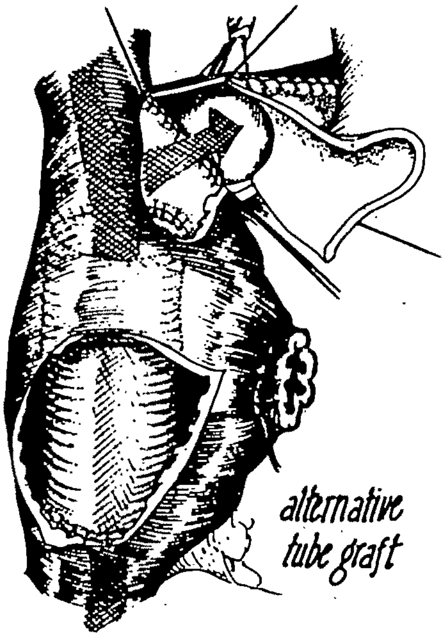


Fig 19. Second-stage Fontan: alternative tube graft. (Reprinted from Edmunds LH Jr, Norwood WI, Low DW. Atlas of cardiothoracic surgery. Malvern, PA: Lea & Febiger, 1989, by permission.)

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EDITOR'S NOTE

This article traces the important evolution of multiple changes in technique and management of the newborn with hypoplastic left heart syndrome. Norwood recently updated his results with 386 patients; 163 had completed a Fontan operation with 29 deaths (18%), with another 39 patients awaiting hemi-Fontan and 60 awaiting Fontan. He highlights the development from his "original" first stage, which incorporated an atrial septal baffle with 10 deaths (37%), to the use of an intraatrial Gore-Tex tunnel going from the inferior vena cava to the pulmonary artery in 80 patients with 15 deaths (19%). As noted in the final discussion in his article, these early deaths were attributed to the sudden conversion to a Fontan physiology with its rapid decrease in end-diastolic volume resulting in an abrupt change in ventricular geometry, with increased wall thickness and decreased diastolic compliance, leading to decreased pulmonary blood flow.

The more recent "routine" employment of a hemi-Fontan has resulted in a further spectacular decrease in the overall mortality. In this procedure, the superior vena cava is divided and both ends are anastomosed end-to-side to the right pulmonary artery—the superior end to the upper surface, and the cardiac end to the inferior surface of the right pulmonary artery. The intraatrial superior vena caval orifice is occluded with a patch. This is excised at a subsequent procedure when the patient is converted to a Fontan with an intraatrial Gore-Tex tunnel. This hemi-Fontan creates a bidirectional Glenn type of physiology but minimizes the dissection at the subsequent procedure.

Using this three-stage approach, Norwood has performed the hemi-Fontan in 106 patients with 6 deaths (6%); 56 patients have undergone the completed Fontan with only 4 deaths (7%) and only 1 death in the last 30 patients (his overall experience is 193 hemi-Fontan operations with 22 deaths [11%], with 3 patients undergoing transplantation rather than Fontan).

Norwood's recommendation is to perform the first stage in the newborn period, perform the hemi-Fontan at 6 to 8 months of age, and complete the modified Fontan operation at 12 to 18 months.

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Intermediate Procedures After First-Stage Norwood Operation Facilitate Subsequent Repair

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Actuarial analysis of survival after first-stage palliative reconstructive operation for hypoplastic left heart syndrome has revealed a high out-of-hospital attrition rate over the first 18 months to 2 years postoperatively. Some of this mortality is related to development of anatomical problems such as restrictive atrial septal defect, neoaortic arch obstruction, and pulmonary artery distortion. The bidirectional Glenn shunt has proved to be an ideal adjunctive procedure for high-risk patients at the time of operation to correct such intermediate-term problems. The fenestrated Fontan procedure, which involves fenestration of the interatrial baffle placed as part of our current standard Fontan procedure, is applied for patients considered to be at moderate risk for a Fontan procedure. The decision regarding closure of the fenestration is made by hemodynamic study including temporary balloon occlusion of the fenestration. The fenestration is closed with the double-clamshell device, which is placed percutaneously in the catheterization laboratory and which is currently used for secundum atrial septal defect closure. Appropriate selection of patients for the bidirectional Glenn shunt or fenestrated Fontan procedure with or without fenestration closure has resulted in a dramatic decrease in mortality and morbidity for patients with all forms of single ventricle and for patients with hypoplastic left heart syndrome.

(Ann Thorac Surg 1991;52:696–700)

This report focuses on the management of the child who has survived first-stage palliative surgical management of hypoplastic left heart syndrome and who must now be maintained as an optimal candidate for a Fontan operation. The principles that apply here are identical to those that should apply for any child with single-ventricle physiology who is ultimately to undergo a Fontan procedure. Careful follow-up early in infancy is critical to maintaining normal ventricular function, low pulmonary vascular resistance, and suitable development of the central pulmonary arteries. It is not sufficient today to perform a palliative procedure during the neonatal period and to expect the child to return as a satisfactory candidate for a Fontan procedure in 4 years.

Figure 1 illustrates the intermediate-term survival for 34 patients who underwent first-stage palliative operation for hypoplastic left heart syndrome in 1984 and 1985 [1]. The hospital mortality was approximately 35%. What is striking is the attrition rate over the next 18 months, such that by 18 months postoperatively, the overall survival was between 40% and 45%.

It is possible to blame this attrition on the severe degree of endocardial fibroelastosis that is frequently present in the small hypertensive left ventricle and which possibly is worse in patients with an atretic aortic valve and a patent mitral valve. Sauer and colleagues [2] have suggested that the latter group of patients, that is, those with aortic atresia and mitral stenosis, has a greater than 50% incidence of coronary artery abnormalities, particularly stenoses, which would appear to make this group at higher risk for both early and late death. My colleagues and I at Children's Hospital have been unable to substantiate increased risk in this subgroup. We [3] have preferred to focus our attention on the anatomical abnormalities that frequently result after first-stage palliative procedures (Fig 2). These various problems, as outlined here, can contribute to the observed attrition rate in the first 1 year to 2 years postoperatively.

Anatomical and Hemodynamic Complications of First-Stage Surgical Palliation

Restrictive Atrial Septal Defect

It was our initial belief that little more than an incision along the full length of the septum primum, analogous to the tear seen with a balloon septostomy, was all that would be required to maintain an adequate atrial communication under the circumstances of an obligatory left-to-right shunt at the atrial level. In fact, it has become clear that under these circumstances, there is a greater than usual tendency for fibrous obliteration to narrow any atrial septal defect (resulting in pulmonary venous obstruction), whether it be enlarged surgically or by blade or balloon septostomy [4]. Since we adopted the more aggressive approach of excising the septum primum in its entirety as well as part of the limbus of the fossa ovalis, extremely rarely have patients returned with a restrictive atrial septal defect. In the occasional patient, however, a fibrous obliterative process appears to be set up, which can progress to complete obliteration of the pulmonary

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Presented at the Current Controversies and Techniques in Congenital Heart Surgery Meeting, Baltimore, MD, Sep 8–9, 1989.

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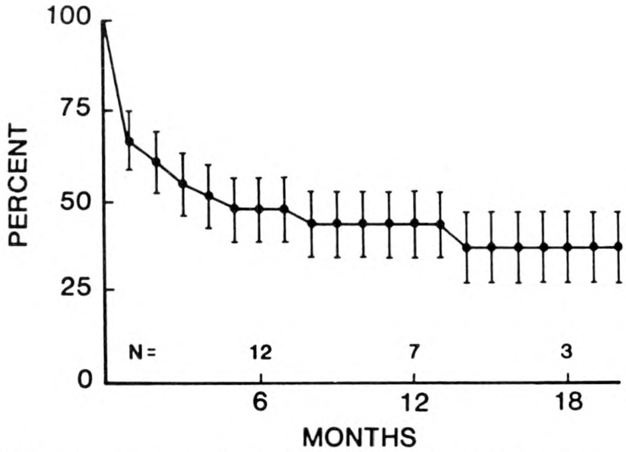

Fig 1. Actuarial survival including hospital deaths of 34 patients undergoing first-stage palliative reconstructive operation for hypoplastic left heart syndrome in 1984 and 1985.

veins, thereby rendering the patient quite unsuitable for a Fontan procedure and usually resulting in death.

Neoaortic Arch Obstruction

Early in our series, this problem was relatively frequent when we failed to appreciate the very high incidence of coarctation as part of hypoplastic left heart syndrome [1]. Since we began to use a more aggressive policy of neoaortic arch reconstruction including opening the proximal descending aorta up to 1 cm beyond the level of the coarctation shelf, this problem has been much less frequent. Avoidance of synthetic material has also played a role in decreasing the incidence of neoaortic arch obstruction [5]. Homograft tissue has no tendency to accumulate pseudointima, and unlike various pericardial substitutes including even autologous pericardium, there appears to be less tendency for homograft tissue to constrict and fibrose with time.

Balloon dilation of neoaortic arch obstruction has carried a relatively high morbidity and mortality in the context of hypoplastic left heart syndrome. Why this should be when balloon dilation of recurrent coarctation in other settings carries very little risk is not clear [6]. However, it does seem that neoaortic arch obstruction is a potent cause of ventricular dysfunction. It may be the combination of ventricular dysfunction and a shunt-dependent circulation that precipitates the cardiovascular collapse that has been witnessed on a number of occasions after attempted balloon dilation of neoaortic arch obstruction.

Care must also be taken in dealing with neoaortic arch obstruction in a closed fashion surgically. Currently, we prefer to employ deep hypothermic circulatory arrest and work through a median sternotomy in conjunction with placement of a bidirectional Glenn shunt, as will be discussed.

We [1] continue to use complete tube grafts as part of first-stage palliation for the occasional child who has a particularly small ascending aorta, that is, less than 2 mm in diameter. We have found that performing a side-to-side anastomosis between a proximal pulmonary artery 11 to 12 mm in diameter and an ascending aorta 1.5 mm in diameter is technically difficult. Such an anastomosis frequently results in coronary compromise. An alternative that we have used on occasion is to divide the ascending aorta and implant it end-to-side to the reconstructed neoaorta.

When a complete tube graft, either synthetic or homograft, has been used as part of the neoaortic arch reconstruction, the child ultimately will outgrow it. Because the tube is extremely short, generally less than 1 cm in length, it is surprising how much a child can grow before an important gradient develops across this area. Thus, by the time surgical intervention is needed, the original tiny ascending aorta has grown from a diameter of 2 mm to greater than 4 mm. We prefer to remove the tube graft in entirety, open out the ascending aorta as for a classic Norwood procedure, and perform a pulmonary artery to aorta anastomosis with supplementary homograft tissue as necessary, though it is often possible to achieve a direct anastomosis.

Pulmonary Artery Distortion

This is the most common and most serious anatomical problem that we have faced after first-stage surgical

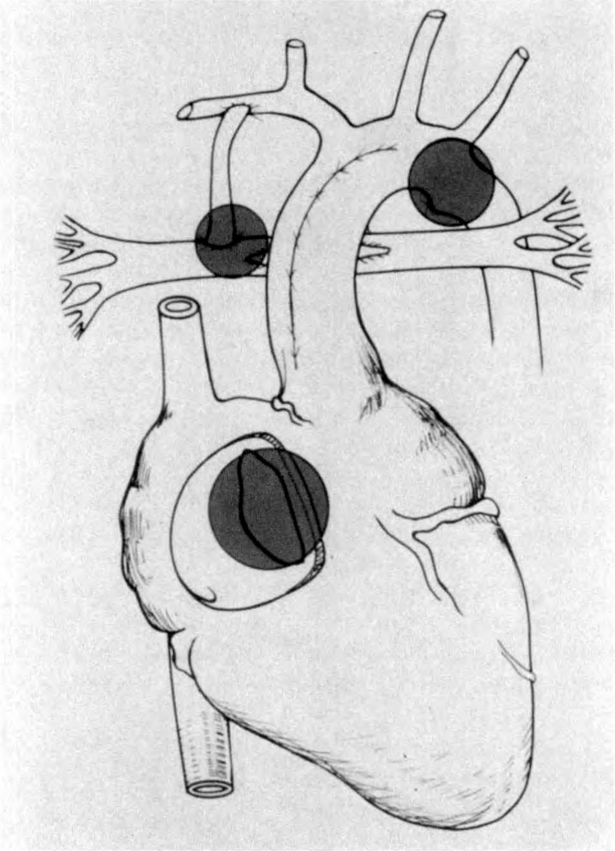

Fig 2. Late anatomical complications of first-stage reconstructive operation for hypoplastic left heart syndrome include restrictive atrial septal defect, neoaortic arch obstruction or coarctation, and pulmonary artery distortion.

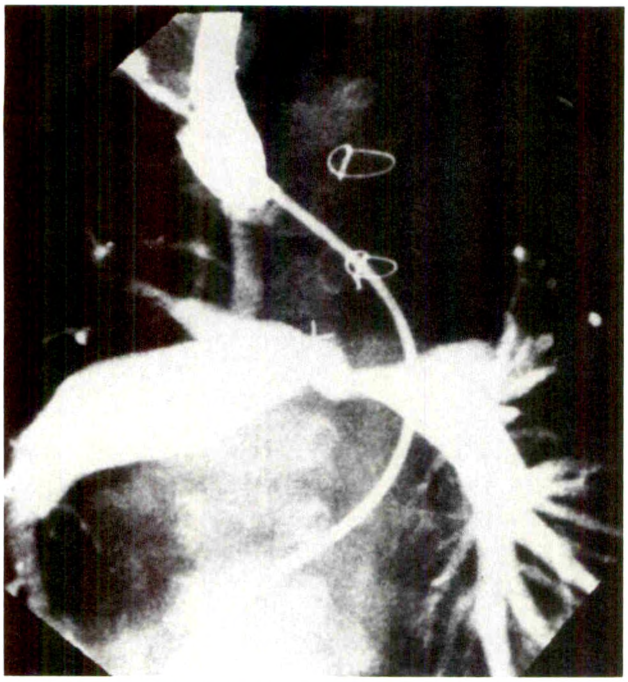

Fig 3. Typical central pulmonary artery distortion seen after first-stage surgical palliation of hypoplastic left heart syndrome.

palliation (Fig 3). It is our clinical impression that at the more severe end of the spectrum of hypoplastic left heart syndrome, the pulmonary arteries are at least moderately hypoplastic. This may be a reflection of decreased blood flow through the lungs in utero because of the left-sided obstruction. Consequently, encouraging uniform growth and development of the pulmonary arteries is essential during postnatal development. It is important to ligate the ductus as distally as possible and to divide the main pulmonary artery as proximally as possible, that is, very close to the pulmonary valve. We generally use a patch of homograft tissue to close the pulmonary arteries centrally. It is important that the neoaorta not be redundant, because this can cause posterior compression of the central pulmonary artery area.

Previous attempts to deal with pulmonary artery distortion were often disappointing in that there tended to be a temporary elevation of pulmonary vascular resistance after a period on cardiopulmonary bypass. Thus, to wean the patient from bypass, it was necessary to place a relatively large systemic to pulmonary artery shunt, which subsequently resulted in an excessive volume load for the single ventricle. Within months, this could result in ventricular dysfunction. This problem has been practically eliminated since we began to apply the bidirectional Glenn shunt in this setting.

Ventricular Dysfunction and Tricuspid Regurgitation

This is generally secondary to excessive pulmonary blood flow due to an overlarge systemic to pulmonary artery shunt or due to development of neoaortic arch obstruction.

Pulmonary Vascular Disease

Elevation of pulmonary vascular resistance to greater than 2 units is considered to be a risk factor for a Fontan procedure [7]. Although pulmonary vascular disease after first-stage management of hypoplastic left heart syndrome is rare bilaterally, it may be seen in the right lung if there is central pulmonary artery stenosis with a right-sided Blalock-Taussig shunt. Under these circumstances, most of the shunt flow is directed into the right lung.

Surgical Options

Bidirectional Glenn Shunt

One of the most serious problems in attempting to undertake an intermediate procedure such as pulmonary arterioplasty in the child with hypoplastic left heart syndrome whose pulmonary circulation is dependent on a systemic to pulmonary artery shunt is the intraoperative lability of pulmonary vascular resistance. This frequently results in a very cyanotic child in the early postbypass period and necessitates placement of an excessively large shunt, which subsequently leads to development of ventricular dysfunction.

The bidirectional Glenn shunt, in contrast, has proved to be remarkably free from this particular problem and has substantially decreased the risks of intermediate procedures [8]. It also offers the important advantage of decreasing the volume load on the single right ventricle on a long-term basis because it results in an increase in effective pulmonary blood flow. No longer is a mixture of blue and red blood passing through the lungs, but only systemic venous blood. The lower venous pressure to which the kidneys, liver, and gut are exposed facilitates function of these organs. Most remarkably, the incidence of pleural effusions has been practically zero in this high-risk group of patients, despite the fact that the superior vena caval pressure is often elevated to levels similar to those seen after a Fontan procedure, where in the past, pleural effusions were relatively common.

Between June 1988 and September 1989, 40 bidirectional Glenn shunts were undertaken at Children's Hospital in Boston for various forms of single ventricle including hypoplastic left heart syndrome. Only 1 child required takedown of the bidirectional Glenn shunt to a systemic to pulmonary artery shunt because of persistent cyanosis. Median arterial oxygen saturation at discharge was 0.85. The most frequent concomitant procedure was pulmonary arterioplasty. Even though these patients were not considered to be candidates for a Fontan procedure, frequently had distorted pulmonary arteries, and in some instances had elevated pulmonary vascular resistance or ventricular dysfunction, no patient had pleural effusion drainage for greater than 1 week.

Fenestrated Fontan Procedure

An alternative approach for patients whom we consider to have intermediate risk factors that place them between patients who could proceed directly to a completed Fontan procedure and those who are at such high risk that we undertake a preliminary bidirectional Glenn shunt is the so-called fenestrated Fontan procedure [9]. This procedure is identical to our current standard technique for the Fontan procedure with the addition of a hole or fenestration in the interatrial baffle. This baffle is generally constructed of polytetrafluoroethylene and directs inferior vena caval blood to the internal orifice of the superior vena cava [10]. The superior vena cava is divided at the level of the right pulmonary artery. Both the cardiac and cephalic end of the divided superior vena cava are anastomosed to the central pulmonary arteries (Fig 4). At present, we do not have any scientifically based formula to help us determine the size of the fenestration. In practical terms, the diameter of the hole has ranged between 4 and 7 mm in children whose weight has ranged from 8 to 25 kg, whose pulmonary vascular resistance has often been between 2 and 3 units, and whose central pulmonary arteries were generally no worse than mildly to moderately hypoplastic. Ventricular function has generally been at most mildly depressed. Concomitant pulmonary arterioplasty has been required in a number of patients. Usually, however, if a patient requires extensive additional surgical procedures such as neoaortic arch reconstruction as well as pulmonary arterioplasty, then we tend to complete that procedure with a bidirectional Glenn shunt rather than the more extensive fenestrated Fontan procedure.

Comparison Between These Two Options

There are clearly important physiological differences between the bidirectional Glenn shunt and the fenestrated Fontan procedure. In the latter, the abdominal organs are exposed to the same elevated venous pressure as the superior vena caval system and the pulmonary arteries. Although the incidence of pleural effusions has been lower than in patients having completed Fontan operations, the addition of the fenestration does not eliminate the risk of development of pleural effusions. Presumably, the mechanism by which patients benefit from a fenestration in this setting is related to their ability to maintain cardiac output in the face of temporarily elevated pulmonary vascular resistance. The fenestration also prevents development of very high right-sided pressures. These might occur in the patient who has an element of reversible ventricular dysfunction, which causes some elevation of the left-sided filling pressure with subsequent elevation of right-sided filling pressure. The principle of the fenestrated Fontan is similar to that employed by Laks with the "adjustable ASD" (Laks H; personal communication, 1989).

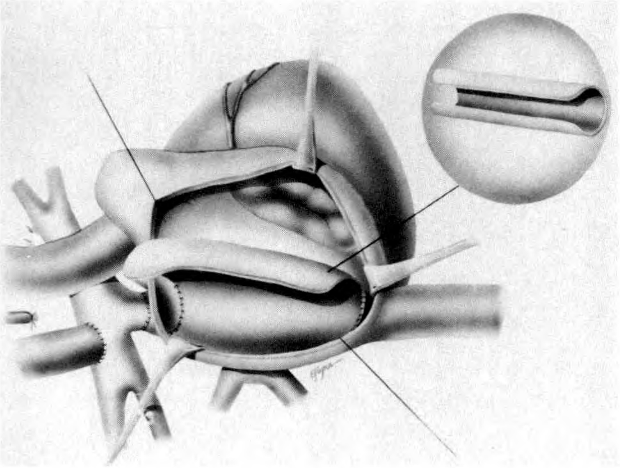

Fig 4. Current technique of modified Fontan procedure includes a double cavopulmonary anastomosis as well as placement of a polytetrafluoroethylene baffle to direct inferior vena caval blood to the internal orifice of the superior vena cava. A fenestration 4 to 7 mm in diameter is made in the baffle, thereby completing the so-called fenestrated Fontan procedure.

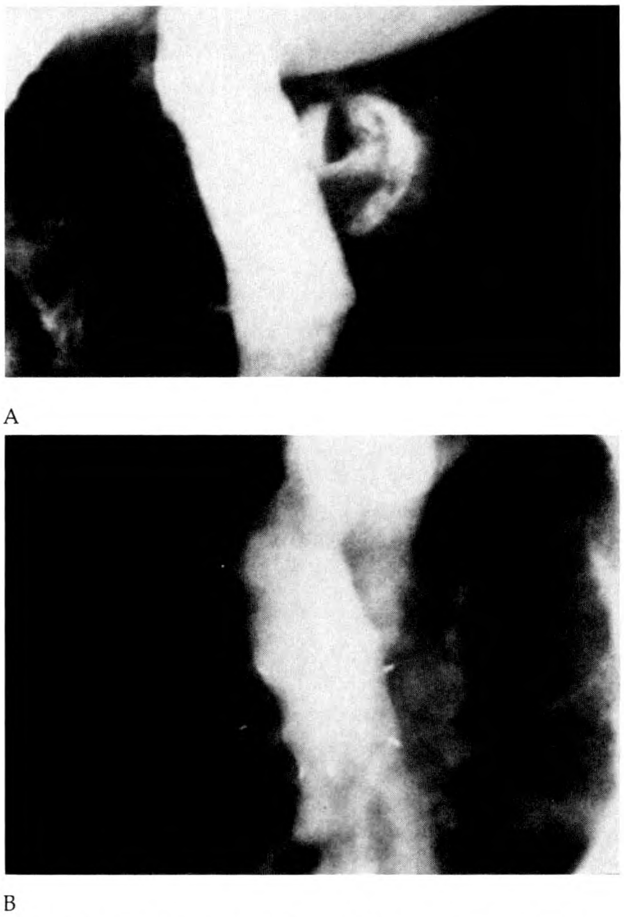

Fig 5. (A) Right-to-left shunt through baffle fenestration is illustrated. (B) Placement of a double-clamshell device has resulted in satisfactory occlusion of the fenestration.

The principal advantage of the fenestrated Fontan procedure, we believe, is that it allows thorough hemodynamic assessment in the catheterization laboratory when the child has fully recovered from the operation. In some patients, this will be as early as 1 week postoperatively, whereas in others, elective catheterization can be undertaken weeks to months postoperatively. At that time, temporary balloon occlusion of the fenestration permits assessment of change in cardiac output, change in atrial filling pressures, and arterial oxygen saturation. Of 20 patients undergoing the fenestrated Fontan procedure in

Table 1. Hemodynamic Data During Balloon Occlusion of Fontan Fenestration

Variable	Suitable[a] (n = 11)	Unsuitable[a] (n = 6)
Right atrial pressure increase (mm)	1.6 ± 3.4	4–6
Cardiac output decrease (%)	21	31–56
Arterial oxygen saturation increase (%)	8	7

[a] Hemodynamics were either suitable or unsuitable for permanent fenestration closure.

1989, 11 were considered to have suitable hemodynamics for permanent closure with the double-clamshell device, the same device currently used for closure of secundum atrial septal defects in the catheterization laboratory (Fig 5). Table 1 illustrates the hemodynamic differences between this group of patients and a second group of patients who were considered to have unsuitable hemodynamics to allow permanent fenestration closure. The latter patients are left permanently with the capacity to boost cardiac output during exercise, for example, by a right-to-left shunt at the atrial level. They pay a price in terms of a degree of arterial desaturation for the benefit of improved cardiac output. The median arterial oxygen saturation of patients having bidirectional Glenn shunts has ranged from 0.80 to 0.85, whereas the majority of patients having fenestrated Fontan procedures have had an arterial oxygen saturation in the 0.85 to 0.90 range. Permanent fenestration closure results in a mean increase of 0.08 in arterial oxygen saturation, which makes the arterial oxygen saturation close to normal.

Conclusion

The management of hypoplastic left heart syndrome continues to evolve at a rapid pace. Technical modifications of the Fontan procedure itself, and the development of intermediate procedures such as extensive patch pulmonary arterioplasty, the bidirectional Glenn shunt, and the fenestrated Fontan procedure, have all resulted from the critical needs of children with hypoplastic left heart syndrome. Not only have these modifications aided the specific population of children with hypoplastic left heart syndrome, but undoubtedly they have also improved the outlook for children with all forms of single ventricle.

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Transplantation After First-Stage Reconstruction for Hypoplastic Left Heart Syndrome

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The surgical treatment of infants born with hypoplastic left heart syndrome has received considerable attention in recent years. Although this lesion was previously considered uniformly fatal, dramatic successes have been achieved with the use of staged reconstructive and replacement therapies. However, both surgical options have benefits and limitations, and neither has demonstrated clear superiority over the other. As survival for first-stage reconstruction by the Norwood procedure has improved, a greater number of patients are potential candidates for the Fontan operation, but not all will be suitable at an acceptable risk. These patients may be treated by cardiac transplantation. The results of a treatment protocol for 90 patients with classic hypoplastic left heart syndrome or its variants at The University of Michigan are reported, with particular attention given to those patients surviving initial palliation but judged to be unsuitable for a subsequent Fontan procedure.

(Ann Thorac Surg 1991;52:701–7)

Hypoplastic left heart syndrome (HLHS) represents an anatomical spectrum of disease with varying degrees of underdevelopment of the left-sided cardiac structures [1]. In its most severe form, the aortic and mitral valves are atretic, and the ascending aorta is diminutive. Systemic perfusion is dependent on the patency of the ductus arteriosus, and the right ventricle provides blood flow to both the systemic and pulmonary vascular beds. Other important variants include less severe degrees of hypoplasia of the left ventricular cavity and aortic valve annulus as well as common atrioventricular orifice with severe right ventricular dominance. Anatomical studies [2, 3] have shown an increased incidence of coronary artery anomalies, particularly persistent ventriculocoronary connections, in patients with HLHS and a patent mitral orifice. Hypoplastic left heart syndrome is a common congenital anomaly, comprising 7% to 9% of all infants born with congenital heart disease, and is the most frequent cause of death from heart disease in the first month of life. Genetic disorders and major extracardiac anomalies are frequent and have been found in as many as 28% of patients [4].

Efforts to palliate HLHS in infants have focused on establishing unobstructed blood flow from the right ventricle to the systemic and coronary circulations by anastomosing the proximal pulmonary artery to the augmented ascending, transverse, and proximal descending aorta [5–7]. Pulmonary blood flow is provided through a controlled shunt, generally a 4-mm Gore-Tex conduit placed between the innominate artery or aortic arch and the central portion of the pulmonary artery. The atrial septum is excised to allow unobstructed drainage of the pulmonary veins to the tricuspid valve. These patients then become candidates to undergo complete physiological repair by a Fontan operation at an older age. For this staged approach to be successful, however, survivors of the first-stage reconstruction must be suitable candidates for the Fontan procedure according to well-established surgical principles [8–12]. Although the results of reconstructive operations by the techniques of Norwood have continued to improve in recent years, not all patients will be acceptable candidates for a Fontan procedure [13–17].

Replacement therapy for patients with HLHS by means of orthotopic cardiac transplantation has achieved considerable success in the past decade [18, 19]. A number of groups have claimed that transplantation is the optimal mode of therapy and can best be performed in the neonate, avoiding the need for palliative procedures. As the success of cardiac transplantation has improved, it is logical to apply this treatment to survivors of the Norwood procedure who, for whatever reason, are unsuitable for complete physiological repair. This report outlines the recent experience with surgical intervention, including both reconstructive and replacement therapies, for patients with HLHS at The University of Michigan Medical Center.

Patient Data

In July 1985, a policy of offering a palliative reconstructive operation to all patients with either classic HLHS or its variants seen in this institution was initiated. The rationale was that it is important to maintain a uniform protocol of treatment to evaluate the results in a mean-

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ingful way. With only one exception, this policy has been followed for all patients whose parents agreed to surgical intervention, regardless of preoperative anatomy or right ventricular function. The single exception was a 2-week-old neonate who underwent successful primary orthotopic cardiac transplantation at the request of the parents.

Reconstructive Procedures and Results

First-stage reconstruction by the Norwood procedure has been performed in 89 patients with 50 hospital survivors. Since January 1988, 36 (71%) of 51 patients have survived Norwood procedures. Early in our experience, my colleagues and I performed the Fontan procedure when the patient was approximately 12 months of age to reduce cyanosis and right ventricular volume overload as early in life as possible. This was done in 8 patients with HLHS, and 5 (63%) survived. However, the postoperative course even of the survivors was often difficult, and a policy of performing a bidirectional cavopulmonary shunt procedure between 6 and 12 months of life was initiated in October 1989. Since that time, 20 patients have undergone the bidirectional cavopulmonary shunt with one death.

Survivors Not Candidates for Fontan Procedure

In our experience, there have been 7 patients who survived the initial Norwood procedure but were believed to be unsuitable candidates for the Fontan operation. The first 3 of these patients were at home, were growing satisfactorily, and were asymptomatic, although cardiac catheterization demonstrated poor right ventricular function. All 3 patients died suddenly. The fourth patient was also judged not to be suitable for a Fontan procedure secondary to diminished right ventricular function. This patient was placed on the list for cardiac transplantation, but a suitable donor never became available. One year later, the patient's saturation had progressively declined to less than 0.50, and a Fontan operation was performed after all the options had been reconsidered. Although the preoperative risk factors predicted a high mortality, the patient has done very well.

The final 3 patients underwent transplantation. The first patient was in extremis at the time of transplantation and was receiving closed cardiac massage during preparation for cardiopulmonary bypass. The patient died 24 hours after operation secondary to extensive multiorgan failure. The second patient did well initially but died 2 months after transplantation secondary to graft dysfunction. The third patient underwent successful transplantation and remains well 2 years later. His case report follows.

Case Report

A male neonate was born with HLHS and underwent a Norwood procedure at 5 days of age. Recovery was uneventful, and he was discharged 11 days later. At 3 months of age, cardiac catheterization demonstrated major distal aortic arch obstruction, and reoperation was performed. Recovery was again uneventful. A second catheterization was performed 8 months later and showed poor right ventricular function. The patient was listed for transplantation and received a donor heart at age 14 months. His recovery was complicated only by two brief generalized seizures, presumably secondary to cyclosporine administration, and he was discharged 1 month after transplantation. Initial routine endomyocardial biopsy specimens were negative for rejection, but the results of a biopsy performed 3 months after transplantation demonstrated moderate rejection. A 3-day course of bolus steroid therapy was administered even though the patient was entirely asymptomatic. The subsequent biopsy specimen obtained after steroid therapy was negative, but the results of another biopsy 2 months later again demonstrated moderate rejection. This time, no further therapy was given, and all subsequent biopsy results have been negative. The patient remains well on a regimen of triple-drug immunosuppression consisting of prednisone, Imuran (azathioprine), and cyclosporine.

Comment

It seems clear that the results of surgical treatment for newborns with HLHS is improving. In our experience, 71% of patients undergoing first-stage reconstruction with a Norwood procedure since January 1988 have been hospital survivors (36/51). Refinements in operative technique as well as in postoperative management have been largely responsible for this improved outcome [5, 6, 13]. Reconstruction of the aortic arch well beyond the insertion of the ductus is essential to avoid residual obstruction. Even minor gradients can lead to a poor outcome by causing right ventricular pressure overload, increased shunt flow, diminished systemic flow, and persistent acidosis. The use of a relatively small shunt is also important to prevent ventricular volume overload. The low diastolic blood pressure resulting from too large a shunt can also decrease coronary blood flow, further impairing right ventricular function.

Proper ventilator management is crucial to achieve a balance between pulmonary and systemic blood flow. If arterial oxygen tension is low secondary to decreased pulmonary blood flow after operation, pulmonary vascular resistance can be decreased by hyperventilation to lower the carbon dioxide tension to less than 25 mm Hg and raise the pH higher than 7.5, by increasing supplemental oxygen administration, and by eliminating positive end-expiratory pressure. Patients with high pulmonary blood flow (generally with an oxygen tension greater than 45 mm Hg) and poor systemic perfusion should be managed in the opposite way, that is, by reducing ventilation to achieve a carbon dioxide tension between 40 and 45 mm Hg and a pH of approximately 7.35, by lowering the inspired concentration of oxygen to room air if necessary, and by increasing positive end-expiratory pressure to raise the overall pulmonary vascular resistance. We have also found that a continuous infusion of fentanyl is helpful in providing analgesia as well as in preventing labile pulmonary hypertensive changes.

Despite the improvement in early survival, not all of our patients have been suitable for a subsequent Fontan procedure. Currently, factors predictive of an increased mortality after the Fontan procedure or even contraindicating its use include an elevation in pulmonary vascular resistance above 3 units, right ventricular dysfunction, pulmonary artery hypoplasia, and moderate or severe tricuspid valve regurgitation. The most common finding precluding a Fontan repair in our experience was right ventricular dysfunction. This can be due to any number of factors, including preoperative ischemia and infarction [20], poor intraoperative myocardial protection, continuing ischemia postoperatively, persistent volume overload from the systemic to pulmonary artery shunt, and pressure overload secondary to right ventricular outflow tract obstruction. Most survivors of the first-stage procedure can be expected to have any number of these factors. It is hoped that the use of an intermediate stage in the reconstructive process, the bidirectional Glenn procedure, will further improve the overall result by early elimination of remaining risk factors. Reducing the volume overload on the right ventricle early after the first stage may well protect the right ventricle from continued fibrosis and hypertrophy, increasing the likelihood of a successful Fontan operation.

Both surgical treatment options for infants with HLHS possess benefits as well as limitations. Transplantation as a primary procedure performed in the neonate offers the benefits of normal hemodynamics and oxygen saturations, something that cannot be achieved with any form of palliation. Possibly, transplantation at such an early age may confer a degree of immune tolerance, a supposition that remains unproven and has not been supported by the clinical experience of some groups [19]. The major risks involved relate to the lack of available donor hearts and the substantial mortality while waiting for a transplant, and to the long-term consequences of immunosuppression.

Staged reconstruction eliminates the need for immunosuppression and is applicable to nearly all patients, although its major drawback remains the high operative mortality. Late mortality is also considerable, owing to the persistent cyanosis and volume overload of the right ventricle. Both treatment modalities are beneficial, and the clear superiority of one over the other has not been shown. It is also clear that transplantation can be successfully applied following the Norwood procedure when the hemodynamic result predicts an unacceptably high risk for a Fontan operation. If, however, the Fontan procedure appears suitable on the basis of hemodynamic assessment after first-stage reconstruction, then the use of an early bidirectional Glenn procedure may substantially lower the subsequent operative risk in these patients. Nevertheless, not all survivors of the first stage will become candidates for complete repair, and it seems reasonable to consider them for transplantation.

Our experience indicates that these patients should undergo early catheterization after the Norwood procedure (3 to 6 months) to evaluate their suitability for a subsequent Fontan procedure. If transplantation is considered the best option, generally because of poor right ventricular function, delay in listing must be avoided, as donor availability remains limited. Clearly, transplantation cannot be done as an emergency procedure and must be performed before serious clinical deterioration occurs. Furthermore, the excellent clinical course in our single long-term survivor appears similar to that reported for many patients undergoing transplantation in the neonatal period. Our patient received bolus steroid therapy on only one occasion to treat asymptomatic biopsy-proven moderate rejection. It is entirely possible that a policy of only clinical surveillance without routine, periodic biopsies would have missed this subclinical rejection episode.

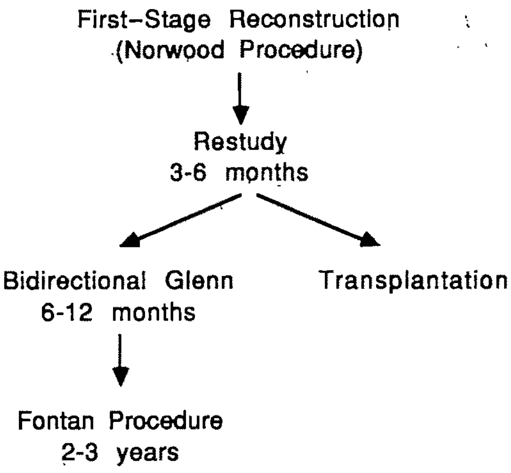

Fig 1. University of Michigan protocol for hypoplastic left heart syndrome.

The technical considerations required for transplantation after a Norwood procedure have not proved difficult. The donor aorta is easily sutured to the reconstructed recipient arch, leaving the previous homograft patch undisturbed. The atrial septum is reconstructed with donor tissue, "borrowing" from the right atrium to enlarge the small left atrium of the recipient. Finally, although augmentation of the branch pulmonary arteries is generally required, this can easily be done using readily available donor pulmonary artery.

In summary, the two treatment options currently available for patients born with HLHS offer considerable promise. Neither has demonstrated clear superiority, and overall survival appears comparable. The results after first-stage reconstruction are improving, and the use of intermediate cavopulmonary shunt procedures may well lower the risks for the subsequent Fontan operation. It remains our policy at The University of Michigan to offer staged reconstruction beginning with the Norwood procedure for neonates with HLHS (Fig 1). Patients with serious right ventricular dysfunction or tricuspid valve regurgitation, a rare occurrence, are offered transplantation as a primary procedure. Survivors of the first stage undergo complete cardiac evaluation between 3 and 6 months of age. Ideally, a bidirectional Glenn procedure is performed before 6 to 12 months of life and a Fontan procedure, by 2 to 3 years. For survivors of the first stage who are not candidates for complete repair by the Fontan approach, transplantation can still be offered as a suitable option.

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COMMENTARY: Neonatal Heart Transplantation: Lessons Learned

My colleagues and I have performed cardiac transplantation on 5 babies with hypoplastic left heart syndrome at the University of Iowa. Four were newborns, and 1 had undergone a previous Norwood procedure. From this experience, and from advice provided by Dr Leonard Bailey when he visited us, we have learned some lessons that have improved our results.

We think that it is important to give the cardioplegia to the hearts of newborns very gently. Thus, we allow it to run slowly at 30 cm H_2O pressure. Also, we agree with Bailey that it may be important to use crystalloid cardioplegia to wash out the red cells and avoid capillary sludging. In our initial experience, when we did not follow these precautions, we were troubled with low cardiac output and had to open the sternum in 1 baby, a problem others [1] also have encountered. Since adopting these principles, we have not seen these complications.

Initially we shared the belief that many newborn hearts might be more sensitive to ischemia than older hearts; therefore, we insisted on on-site donors. In fact, the opposite may be the case. Bailey and associates have had success with ischemic times in excess of 8 hours. We have had similar success with ischemic times between 4 and 5 hours. I do not really think that ischemic time in the newborn heart is a critical issue within these limits.

Most patients with hypoplastic left heart syndrome have either an actual coarctation or the potential of developing one later. This being the case, the arch should be completely reconstructed well beyond the ductus arteriosus at the time of transplantation. Thus, the arch anatomy becomes irrelevant, and there is no need for preoperative aortography, which is invasive and possibly hazardous. The operation can be performed based entirely on echocardiography. The most difficult aspect of this operation is extending the patch far enough beyond the ductus arteriosus. Although it is tempting to use the innominate artery of the donor for recannulation, it is necessary to be certain to leave enough flap of aorta distally to be able to reconstruct the coarctation site.

Following the lead of Bailey and Yacoub, we have not used steroids as part of the immunosuppression of newborns, but rather rely entirely on cyclosporine and Imuran (azathioprine). Cyclosporine toxicity has not been the problem that it is in adults; we have not had problems with hypertension or impaired renal function. It has been pointed out that cyclosporine must be given in larger doses and more frequently in newborns than in adults to achieve reasonable blood levels. There can be no doubt that rejection is infrequent and readily managed in newborns. We have not documented rejection in any of our patients, although we have given each patient steroid pulses when we were concerned about the clinical condition. Thus we agree that routine biopsies are unnecessary. Again following Bailey's lead, we have discontinued Imuran for our first newborn heart transplant patient.* She is now 3 years of age and on a regimen of only low-dose cyclosporine.

We have been struck by the contrast between these newborns and the 1 patient having transplantation after a previous Norwood procedure. This patient has had troublesome rejection and has required not only steroids but also methotrexate sodium. Although from a technical standpoint transplantation can certainly be accomplished after a previous Norwood operation, I am not sure that a strategy of deliberately using this as a way to stage a subsequent transplantation is advisable. In so doing, the surgical team may lose the window of immunological opportunity that the newborn may present.

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Editor's note (Dr Anthony L. Moulton): After several sudden episodes of severe rejection and sudden death, the Loma Linda group has generally resumed maintenance Imuran in their patients.

COMMENTARY: Orthotopic Cardiac Transplantation in Neonates

My colleagues and I do not have the extensive experience that has developed over time at Loma Linda. We started about 3½ years ago in Louisville, and since my move to Chicago, 5 patients have been added during the last year. The oldest survivor is 3½ years old and is doing well. We thus have a total of 16 patients with a 75% survival.

I am not going to repeat the recipient criteria, which are similar to those of the Loma Linda group, but I will explain how we choose donors who are larger than the neonatal recipient. We measure the length in centimeters from the right atrial border to the tip of the right ventricle and have obtained good correlation between recipient and

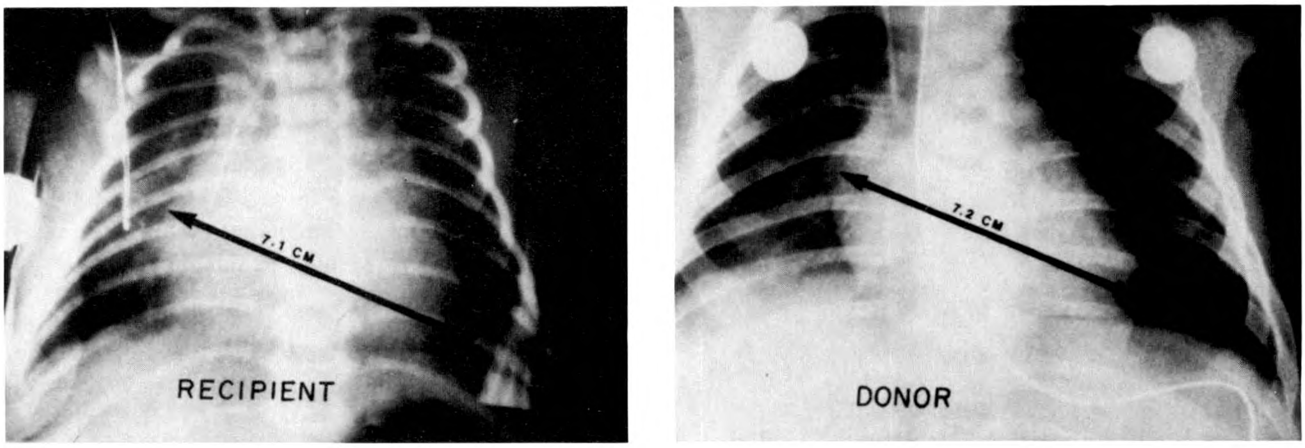

Fig 1. Recipient and donor anteroposterior chest roentgenograms taken by standard portable equipment with a 36-inch focal distance. Arrows *show the direction (mid-right atrium to apex of the heart) and magnitude of measurements. When donor-recipient weight variance was greater than 1,000 g, this method of size evaluation proved helpful. (Reprinted from Mavroudis C, Harrison H, Klein JB, et al. Infant orthotopic cardiac transplantation. J Thorac Cardiovasc Surg 1988;96:912–24, by permission of Mosby-Year Book.)*

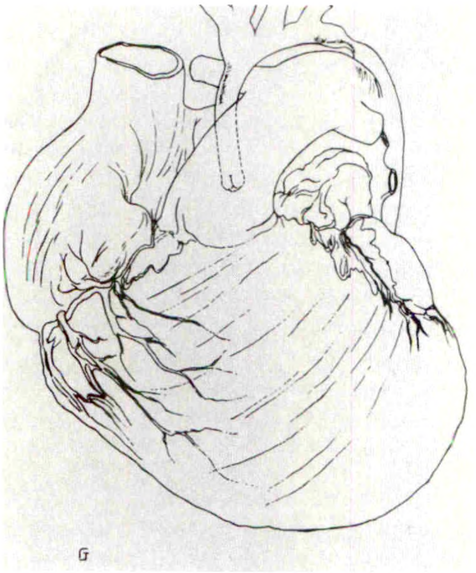

Fig 2. Type I hypoplastic left heart syndrome characterized by hypoplastic left heart, hypoplastic aortic arch, and coarctation of the aorta. (Reprinted from Kron IL, Mavroudis C, eds. Innovations in congenital heart surgery. Philadelphia: Hanley & Belfus, 1989, by permission.)

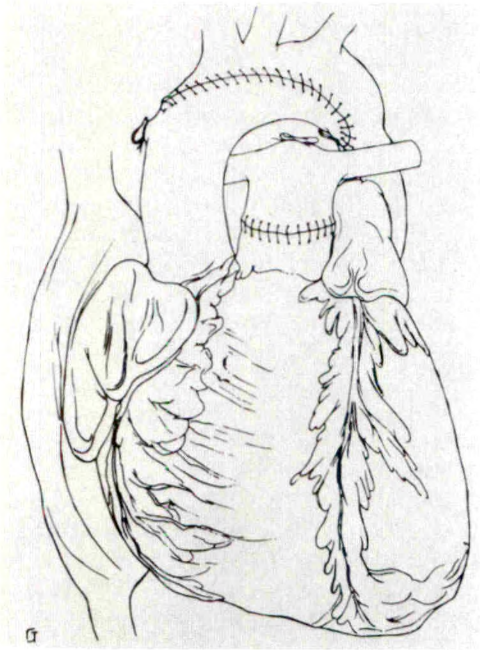

Fig 4. Aortic arch reconstruction performed in conjunction with orthotopic cardiac transplantation for type I patients. (Reprinted from Kron IL, Mavroudis C, eds. Innovations in congenital heart surgery. Philadelphia: Hanley & Belfus, 1989, by permission.)

donor. In one instance, for example, the measurements were 7.1 cm for the recipient and 7.2 cm for the donor (Fig 1). The donor weighed 6.5 kg and the recipient, 4 kg. The operation was uncomplicated, and the heart fit very well. We prefer to use this cardiac measurement as a guide to avoid donor-recipient size discrepancies.

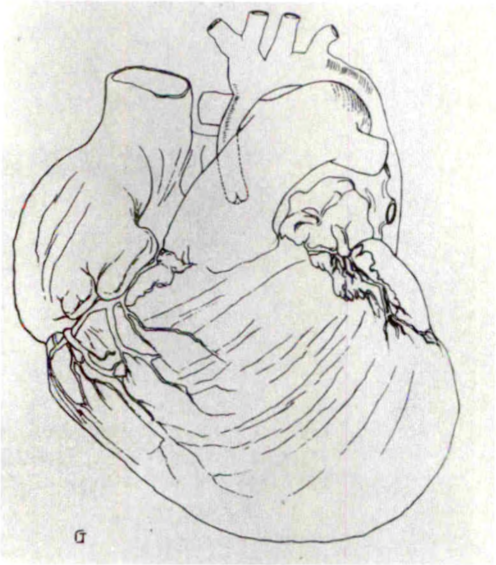

Fig 3. Type I hypoplastic left heart syndrome characterized by hypoplastic left heart, hypoplastic aortic arch, and relative coarctation of the aorta. (Reprinted from Kron IL, Mavroudis C, eds. Innovations in congenital heart surgery. Philadelphia: Hanley & Belfus, 1989, by permission.)

One other point must be made about these patients. Figure 2 is a drawing of hypoplastic left heart syndrome, which includes a small ascending aorta and a coarctation, which in this case is discrete. In Figure 3, a similar form is shown with a relative coarctation caused by the size discrepancy between the transverse arch and descending aorta. We call these variants type I hypoplastic left heart syndrome. These patients will require the usual operation, which Dr Bailey has described, with an extensive arch reconstruction and an end-to-end pulmonary artery anastomosis (Fig 4). However, we and others have had some patients with a relatively good-sized ascending aorta (Fig 5). We call this type II hypoplastic left heart syndrome. We have arbitrarily defined the ascending aorta "adequate" if the ascending aorta measures 0.8 of the descending aorta right below the inflow of the ductus. I have no supporting reason for this except that it worked in 2 of our patients. Transplantation in these patients required the usual classic end-to-end anastomosis (Fig 6). Clearly the circulatory arrest time will be shorter, as no arch reconstruction is necessary. Care must be taken, however, to ascertain the absence of a coarctation, which could adversely affect the outcome if not recognized.

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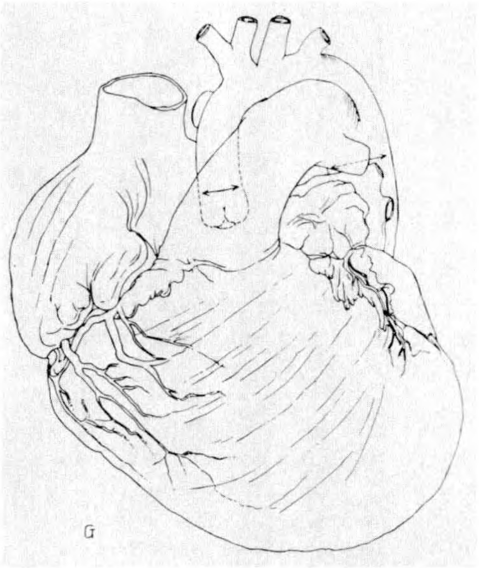

Fig 5. Type II hypoplastic left heart syndrome characterized by hypoplastic left heart and adequately sized ascending aorta, which is defined by a ratio between the diameter of ascending aorta, just below the innominate takeoff (arrow)*, and the descending aorta, just downstream to the ductus arteriosus* (arrow)*, of 0.80 or greater. The aortic valve may be fused (aortic atresia and hypoplastic left heart) or stenotic (critical aortic stenosis and hypoplastic left heart). (Reprinted from Kron IL, Mavroudis C, eds. Innovations in congenital heart surgery. Philadelphia: Hanley & Belfus, 1989, by permission.)*

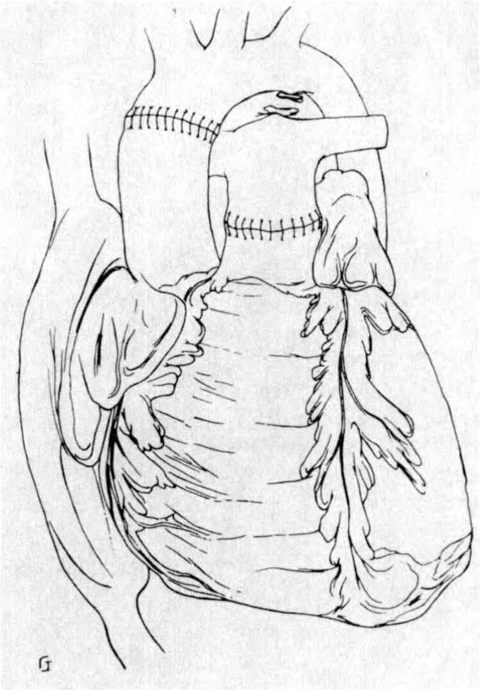

Fig 6. Aortic anastomosis performed in conjunction with orthotopic cardiac transplantation for type II patients. (Reprinted from Kron IL, Mavroudis C, eds. Innovations in congenital heart surgery. Philadelphia: Hanley & Belfus, 1989, by permission.)

IV. Pediatric Heart Transplantation

Pediatric Heart and Heart-Lung Transplantation

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Cardiac transplantation is the best therapeutic option available to the adult patient with end-stage heart disease. Most centers are reporting survival after heart transplantation that approaches 85% at 1 year and 75% at 5 years. This is in sharp contrast to the less than 40% 1-year survival for medically treated patients with New York Heart Association class IV symptoms and ejection fractions of 0.20 or less [1, 2]. The efficacy of heart transplantation has been clearly established in adult patients and has permitted this procedure to be extended well beyond desperately ill patients. This is further supported by the fact that since the introduction of cyclosporine in 1981, there has been approximately a tenfold increase in both the number of procedures performed and the number of worldwide heart transplantation centers. The registry of the International Society for Heart Transplantation (ISHT) reported that more than 12,600 heart transplantations were performed as of December 31, 1989, with approximately 2,400 procedures done in 250 worldwide centers in 1989 [3].

However, the application of cardiac transplantation to the pediatric population has not yet evolved to the degree that it has in the adult population, despite the fact that the procedure was first applied clinically in 1967. In that year, Kantrowitz and his team [4] transplanted the heart of an anencephalic newborn into a 2-week-old infant with tricuspid atresia. The child survived only a few hours. Between 1967 and 1980, cardiac transplantation was infrequently attempted in pediatric patients. A review of the data from the registry of the ISHT shows that before 1980, fewer than 5 children each year received cardiac transplants, and the majority of these children were adolescents. Since 1981, there has been a steady increase in the number of pediatric procedures coincident with the improved life-style and survival attributed to cyclosporine immunosuppression.

The survey data presented by Pennington and associates in this issue of *The Annals* [5] confirm the recent trends in pediatric cardiac transplantation identified by the registry of the ISHT. First, the number of pediatric heart transplantations performed annually continues to increase, and this increase has occurred primarily as a result of infant transplantation. Second, the overall 1-year survival now approximates 76% for children older than 1 year. Third, congenital heart disease is a growing indication for pediatric heart transplantation and now accounts for more than 40% of patients having transplant procedures. Perhaps this survey will soon provide answers to the questions that were designed to distinguish it from the ISHT registry.

For example, the survey data do not yet allow preliminary conclusions with respect to actuarial survival by patient age, growth after transplantation, or the incidence of infection and rejection in pediatric patients. Nevertheless, an attempt is made to contrast the incidence of rejection in neonates with that of children and adults. It is difficult to interpret these data since the technique employed in the diagnosis of rejection in neonates is entirely different from that in children and adults. The neonates' small size precludes safe and repetitive endomyocardial biopsies. Therefore, rejection in neonates is largely a clinical diagnosis, made independently of the gold standard of endomyocardial biopsy, and hence is easily and understandably subject to misdiagnosis.

It has been estimated that 10% to 20% of all patients with congenital heart disease will eventually become candidates for heart or heart-lung transplantation [6]. Most of these children will be unsuitable for palliative or corrective procedures either because of severe ventricular dysfunction or because they have exhausted all available reparative surgical options. Because many of these patients will have previously undergone corrective or palliative procedures, the surgeon must be prepared to manage the distorted anatomy created by prior operations as well as the underlying complex congenital anatomy. It has been our contention that there is no congenital cardiac malformation that precludes successful orthotopic cardiac transplantation.

We have performed 54 cardiac transplantations in 52 pediatric patients at Columbia-Presbyterian Medical Center in New York [7]. Nineteen patients (37%) had complex congenital heart disease: corrected transposition of the great arteries (4); single ventricle variants (8) including one with visceroatrial situs inversus and two with hypoplastic left heart syndrome; Ebstein's anomaly (1); ventricular septal defect (2); tetralogy of Fallot (1); atrioventricular canal (1); and coarctation of the aorta with left ventricular outflow tract obstruction (2). Fifteen (79%) of these patients had undergone 30 prior operations (14 palliative and 16 corrective). There were no deaths attributable to surgical technique, and the operative mortality and actuarial 1-year survival equaled those of patients with a diagnosis of cardiomyopathy who were transplanted.

Cardiac transplantation for congenital heart disease is

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both a reparative and a replacement operation; the intracardiac malformation is replaced, and the extracardiac abnormality is repaired. In our series of pediatric heart transplants for congenital heart disease, the most frequently encountered anatomical problem was distortion of the pulmonary arteries secondary to previous bands, shunts, conduits, and Fontan connections. To ensure an excellent functional result, this distorted anatomy must be precisely corrected and, where possible, we have used excess donor tissue for the repair. Many reconstructive techniques have been described, and in this issue of *The Annals*, Menkis and associates [8] and Chartrand [9] present several important techniques they have employed in their respective institutions. For example, they describe valuable techniques for tailoring the donor and recipient atria to overcome size discrepancies, and they point out the importance of extensive mobilization of the aorta and pulmonary artery in cases of malposition and transposition of the great arteries.

Visceroatrial situs inversus is one of the more technically challenging congenital heart lesions for the transplant surgeon. As with any anomaly of systemic and pulmonary venous connection, the repair must be reproducible, simple, and free from potentially distorting effects of the reconstruction. Our experience with this lesion has led us to advocate systemic and pulmonary venous rerouting using two intraatrial baffles created from the free wall of the left-sided atrium.

Finally, the ultimate success of these complex reconstructive procedures is critically dependent on constant communication between the donor and recipient surgical teams. To minimize graft ischemic time, the recipient surgeon must be prepared to implant the donor heart upon its arrival in the operating room. Therefore, recipient cardiectomy and reconstructive procedures often must precede the arrival of the donor heart.

At the symposium "Current Controversies and Techniques in Congenital Heart Surgery," Dr Magdi Yacoub reviewed his impressive experience with heart-lung transplantation in children. He emphasized the evolving issues in heart-lung transplantation, such as long-term steroid immunosuppression, the neonate as an immunologically tolerant host, techniques for preservation of the donor axis, the etiology and management of obliterative bronchiolitis, and the indications and contraindications for heart-lung transplantation. His discussion is not included in this Supplement, and editorial space considerations prevent us from reviewing each of these issues. However, because the indications for heart-lung transplantation are evolving so rapidly, we believe that they merit discussion. It has been the policy of many institutions, including our own, to offer heart and lung transplantation to all patients with either primary pulmonary hypertension or the Eisenmenger syndrome. In Dr Yacoub's series, 32 of the 49 heart-lung recipients underwent transplantation for either primary pulmonary hypertension (8) or the Eisenmenger syndrome (24).

Our recent experience leads us to question whether heart-lung transplantation remains the operation of choice for all of these patients. We have performed single-lung transplantation in 5 patients with primary pulmonary hypertension and single-lung transplantation combined with closure of a ventricular septal defect in 1 patient with the Eisenmenger syndrome. Immediately after transplantation these patients demonstrated a dramatic decrease in pulmonary artery pressure to less than half systemic pressure with oxygen partial pressures exceeding 400 mm Hg. As a result, we have been encouraged to pursue single-lung transplantation in carefully selected patients with these diagnoses.

In this era of donor organ shortage, every effort must be made to use the donor heart-lung axis for the benefit of multiple recipients. We believe that single-lung transplantation for primary pulmonary hypertension and the Eisenmenger syndrome (when combined with closure of the cardiac defect) will safely and effectively expand the pool of donor organs.

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Heart Transplantation in Children: An International Survey

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A survey of cardiac transplantation in children provided data from 381 transplantations in 362 patients from 32 centers in the United States and ten international centers. The number of transplantations continues to increase, in part because of transplantations in infants with hypoplastic left heart syndrome and patients with congenital defects. The immunosuppression regimens were more uniform than in the 1985 survey, and triple therapy was most common. Actuarial survival rates were 85% at 1 month, 72% at 1 year, 64% at 3 years, and 60% at 5 years. However, these improved rates are still not equal to the survival of the overall cardiac transplant population, in part because of lower survival rates in neonates. Ventricular dysfunction and rejection, rather than infection, were the leading causes of death. Rejection and infection were the most frequent complications. Also common were hypertension (39%) and seizures (25%), whereas coronary artery disease (8%) was unusual. Functional results were excellent in 85%, and only 7% were disabled. Questions concerning growth rates and many other aspects cannot yet be answered. However, it is apparent that cardiac transplantation in the pediatric population is a very worthwhile endeavor.

(Ann Thorac Surg 1991;52:710–5)

In the early 1980s, the performance of heart transplantation in children was a controversial issue, which had not been resolved because of the paucity of published data [1–3]. In 1985, to better analyze the status of heart transplantation in children, we [4] conducted a survey that provided information about 95 children undergoing cardiac transplantation. Since that time, there have been many new developments in the indications for and the outcome of transplantation, particularly in regard to heart transplantation in infants [5, 6]. These exciting events prompted us to repeat the survey in 1990 in an attempt to broaden our database and identify current trends.

Material and Methods

Data again were collected for heart transplant recipients who were 18 years old or younger at the time of transplantation. The questionnaire was similar to that of the previous survey and included information about the patient's vital statistics, diagnosis, preoperative condition, donor information, immunosuppression regimen, postoperative complications, long-term follow-up including evaluations of growth and quality of life, and survival rates. The data again were analyzed by age groups at the time of transplantation. The increased activity in infants allowed separate categorization of the newborn to 1-year-old group.

Questionnaires were sent to 55 worldwide cardiac transplantation centers that had previously participated in the study, published data regarding children, or were thought to have performed transplantations in this age group. Forty-two centers responded to the questionnaire, 32 in the United States and ten international centers. Information was collected on 362 patients who underwent 381 transplantations. In Figure 1, it is obvious that our survey patients do not represent the entire population of patients in this age range having transplantation. Data from the International Society for Heart Transplantation (ISHT) Registry [7] suggest that almost twice as many transplantations were done in this age group in the United States alone. However, the information in the registry is limited in scope. The age distribution of the children undergoing transplantation illustrates the sharp increase in patients less than 1 year of age (Fig 2). Ninety-eight percent of the 362 children underwent orthotopic transplantation, and 2% had heterotopic transplantation. The indication for transplantation was cardiomyopathy in 62%, congenital defects in 36%, and other in 2%. The types of cardiomyopathy are shown in Table 1; more than half of the patients with cardiomyopathy had idiopathic cardiomyopathy.

Congenital heart defects accounted for a much larger percentage of the indications than in our 1985 survey (36% versus 17%), in large part because of the increased number of patients with hypoplastic left heart syndrome. It is also obvious that the complexity of the congenital defects is much greater than it was for those reported in 1985 (Table 2). Furthermore, 70 of the 290 patients about whom we have information had undergone a previous cardiac operation. Fifty-five percent of the patients were listed as United Network for Organ Sharing (UNOS) status I and 45% as status II.

The donors were obtained from long distance in 74% of

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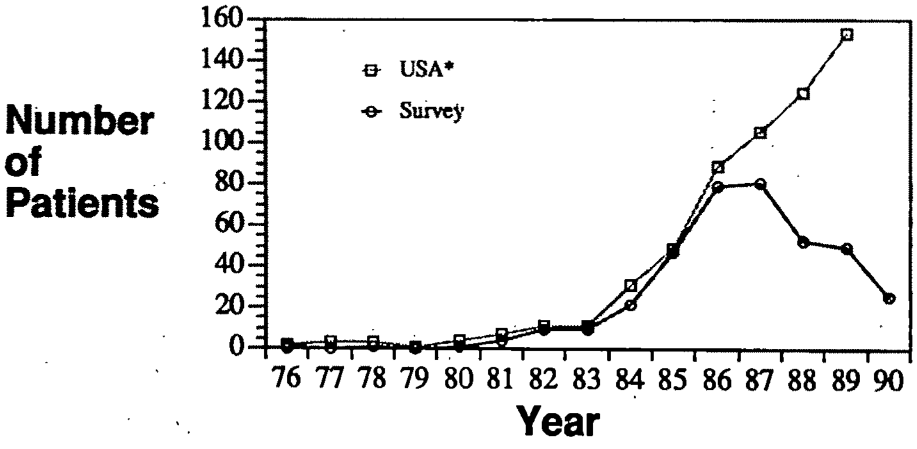

Fig 1. Number of cardiac transplantations by year in patients 18 years old or younger for the current international survey and for the United States. (= data from International Society for Heart Transplantation Registry.)*

the cases and locally in 18%; in 8%, the donor was transported to the transplanting hospital. The average waiting period for a heart was 26 days with a range of 1 day to 176 days. Only one xenograft was reported in this survey. The immunosuppression regimens were considerably more uniform than in our previous survey in that more than half of the patients received triple therapy with cyclosporine, prednisone, and azathioprine (Table 3). The methods of diagnosing rejection were quite varied. Although the majority of patients underwent endomyocardial biopsy, many patients in the infant group did not undergo biopsy, and the diagnosis of rejection was made by noninvasive means including clinical observations, echocardiography, electrocardiography, magnetic resonance imaging, and immunological testing.

Results

The actuarial survival rates for the entire population of the survey are similar to those from the ISHT Registry for patients in this age group (Fig 3). When the survival rates were examined by age group, there were some differences. For example, the 1-month and 1-year survival rates in the group of patients 1 month of age or less were lower than those of the other groups (Fig 4). The most frequent causes of death were different from those in the 1985 survey (Table 4). The most common causes of death were ventricular failure and acute rejection, with infection as the third most common cause. However, if the causes of acute and chronic rejection are combined, rejection becomes the most common cause of death.

As expected, the most common complications were those of rejection and infection. Among the 256 patients for whom sufficient information regarding rejection was submitted, there were 440 episodes of rejection, an average of 1.7 rejections per patient. However, 28% of the patients had no rejection at all, 29% had one episode, 17% had two episodes, and 26% had three or more episodes. The incidence of rejection was not related to age in that rates were similar among the age groups. Neonates (1 month of age or younger) did not demonstrate evidence of tolerance, as their incidence of rejection was 1.5 rejections per patient. The rejection episodes were treated with steroids in 70% of the instances, antilymphocytic agents in 10%, a combination of steroids and antilymphocytic agents in 12%, cyclosporine in 3%, and other agents in 6%.

Two hundred sixty-one infections occurred in 256 patients, an approximate average of one infection per patient. Forty-six percent of patients had no infections, 28% had one, 12% had two, and 14% had three or more episodes. The infections were bacterial in 46% of the instances, viral in 34%, fungal in 14%, and unknown in 6%. We could establish no correlation between the incidence of infection and rejection (ie, patients without infection did not have a lower incidence of rejection).

The other common complications were hypertension in 39% and a surprisingly high 25% incidence of seizures

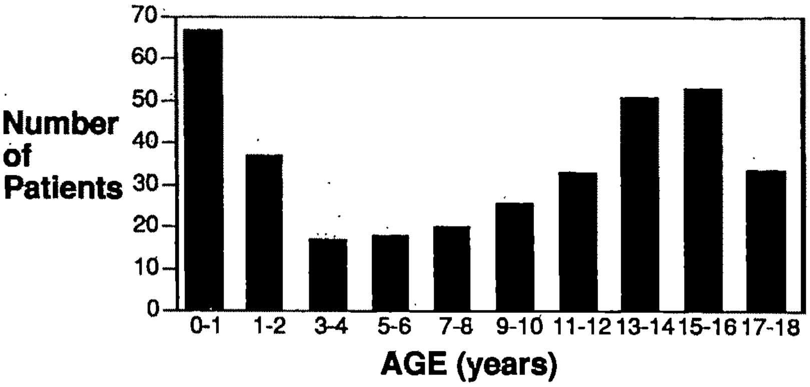

Fig 2. Age distribution of the 362 patients 18 years old or younger undergoing cardiac transplantation and reported to the current international survey.

Table 1. Types of Cardiomyopathy in 194 Patients

Type	No. of Patients[a]
Idiopathic	100 (52)
Viral	44 (23)
Familial	28 (14)
Hypertrophic	9 (5)
Adriamycin induced[b]	8 (4)
Other	5 (3)

[a] Numbers in parentheses are percentages. [b] Adriamycin is doxorubicin hydrochloride.

Table 3. Immunosuppression Regimens

	1985 (n = 95)		1990 (n = 218)	
Regimen	No. of Patients	%	No. of Patients	%
Cyclo/Pred/Aza	2	2	125	57
Cyclo/Pred	38	40	37	17
Cyclo/Aza	. . .	. . .	36	17
Cyclo	. . .	. . .	20	9
Aza/Pred/ATG	27	28	. . .	. . .
Cyclo/Pred/Aza/ATG	11	12	. . .	. . .

ATG = antithymocyte globulin; Aza = azathioprine; Cyclo = cyclosporine; Pred = prednisone.

(Table 5). The seizures were apparently related to cyclosporine therapy. The incidence of seizures in the various age groups was as follows: 0 to 1 month, 4%; older than 1 month to 12 months, 9%; older than 1 year to 4 years, 8%; 5 to 13 years, 22%; and 14 to 18 years, 30%. The reported incidence of coronary artery disease was only 8% and may be unrealistically low. Medical therapy other than that to treat rejection consisted of antiplatelet drugs in 41%, antihypertensives in 39%, diuretics in 34%, digoxin (Lanoxin; Burroughs Wellcome, Research Triangle Park, NC) in 8%, and antiarrhythmic drugs in 4%.

The overall length of follow-up ranged from 0 to 99 months. One hundred fifty-six patients were followed up for more than 1 year, 101 for more than 2 years, 60 for more than 3 years, 31 for more than 4 years, and 12 for more than 5 years. The functional classes were determined on the basis of a previously used scale [4] in which the best category comprised patients who were able to attend school and participate in sports or other physical activities. One hundred sixty-two, or 85% of the 190 patients for whom these data were available, were in this category. Fifteen patients (8%) attended school but were unable to participate in sports, 5 (3%) were unable to attend school, and 8 (4%) were bedridden. Thus, 85% of the patients are leading functionally normal lives.

Although one of the major objectives of the study was to determine the growth potential of these patients, the data submitted were inadequate to draw conclusions. There were some data regarding height and weight changes in 11 patients ranging in age from 6 months to 13 years at the time of transplantation. These data covered periods of 1 year, 2 years, or 3 years of follow-up. From these observations, it appeared that the majority of patients increased their weight percentile rather strikingly, although a few remained the same. Three of the 11 patients were in the fifth percentile for weight before transplantation and remained so at 2 and 3 years. Only 2 of the 11 showed a substantial increase in height percentile, and in 3 patients, the height percentile decreased. These very limited data suggest that weight would be more nearly normal, whereas height would be diminished in young recipients of cardiac transplants.

Table 2. Types of Congenital Heart Defects

Defect	1985 (n = 10)	1990 (n = 112)
Hypoplastic left heart	1	43
Multiple valve defects	1	15
DORV/tetralogy of Fallot	0	13
EFE	0	9
Complex VSD	1	8
Single ventricle	1	6
Complex TGA	1	4
Others or unknown	5	14

DORV = double-outlet right ventricle; EFE = endocardial fibroelastosis; TGA = transposition of the great arteries; VSD = ventricular septal defect.

Comment

Since the publication of our survey in 1985, several centers have reported their results with heart transplantation in various populations of patients ranging from newborn to 18 years old. However, most of the reports are heavily weighted toward certain age groups. The studies of Bailey and associates [5] and Mavroudis and Harrison [6] were composed largely of infants and neonates, whereas those of Addonizzio [8], Dunn [9], Starnes [10], Zuberbuhler [11], Braunlin [12], and their co-workers comprised predominantly children rather than infants. Recently, a comprehensive description of the entire pediatric cardiac transplantation experience was summarized by Haas and coauthors [13].

The excellence of these reports and the data tabulated in the Registry of the ISHT [7] raise the question of the need to carry out this current survey. However, although the ISHT Registry provides excellent tracking data in terms of numbers and mortality rates, it does not supply the more detailed information that would identify the trends we wish to describe. Therefore, we repeated this more detailed questionnaire in the hopes of performing a more in-depth analysis. An inherent weakness of this study is the fact that the data are only as complete as the responses we received. It is obvious that these 362 patients represent only half of the total patients in the ISHT Registry. Furthermore, the questionnaires from our survey were not always answered in the complete detail requested.

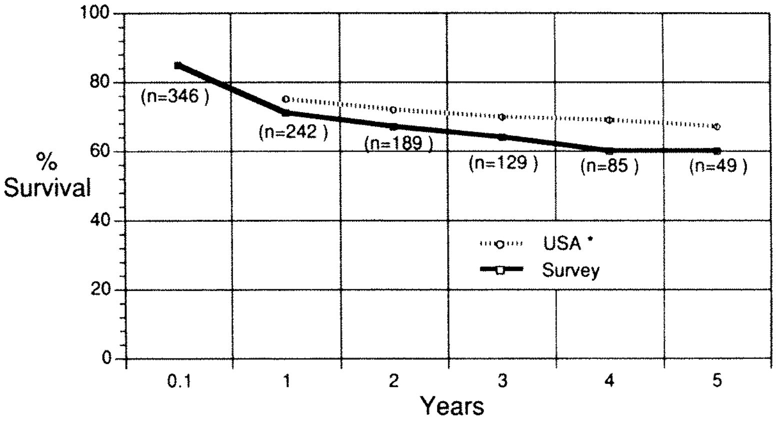

*Fig 3. Actuarial survival rates for patients in the current international survey and for the same age group (0 to 18 years) of patients in the United States. (n = number of patients in the survey; * = data from the International Society for Heart Transplantation Registry.)*

Therefore, the number of submitted answers varied considerably for the different questions asked.

In spite of these obvious limitations, it is apparent from this survey that several important trends are occurring. The actual number of cardiac transplantations in patients 18 years of age or younger continues to increase. A large part of this increase is attributable to the growing number of transplantations in infants and patients with complex congenital heart disease. At the time of our previous survey, both of these patient groups were considered very high risk, and therefore only a few procedures had been performed. The number of infants included in the surveys increased from 2 in 1985 to 88 in 1990, 68 of whom were 1 day to 1 month of age at operation. In the present survey, the entire group of patients 1 year of age or younger represented 19% of the total population. In 1985, only 16 (17%) of the 95 patients had transplantation for congenital heart defects, but in the current survey, 112, slightly more than one third, had congenital heart defects. The defects leading to transplantation were much more complicated that those reported in 1985. Although 38% of these patients had hypoplastic left heart syndrome, the application of transplantation to other complex defects has been promoted. In fact, almost a fourth of the patients in the current survey had undergone previous cardiac operations, usually directed toward the correction of congenital defects. Our survey reflects the trend in recent reports [14–17] of heart transplantation in patients with complex congenital heart defects and previous operations.

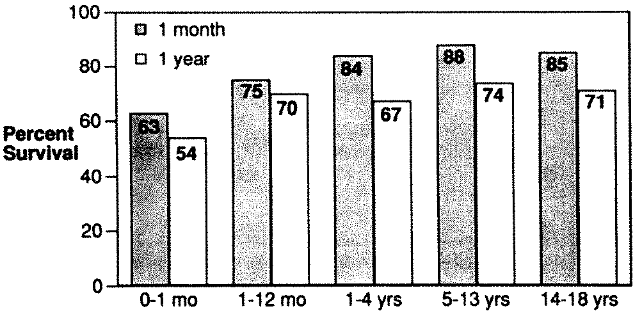

Fig 4. One-month and one-year survival rates by age group for the 362 patients in the current international survey.

Cardiomyopathy is still the most common indication for cardiac transplantation in children, but the etiology differs significantly from that of the adult population. Fifty percent of the cases were thought to be idiopathic and 23% viral, whereas ischemic cardiomyopathy was extremely rare in the pediatric population. In adults, coronary artery disease accounts for 41% of the patients undergoing cardiac transplantation [7]. A particularly interesting note from our study is that familial cardiomyopathy represented 14% of the cases, a rather disturbing figure considering the potential number of family members who may eventually become candidates for transplantation.

The sources of donor hearts in this study are similar to those used in the adult population; one third resulted from motor vehicle accidents, one fourth from closed head injury, and one fourth from cerebral hemorrhage or gunshot wounds. Birth trauma and sudden infant death syndrome accounted for only 7%. Although there may have been anencephalic donors, they were not specified in the responses. The average waiting period for patients in this survey was 26 days with a range of 1 day to 176 days. This average period is much shorter than that for

Table 4. Causes of Death

Cause	1985 (n = 36) No. of Patients	%	1990 (n = 125) No. of Patients	%
Ventricular failure	0	0	33	26
Infection	13	36	23	18
Rejection	10	28	39	31
Acute	. . .	. . .	24	19
Chronic	. . .	. . .	15	12
Dysrythmia	3	8	7	6
Malignancy	3	8	4	3
Pulmonary hypertension	3	8	0	0
Cerebrovascular accident	2	6	0	0
Renal failure	2	6	0	0
Other	0	0	11	9
Unknown	0	0	8	6

Table 5. Complications

Complication	% of Patients
Hypertension	39
Central nervous system (seizures)	25
Renal	17
Dermatological	15
Hepatic	12
Pulmonary	10
Coronary artery disease	8
Endocrine	5
Muscular/skeletal	4
Malignancy	3

Rates among 362 patients in 1990 International Survey.

the adult population, suggesting that donor hearts may be more available for the pediatric candidate.

The overall actuarial survival rates of 85% at 1 month, 70% at 1 year, 64% at 3 years, and 60% at 5 years are clearly superior to those reported in 1985, when the 3-month survival rate was only 68%. These improved results can probably be attributed to many factors including better patient selection, improved operative techniques, and better methods of diagnosing and treating episodes of rejection. The mortality in the different age groups was similar except for the group of patients less than 1 month of age, in whom the mortality rate was higher. The neonatal survival rate of this study cannot be compared with that of our previous survey, as too few infants were included in the previous survey.

However, the actuarial survival for the entire population of cardiac transplant patients reported in the ISHT Registry [7] for 1989 was 80% at 1 year, 75% at 3 years, and 72% at 5 years. The reasons for the lower survival rates in pediatric patients compared with the overall population are not entirely clear. The causes of death in this survey were predominantly ventricular failure or some form of rejection, acute or chronic. At the time of our previous survey, infection was the leading cause of death, but in this survey it accounted for only 18% of deaths. The reasons for this change in the etiology of death are not clear, but they may relate to the use of better immunosuppression protocols, which resulted in fewer fatal infections [12]. Indeed, the immunosuppression regimens from this survey are much more standardized than previously, with triple therapy the predominant one. As suggested from the ISHT Registry [7], the high rate of death due to ventricular failure is probably related to the higher incidence of elevated pulmonary vascular resistance in children who are transplant candidates. However, we could not make this determination from our survey.

Acute rejection was treated most often with intravenous steroids; a much smaller percentage of patients received antilymphocyte therapy. Although the methods of diagnosing rejection were not stated clearly enough to allow for precise determinations, it is known that a large number of the patients in the infant group did not undergo endomyocardial biopsy but were treated for rejection on the basis of clinical findings and other noninvasive means [5]. However, endomyocardial biopsy can be performed safely and effectively in children and infants. Recent techniques employ echocardiography and omit the need of irradiation-inducing fluoroscopy [18].

Although infections averaged one episode per patient, they did not account for as many deaths as in our previous survey. In fact, almost half the patients had no infections at all, a surprising figure considering the prevalence of infections in the general pediatric population. Almost half the infections that occurred were bacterial; viral and fungal infections accounted for most of the others.

Other complications were similar to those in the adult population with the exception of the high incidence of seizures, which occurred in 25% of the pediatric patients. Details of these seizures were not sufficient for critical analysis, but it is thought that the majority of them were due to cyclosporine therapy and that they were usually managed successfully by adjustment of the cyclosporine dose. The seizures were not related predominantly to the use of hypothermic circulatory arrest, as they occurred least often in infants. Thirty-nine percent of patients had systemic hypertension and required antihypertensive medication, a disturbingly high percentage in these young patients, but compatible with results from reports from individual centers [8]. Indeed, one center [11] reported systemic hypertension in all recipients. The low incidence of coronary artery disease (8%) may simply reflect the length of follow-up and, as time progresses, may increase strikingly. It is likely that the true incidence is higher than suggested by this survey. In individual centers, the incidence was 14% [11], 22% [10], and 29% [8]. The malignancy rate was only 3% which encourages us to believe that the immunosuppression therapy has not resulted in a notably high percentage of lymphatic neoplasia in this population.

Perhaps the most gratifying aspect of this survey is the recognition that 85% or 162 of 190 patients for whom we have this information were in the best functional class. They were able to attend school and even participate in sports and recreational activities. Only 4% were bedridden, and 3% could not attend school. These data are similar to those obtained in the previous survey. If attending school is considered the equivalent of returning to work in the adult population, these data suggest that rehabilitation of children after cardiac transplantation has been much more successful than in the adult population. However, many other factors determine an adult patient's employment and would not apply to a child attending school.

We were again disappointed to find that our survey did not reveal sufficient information to determine the effects of cardiac transplantation on growth potential in children. Sparse data suggest that these children are lower in height percentiles than in weight percentiles, but no firm conclusions could be drawn from this small database. Numerous questions remain concerning cardiac transplantation in children, questions we failed to answer in our first survey and cannot answer from the second. Will growth patterns be altered by a steroid-free regimen? Will coronary artery disease increase with time? Will the incidence of atherosclerosis in the children's age group be affected by a steroid-free regimen? Is there immune tolerance in neonates? Can donor availability be increased? Most of these questions will remain unanswered until more complete analyses can be performed.

In summary, we have conducted an international survey of the results of heart transplantation in children. It is clear from this survey that the number of transplantations has markedly increased since our last survey because of the increased number in infants and patients with complex congenital heart defects. Overall, survival rates in children have improved, but neonates still have the highest operative mortality. In spite of improvement, survival rates for patients older than 1 year still do not equal those for adults. The most common causes of death are ventricular failure and rejection rather than infection. Although rejection and infection are the most common long-term complications, other common problems are hypertension and seizures. A small but disturbing incidence of coronary artery disease has been identified. We are hopeful that future assessments will provide answers to the remaining questions. However, it is clear that cardiac transplantation in the pediatric population is quite worthwhile and is providing functional lives for many children.

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Pediatric Cardiac Transplantation Despite Atrial and Venous Return Anomalies

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Congenital anomalies of the atrium, pulmonary venous return, and systemic venous return are often regarded as anatomical contraindications to orthotopic cardiac transplantation. Among our pediatric transplant patients, 10 children aged 3 to 15 years, weighing 9 to 45 kg, and all previously operated on for a total of 18 interventions had 32 anomalies needing correction at the time of transplantation. Besides the 18 instances of great vessel abnormalities, 14 anomalies of the atrium and of the venous return were encountered either alone or in combination: single atrium or previous septectomy (4), hypoplastic left atrium (2), previous Mustard procedure (1), cor triatriatum (1), anomalous pulmonary venous return (3), and anomalous systemic venous return (3). The preparation of the donor heart was modified in four ways: right atrial paraseptal incision, left atrial flap technique, full-length mobilization of the pulmonary arteries, and aortic arch incision. Correction of the atrial and venous return anomalies was carried out at the time of orthotopic transplantation with the following techniques: atrial septation, atrial enlargement, superior systemic venous return reroofing, inferior systemic venous return reroofing, double venous rerouting (pulmonary and systemic), and septal realignment. One child died of pulmonary hypertension in the early postoperative period. After a follow-up ranging from 1 month to 52 months, all survivors are asymptomatic. Based on echocardiography, heart catheterization, and angiography, there are no stenoses and no shunts, and the atrial dimensions are good. Based on the results achieved with these surgical techniques, we conclude that most atrial lesions, anomalous pulmonary venous returns, and anomalous systemic venous returns are correctable at the time of orthotopic transplantation and do not preclude a successful outcome in children.

(Ann Thorac Surg 1991;52:716–21)

Heart transplantation in children has expanded during the past 5 years [1]. A major cause of this expansion is the increasing number of transplantations performed for congenital heart diseases. Within this group of patients, infant orthotopic transplantation accounts for most of the growth [2]. However, according to the 1990 report of the Registry of the International Society for Heart Transplantation [1], the number of transplantations performed for congenital diseases in other age groups has not changed significantly. One explanation for this plateau is that congenital anomalies of the atrium, pulmonary venous return, systemic venous return, and the great vessels are often regarded as anatomical contraindications to orthotopic transplantation.

My associates and I [3] have recently documented the feasibility of surgical correction of these congenital lesions at the time of orthotopic transplantation in our initial group of pediatric patients [3]. In this report, I will update our data on pediatric transplantation performed despite atrial and venous return anomalies.

Material and Methods

Among our pediatric transplant patients, 10 children with congenital heart diseases were selected for transplantation because of profound ventricular failure and prediction of early death. There were 2 girls and 8 boys. They ranged in age from 3 to 15 years and weighed between 9 and 45 kg. All patients had been operated on previously for a total of 18 corrective or palliative interventions.

This group of patients had 32 congenital anomalies requiring additional surgical procedures at the time of transplantation. Besides the 18 lesions of the great vessels, 14 anomalies of the atrium, pulmonary venous return, or systemic venous return were corrected at the time of orthotopic transplantation. The anomalies, encountered singly or in association with other lesions, were as follows: single atrium or previous septectomy (4 patients), a previous Mustard operation (1 patient), hypoplastic left atrium (2), an abortive type of cor triatriatum (1), anomalous pulmonary venous return (3), and anomalous systemic venous drainage (3).

Surgical Techniques

The general approach including timing of the operation, patient preparation, dissection, and native heart excision has been presented previously [3]. Before graft implantation, the identified anomalies were corrected according to

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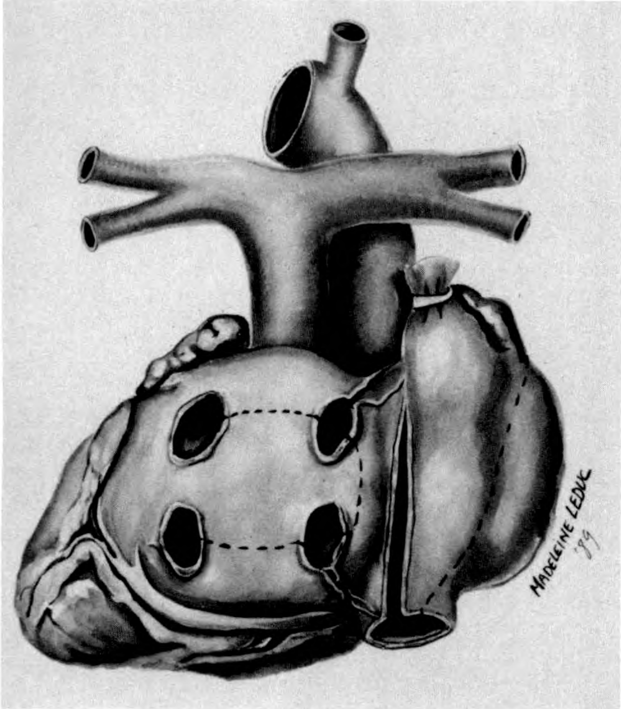

Fig 1. Donor heart preparation. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

techniques that will be described later. Methods of myocardial protection, ventricular venting, hemostasis, and heart rate control have been previously described [4–7].

Preparation of the Donor Heart

The donors were aged from 4 to 16 years (mean age, 9 years) and weighed 18 to 48 kg (mean weight, 30.2 kg). The grafts were distantly procured eight times and on site twice. To adapt the graft structures to the complex recipient anatomy or to enable correction of some of the recipient's anomalies, the usual donor heart preparation has to be modified (Fig 1). To facilitate the atrial–septal anastomosis when atrial septation or double venous rerooting needs to be done, instead of the classic Barnard incision, we do a straight paraseptal incision at the junction of the right atrial wall and the posterior interatrial septum starting at the orifice of the inferior vena cava and ending 1 cm below the junction of the superior vena cava and the atrium [3, 8]. This incision is simple, it obviates trauma to the sinus node, and it allows a single septal anastomosis; we now routinely make this incision in all orthotopic transplantations.

When the recipient left atrium is known to be hypoplastic or when there is some form of cor triatriatum that will necessitate atrial enlargement, a graft (donor) left atrial flap is fashioned by making an incision from the left to the right superior pulmonary vein, down to the right inferior pulmonary vein, and across to the left inferior pulmonary vein (see Fig 1).

When the recipient pulmonary artery or branches need to be reconstructed and when the great vessels are in D- or L-transposition or in malposition, the donor right and left pulmonary arteries are completely freed and divided beyond the pericardial reflection, and the aorta is divided at the brachiocephalic trunk.

Atrial Septation

A Mustard procedure had previously been performed in 1 patient. At transplantation, the baffle was totally resected, and a rectangular synthetic patch was used for septation. In 2 patients with single atrium and in 2 other children in whom total septectomy had previously been done, straight atrial septation was carried out using a Dacron patch or autogenous pericardium. In all patients, aided by the straight paraseptal incision, a single septal anastomosis was performed (Fig 2). Donor pericardium could have been used as well for septation. Postoperative angiography showed normal septal orientation and good left and right atrial sizes.

Atrial Enlargement

In a 5-year-old boy, the left atrium was somewhat small, and we encountered an unusual type of anomalous pul-

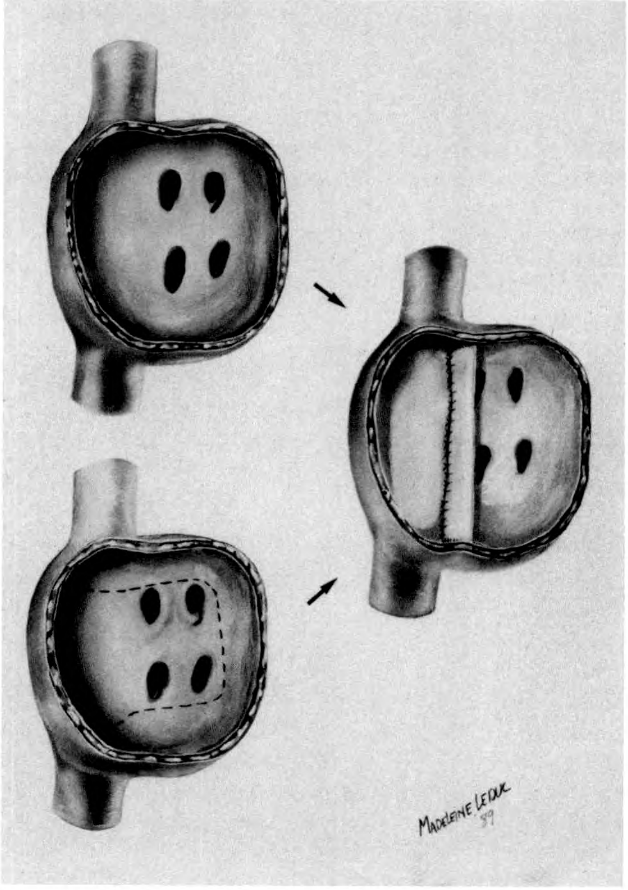

Fig 2. Atrial septation. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9: 608–17, by permission of Mosby-Year Book, Inc.)

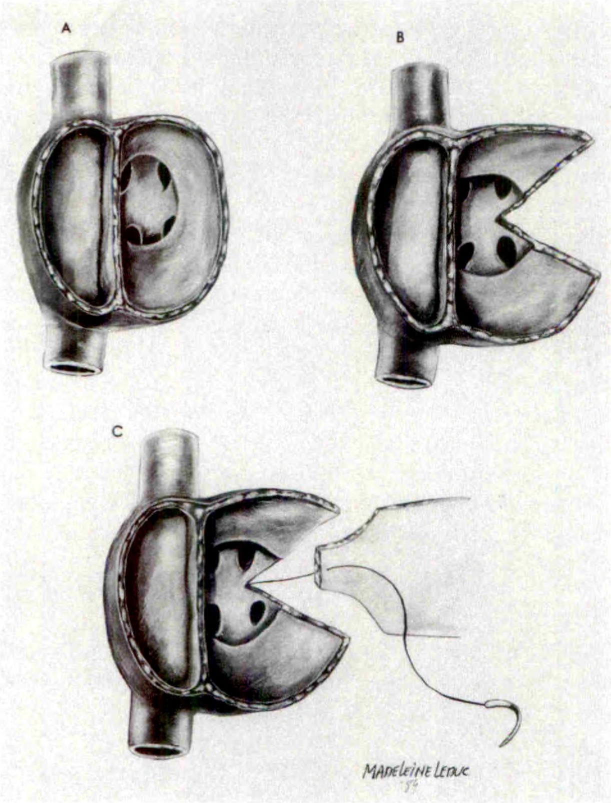

Fig 3. Atrial enlargement. (A) An unusual type of anomalous pulmonary venous return. (B) Vertical incision in left atrium is extended between pulmonary veins. (C) Anastomosis of donor and recipient atria. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

monary venous return called an abortive form of cor triatriatum. At the midpoint of the lateral wall, the left atrium including the anomalous ring was incised vertically, and the incision was extended between the left pulmonary veins (Fig 3). The tip of the donor atrial flap was brought into the incision between the left pulmonary veins, and the donor and recipient atria were anastomosed. In 2 other patients, enlargement of the hypoplastic left atrium was easily performed using the donor left atrial flap technique. Postoperative angiography showed that the reconstructed left atria had good dimensions (Fig 4).

Inferior and Superior Reroofing

When a left superior vena cava drains into the left atrium, two options are available. In 2 patients, an inferior septostomy was made, and a tunnel was created from the inlet of the left superior vena cava down to the septal defect and sutured to the left of the left pulmonary veins (Fig 5A). Synthetic material can be used, but we prefer autologous pericardium because of easier modeling. After transplantation, angiography demonstrated an adequate inferior reroofing in both patients (Fig 6).

In the third patient with superior vena cava–left atrial drainage, the other procedure, applicable solely during transplantation because the recipient ventricles have been excised, was performed. After a defect was made high in

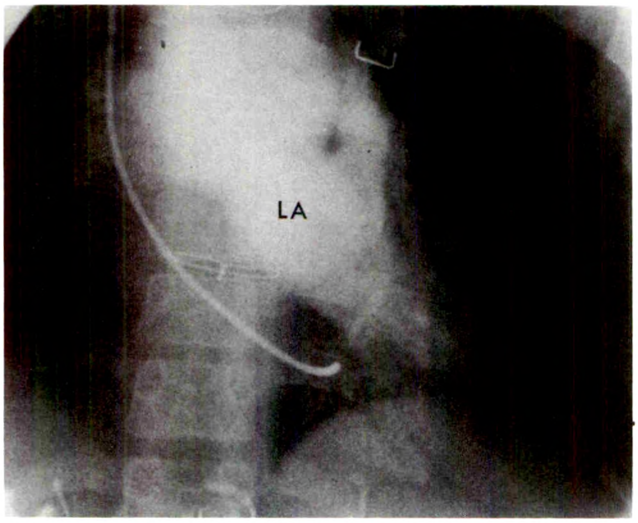

Fig 4. Postoperative angiogram showing result of left atrial enlargement. (LA = left atrium.) (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9: 608–17, by permission of Mosby-Year Book, Inc.)

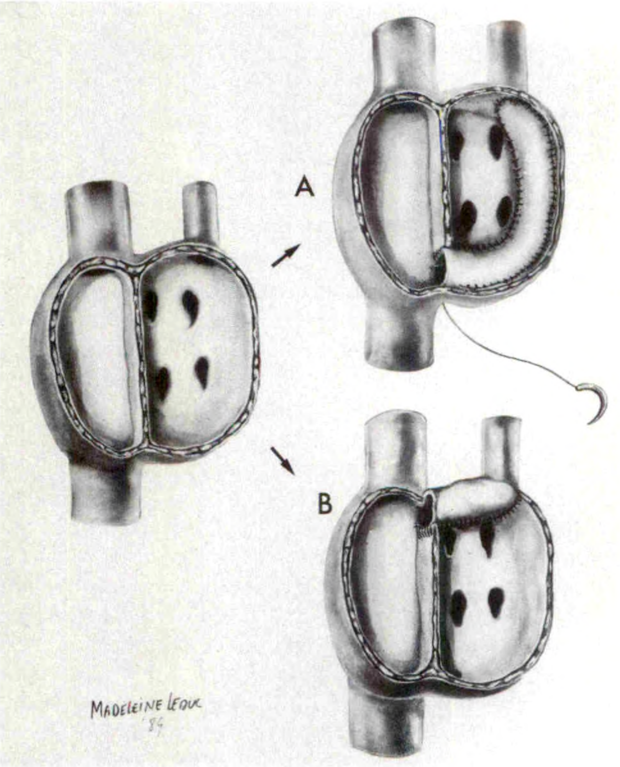

Fig 5. (A) Inferior and (B) superior reroofing. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

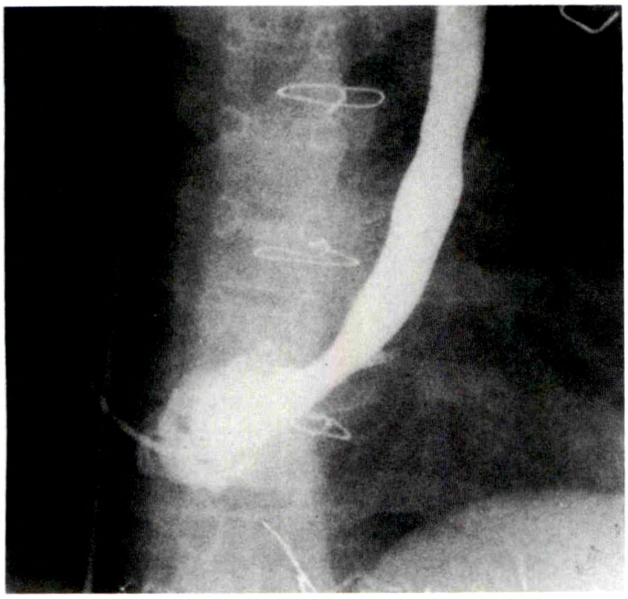

Fig 6. Postoperative angiogram showing adequate inferior reroofing. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

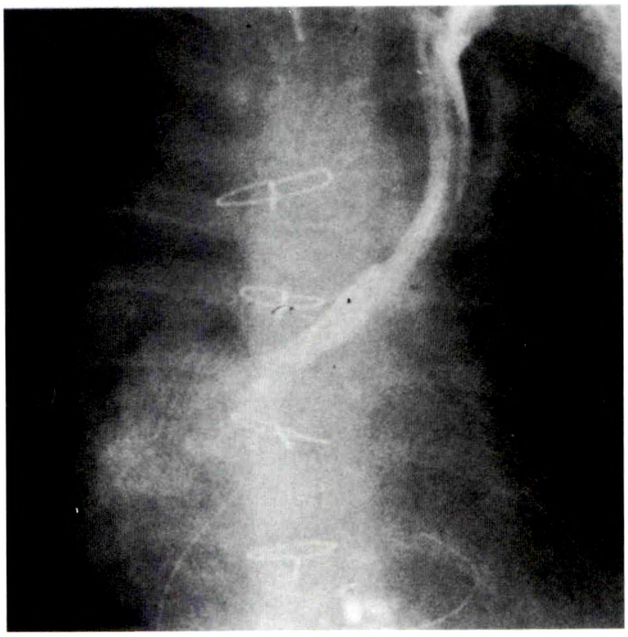

Fig 7. Postoperative angiogram showing adequate superior reroofing. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

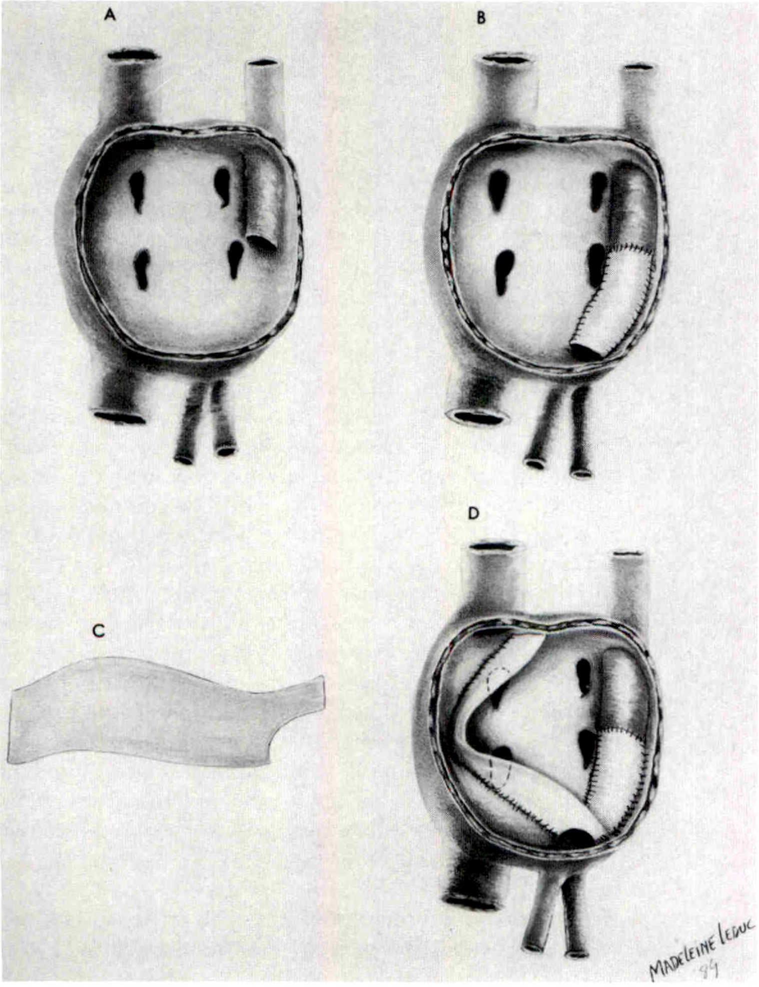

Fig 8. Double venous rerouting. (A) Single atrium and absent coronary sinus plus anomalous pulmonary and systemic venous return. (B) Inferior reroofing. (C) Synthetic patch tailored for double venous rerouting. (D) Completion of double venous rerouting. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

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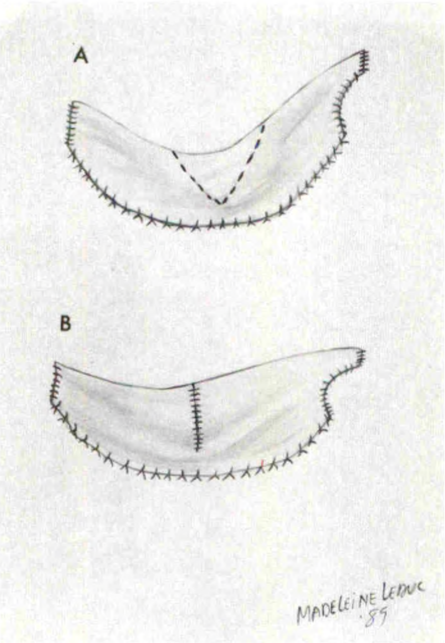

Fig 9. Septal realignment. (A) V-shaped incision is made in center of free edge of patch to allow straightening. (B) The two cut edges have been sutured together, and donor to recipient single septal anastomosis can be performed. (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)

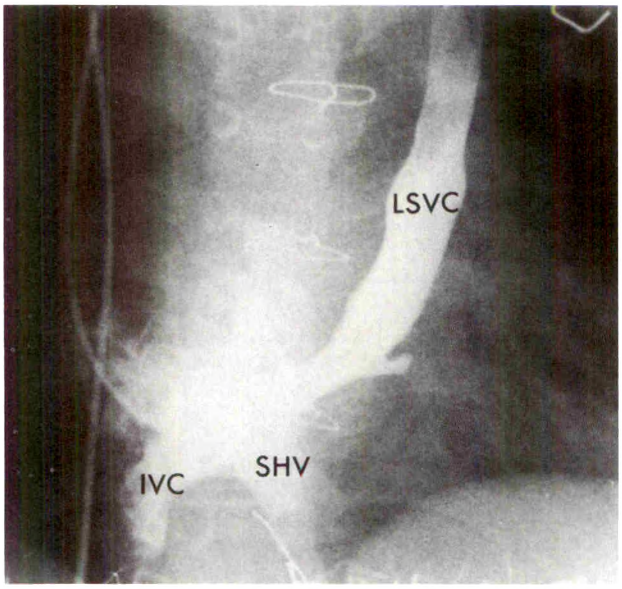

Fig 10. Angiogram made after double venous rerouting. (IVC = *inferior vena cava;* LSVC = *left superior vena cava;* SHV = *suprahepatic veins.) (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)*

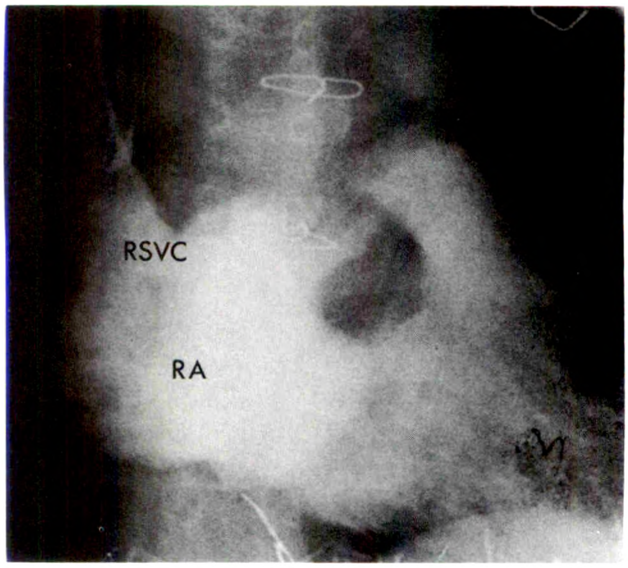

Fig 11. Angiogram made after double venous rerouting. (RA = *right atrium;* RSVC = *right superior vena cava.) (Reprinted from Chartrand C, Guerin R, Kangah M, Stanley P. Pediatric heart transplantation: surgical considerations for congenital heart diseases. J Heart Transplant 1990;9:608–17, by permission of Mosby-Year Book, Inc.)*

the interatrial septum, the cephalic wall of the large left atrium was rolled down and sutured inside the atrium up to the newly created atrial septal defect (Fig 5B). The upper part of the donor left atrium could then be sutured over that superior reroofing. The postoperative angiogram demonstrated patency of the superior reroofing (Fig 7).

Double Venous Rerouting

In 2 patients with single atrium and absent coronary sinus, we encountered a similar pattern of anomalous pulmonary and systemic venous return (Fig 8). As the left pulmonary veins were in their normal position, the right pulmonary veins drained very far to the right of the single atrium. A left superior vena cava, partially unroofed, drained to the left of the left pulmonary veins, and the suprahepatic veins emerged caudally and to the left of the single atrium. We elected first to perform an inferior reroofing, bringing the new left superior vena cava ostium confluent with the hepatic drainage (Fig 8B). Then a synthetic patch was tailored to permit the double venous rerouting (Fig 8C). As outlined in Figure 8D, the flat end of the patch was fixed to the inferior part of the atrial wall, and the inferior angle was anchored to the junction of the new superior vena cava ostium with the orifice of the suprahepatic veins. The patch was sutured over the inferior reroofing to avoid compression and then was anastomosed around the right border of the right pulmonary veins and up to the left of the right superior vena cava inflow. This double rerouting technique permitted reorientation of the systemic and pulmonary venous returns to the appropriate atria.

Septal Realignment

With these septation procedures, the free edge of the patch can become so curved that anastomosis with the donor atrial septum is impossible. In this case, we make a V-shaped excision in the center of the patch and suture the two cut edges together (Fig 9). With this maneuver, septal realignment is achieved, and donor to recipient single septal anastomosis is easily performed. In both patients with double venous rerouting and septal realignment, the postoperative angiograms show that the venous returns from the left superior vena cava, suprahepatic veins, and inferior vena cava are reoriented to the right atrium (Fig 10) and that the right superior vena cava drains into a good-sized right atrium (Fig 11).

Results

One child died in the early postoperative period because of irreversible right heart failure secondary to underestimated preoperative pulmonary hypertension. All survivors made an uneventful recovery and were discharged from the hospital in very good clinical condition. Follow-up ranges from 1 month to 52 months. All survivors are asymptomatic and have been able to resume normal activities after the third postoperative month. The posttransplantation evaluation includes serial echocardiography and right heart catheterization in all patients and left heart catheterization and angiography in all but 2. Based on these studies, there is no evidence of venous, atrial, or great vessel stenosis. No residual shunt has been detected, the atrial cavities are of good size, and there is good orientation of the great vessels.

Comment

Based on our results, we conclude that, using the surgical techniques described here, most atrial, pulmonary venous return, and systemic venous return anomalies are correctable at the time of orthotopic transplantation. They do not preclude a successful outcome in children.

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Expanding Applicability of Transplantation After Multiple Prior Palliative Procedures

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The number of heart transplantations performed over the past 3 years has plateaued. However, the number of pediatric transplantations continues to slowly increase. Unlike adult heart transplantation, for which cardiomyopathy remains the most frequent indication, structural congenital heart disease is the primary indication in children. This report reviews our experience with orthotopic heart transplantation in the presence of structural congenital heart disease with and without prior palliative repair. The diagnoses included transposition of the great arteries, common atrium, left superior vena cava with and without a bridging innominate vein, dextrocardia, and univentricular configurations. The palliative repairs included Blalock-Taussig shunt, bilateral Glenn shunt, Fontan repair, and Mustard and Rastelli procedure. There were no early deaths. Two rejection-related late deaths have occurred at 8 months and at 3 years postoperatively. Extended use of donor tissue and modifications to surgical technique allowed for successful orthotopic heart transplantation in these patients who had structural congenital heart disease with and without prior surgical palliation.

(Ann Thorac Surg 1991;52:722–6)

In 1989, 2,437 heart transplantations were performed worldwide [1]. The overall number of transplantations has plateaued over the past 3 years, but the number of heart transplantations in the pediatric age group (0 to 18 years) is increasing. Since 1984 when approximately 50 transplantations were done in this age group worldwide, there has been a gradual increase to more than 200. Although acquired heart disease continues to be the most frequent indication for heart transplantation in adults, congenital heart disease has become the primary indication for heart transplantation in children. Much of this is due to the dramatic increase in newborns having transplantation for hypoplastic left heart syndrome. There are some congenital heart defects where the morphological right ventricle serves as the systemic ventricle [2]. It has been suggested that the right ventricle cannot sustain systemic performance over the long term, which may lead to end-stage heart failure [3], resulting in referral for consideration of transplantation. As many as 10% to 20% of patients now alive with anatomical congenital heart lesions may undergo transplantation [4].

This report describes our experience with orthotopic heart transplantation for structural congenital heart disease with and without prior palliation. The group of patients with congenital heart defects who underwent subsequent orthotopic heart transplantation underscores the extended use of donor tissue to create a normal anatomical configuration that will make orthotopic transplantation possible.

Material and Methods

Patient Population

To date, our experience includes 9 patients with congenital heart anomalies who have had orthotopic heart transplantation. Three patients, 2 girls 8 and 11 years old and a 6-year-old boy, had had prior palliative procedures.

Several different anatomical defects were present in the 9 patients. Of particular importance were defects of systemic venous drainage, abnormalities of the atria, and abnormalities of the great vessels. Similarly, palliative repairs affecting systemic venous drainage, the great arteries, or the atria were relevant and required modifications of the standard transplantation technique to accommodate the anatomical variations in the structural defects. The anatomical variations encountered included transposition of the great arteries in 3 patients, a common atrium or large atrial septal defect in 3 patients, a left superior vena cava with and without a bridging innominate vein in 2 patients, a Blalock-Taussig shunt in 1 patient, bilateral Glenn shunts in 1 patient, and a Fontan repair as well as a Mustard repair and a Rastelli repair in 1 patient.

Anatomical Conditions and Operative Techniques

ABNORMALITIES OF SYSTEMIC VENOUS DRAINAGE. When there was a persistent left superior vena cava with a bridging innominate vein, the confluence of the vein and atrium was simply oversewn once the existence and patency of the bridging vein had been assured (Fig 1).

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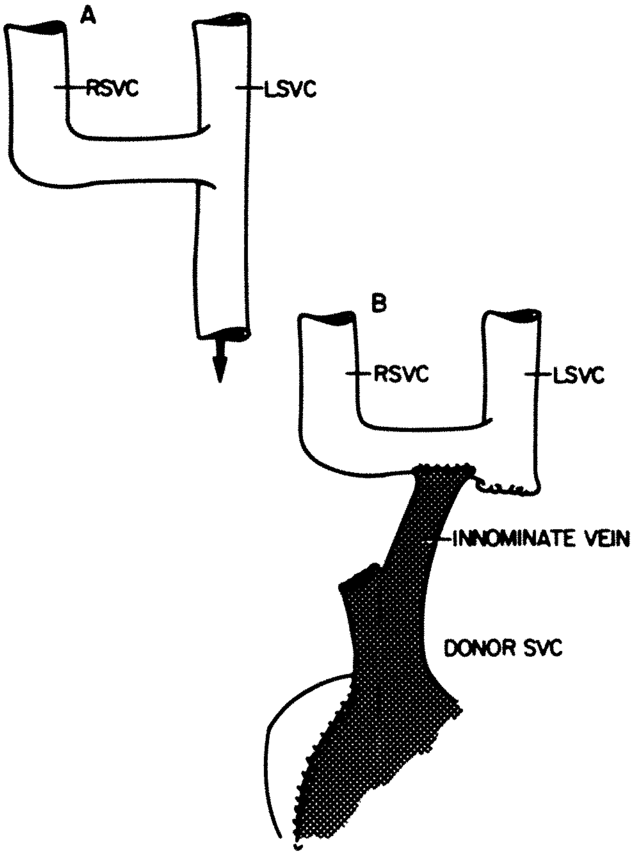


Fig 1. Repair of persistent left superior vena cava (LSVC) with innominate vein by donor end-to-side anastomosis on recipient innominate vein. (RSVC = *right superior vena cava.) (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602–7.)*

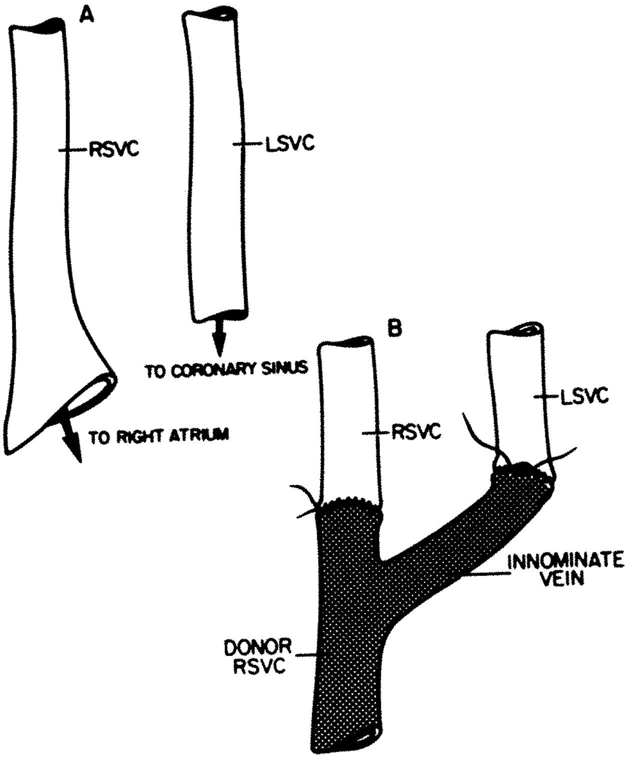


Fig 2. Repair of persistent left superior vena cava (LSVC) without bridging innominate vein using direct end-to-end anastomosis. (RSVC = *right superior vena cava.) (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602–7.)*

When there was no bridging innominate vein, the donor innominate vein was anastomosed directly to the left superior vena cava (Fig 2) [5]. One patient also had an interrupted inferior vena cava with azygos continuation. This was managed by placing the inferior venous cannula in the largest of the veins entering the atrium and using the cardiotomy suction intermittently for the rest of the hepatic drainage. These vessels were included within the right atrium during the creation of the interatrial septum.

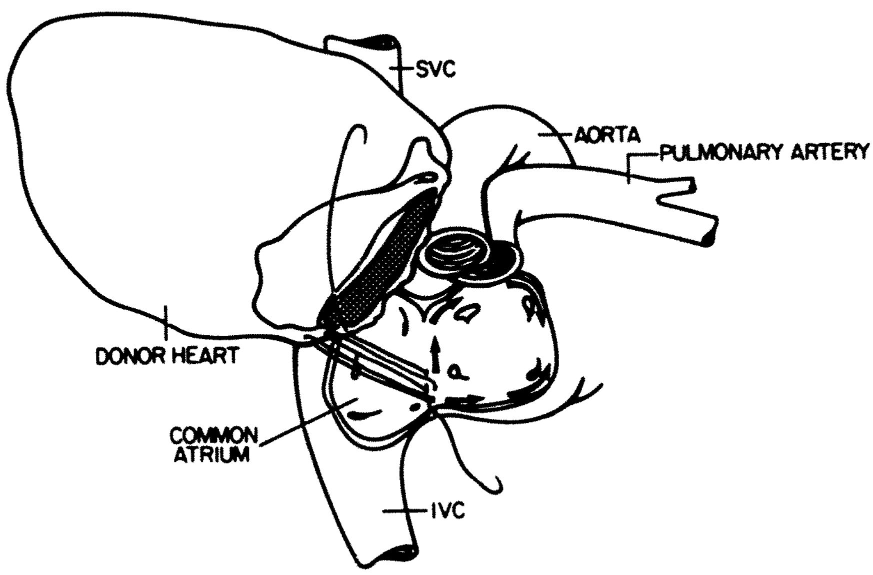
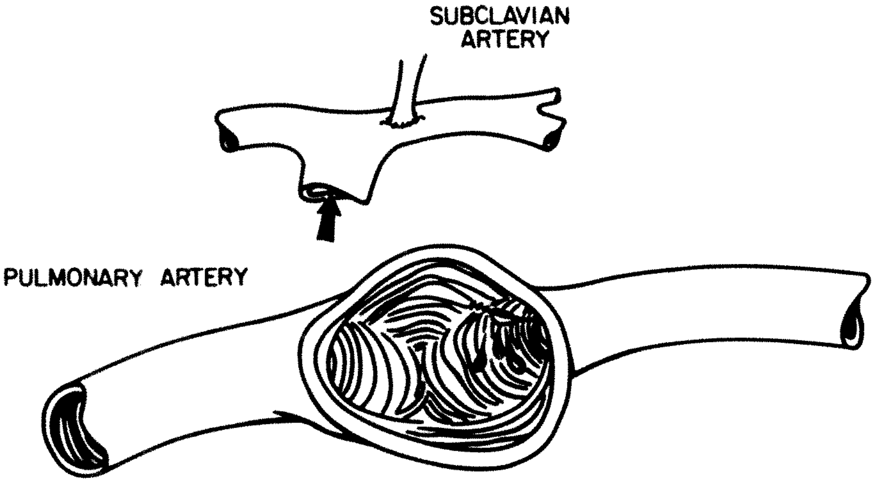


Fig 3. Orthotopic transplantation when common atrium or large atrial septal defect is present. (IVC = *inferior vena cava;* SVC = *superior vena cava.) (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602–7.)*

Fig 4. Closure of Blalock-Taussig shunt from within the pulmonary artery. (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602-7.)



COMMON ATRIUM. In patients with a common atrium or very large atrial septal defect or in several patients with a fenestrated fossa ovalis, which was excised, the donor left atrial wall was minimally trimmed, and this excess tissue was sewn directly to the posterior wall of the common atrium, thereby creating an interatrial septum (Fig 3). The anastomosis was commenced at the most inferior portion of the posterior atrial wall corresponding to where the interatrial septum should be and proceeded superiorly from the left atrial side to the left superior pulmonary vein where the suture was brought out through the recipient atrial wall. The other end of the suture was used to complete the inferior portion of the left atrial anastomosis. The free wall was left untied, and a left atrial vent was placed through the suture line. The vent was removed after the aorta had been unclamped and effective ventricular function returned [5].

LEFT BLALOCK-TAUSSIG SHUNT. The left Blalock-Taussig shunt was oversewn from within the left main pulmonary artery after excision of the native heart (Fig 4). The orifice could be clearly seen within the stump of the pulmonary artery. Visualization was aided by a short period of circulatory arrest [5].

GLENN SHUNTS. The patient with bilateral Glenn shunts was placed on cardiopulmonary bypass through cannulation into the ascending aorta in the usual fashion and a single venous cannula in the pulmonary artery. The left and right pulmonary arteries were dissected to expose the Glenn shunts bilaterally. Cardiopulmonary bypass was then instituted. Excision of the recipient heart was carried out under deep hypothermia and circulatory arrest. The anastomoses of the Glenn shunts to the pulmonary arteries were cut, and the pulmonary arteries were repaired bilaterally (Fig 5). The donor innominate vein was anastomosed end-to-end to the left superior vena cava, and the donor right superior vena cava was anastomosed end-to-end to the recipient right superior vena cava. The donor and recipient inferior venae cavae were anastomosed end to end [5].

FONTAN REPAIR. The same patient who had the bilateral Glenn shunts underwent a Fontan repair. This was characterized by a pericardial conduit extending from the inferior vena cava through the common atrium to the pulmonary artery with end-to-end anastomoses on the

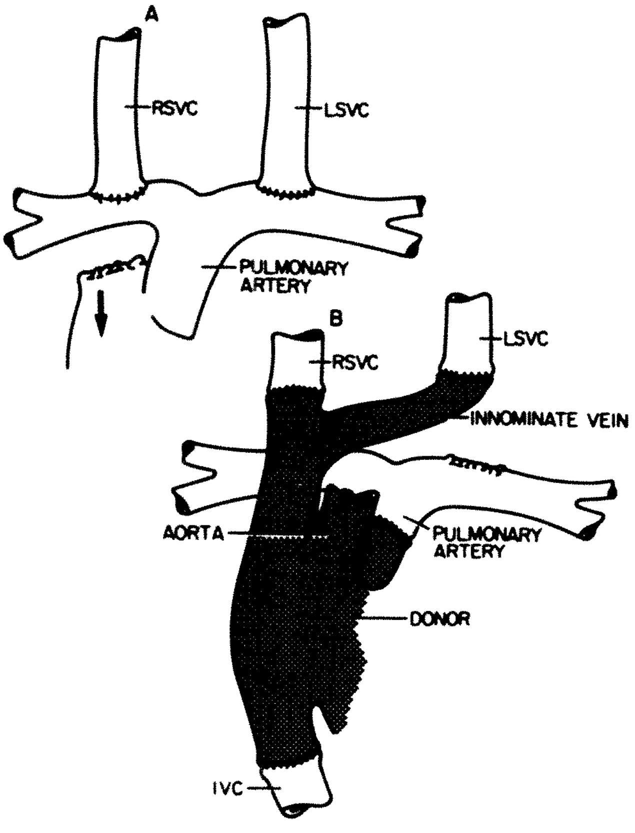


Fig 5. Bilateral Glenn shunts repair of pulmonary artery and restoration of normal venous anatomy. (LSVC = left superior vena cava; RSVC = right superior vena cava.) (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602-7.)

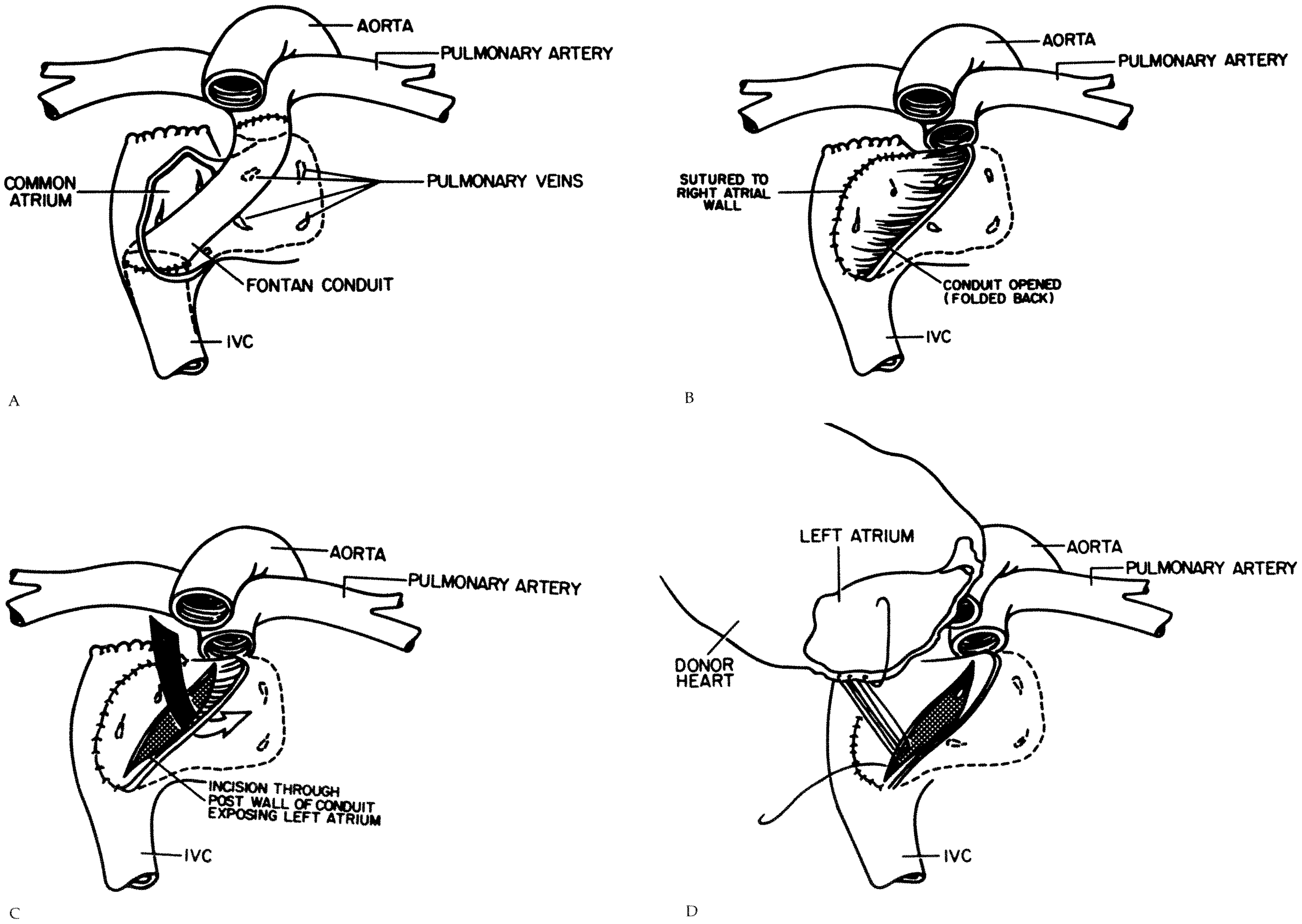


Fig 6. Orthotopic transplantation after Fontan repair: (A) appearance after excision of native ventricle; (B) creation of left atrial chamber; (C) Opening of left atrial chamber for anastomosis; and (D) technique for left atrial anastomosis. (IVC = *inferior vena cava;* POST = *posterior.) (Reprinted from Menkis AH, McKenzie FN, Novick RJ, et al. Special considerations for heart transplantation in congenital heart disease. J Heart Transplant 1990;9:602–7.)*

inferior vena cava and pulmonary artery. After the recipient ventricles had been carefully excised, the conduit came into view (Fig 6A). The anterior wall of the tube was incised and opened after the pulmonary artery had been disconnected. The opened walls of the conduit were then sutured to the free atrial wall, thereby creating a closed left atrial chamber (Fig 6B). The posterior wall of the conduit was then opened, creating an orifice in the newly created left atrium (Fig 6C). To this orifice, the donor left atrium was anastomosed in the usual fashion (Fig 6D). The systemic venous anastomoses were then fashioned as already described, followed by the aortic and pulmonary artery anastomoses (see Fig 5) [5].

DEXTROPOSITION OR DEXTROCARDIA. Two patients had dextropositioned hearts and 1 patient, the little girl with the Fontan repair, dextrocardia. Although the transplanted hearts in all 3 tended to assume a midline or right-sided position, there was no embarrassment of venous filling and no cardiac dysfunction attributable to obstruction to venous inflow.

MUSTARD REPAIR. Since the presentation of our paper, another child with prior palliative repair has undergone transplantation. The patient was a 6-year-old boy with a diagnosis of complex congenital heart disease characterized by levotransposition and pulmonary atresia. When he was 5 days of age, a right-sided Blalock-Taussig shunt was performed. Two years ago, Mustard and Rastelli-type repairs were undertaken. Pericardium was used for the Mustard repair, and a 19-mm cryopreserved pulmonary homograft was used for the pulmonary artery conduit. Venous cannulas were placed in the superior and inferior venae cavae and the aortic cannula was inserted through the ascending aorta. After excision of the native heart, the pulmonary conduit was excised to the level of the pulmonary bifurcation because of moderate calcification of the conduit. The Mustard baffle was identified. Both caval orifices were of adequate size and were not encroached on by the baffle. A portion of the baffle could have been retained to aid in reconstruction of the interatrial septum; however, the entire baffle was removed and the interatrial septum reconstructed as in the technique for common atrium. It was necessary to use the full length of donor pulmonary artery including the bifurcation, which was opened and accommodated the much larger recipient pulmonary bifurcation.

Results

Orthotopic transplantation was possible in all patients. There were no early deaths (<90 days). There have been two late deaths, one at 8 months and one at 3 years. Both were rejection related. All other patients were in functional class I.

Comment

Heart transplantation remains the only viable option for children with end-stage myocardial failure. The presence of structural congenital heart disease or prior palliative surgical intervention necessitates modifications to orthotopic transplantation techniques. In addition to the techniques described in this report, procedures for successful orthotopic transplantation have been described for transposition of the great arteries [6, 7], atrial situs inversus [8], Senning repair [9], and hypoplastic left heart syndrome [10]. The uniform feature in each of these reports is the retention of sufficient contiguous donor tissue to accommodate the technical modifications required. Specifically, excess aorta, pulmonary artery, venae cavae, and atrial wall are retained in continuity with the donor heart at the time of harvest.

Orthotopic heart transplantation is feasible for any patient with end-stage myocardial failure associated with structural congenital heart disease or prior palliative surgical repair. Although the results of pediatric heart transplantation are not as good as those obtained for adult transplantation, we believe that this is a viable therapy that usually results in a good quality of life for the children.

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EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Twentieth World Congress of The International Society for Cardiovascular Surgery, Amsterdam, the Netherlands—September 1–6, 1991

For information on this meeting, contact the Registrar: XX World Congress—ISCVS, International Travel Service, Inc, 104 Wilmot Rd, Suite 300, PO Box 825, Deerfield, IL 60015; or telephone (708) 940-2100 (facsimile: (708) 940-2386); or contact the Congress Secretariat, International Society for Cardiovascular Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Current Issues in Thoracic Organ Transplantation, Essen, Germany—September 19–21, 1991

For information on this meeting, contact Organizing Secretariat, Gisa Brodde Meetings, Saalbau Essen, PO Box 100301, D-4300 Essen 1, Germany; or telephone (0)201-232121 (facsimile: (0)201-232273).

Heart Valve Replacement—Past Performance: Future Prospects, Sheffield, United Kingdom—September 19–21, 1991

For information on this meeting, contact Jenny Pupius, Symposium Secretariat, Department of Medical Physics and Clinical Engineering, Royal Hallamshire Hospital, Glossop Rd, Sheffield S10 2JF, United Kingdom; or telephone (0742) 555250 (facsimile: (0742) 729981).

Fifth Annual Meeting, European Association for Cardio-Thoracic Surgery, London, United Kingdom—September 22–25, 1991

For information on this meeting, contact Organising Secretariat, Conference Associates & Services, Ltd, Congress House, 55 New Cavendish St, London W1M 7RE United Kingdom; or telephone 071 486 0531 (facsimile: 071 935 7559; telex: 934 346 CONFAS G).

Fifth Annual Meeting of the Mediterranean Association of Cardiology and Cardiac Surgery, Alexandria, Egypt—September 23–27, 1991

For information on this meeting, contact Prof M. El-Fiky, Chairman, Cardiac Surgery Department, Ain Shams Faculty of Medicine, Abbassia, Cairo, Egypt; or telephone (202) 834981 or (202) 835495 (facsimile: (202) 834981).

Arterial Grafts for Coronary Bypass Surgery, Brussels, Belgium—September 29–October 2, 1991

For information on this workshop, contact R. Dion, MD, Cliniques Universitaires St. Luc, Chirurgie Cardiaque, ave Hippocrate, 10, B 1200 Brussels, Belgium; or telephone 32/2/764.61.06 or 764.61.07 (facsimile: 32/2/764.37.03).

Arrhythmia Surgery Operative Clinic, St. Louis, Missouri—October 3–5, 1991

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Mitral Surgery Advances and Controversies, Varese, Italy—October 4–5, 1991

For information on this meeting, contact Erreci Congressi sri, Via Negroni, 4/7, 28100 Novara, Italy; or telephone 0321-393021 or 398977 (facsimile: 0321-393026).

American College of Surgeons, Chicago, Illinois—October 20–25, 1991

For information on this meeting, contact American College of Surgeons, 55 Erie St, Chicago, IL 60611; or telephone (312) 664-4050.

Fifty-seventh Annual Scientific Assembly, American College of Chest Physicians, San Francisco, California—November 4–8, 1991

For information on this meeting, contact the American College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068; or telephone (708) 698-2200.

Thirty-eighth Annual Meeting of the Southern Thoracic Surgical Association, Orlando, Florida—November 7–9, 1991

For information on this meeting, contact Hendrick B. Barner, MD, Southern Thoracic Surgical Association, 401 N Michigan Ave, Chicago, IL 60611-4267; or telephone (312) 644-6610 (facsimile: (312) 321-6869).

Third Combined Meeting of the Nederlandse Vereniging Voor Thoraxchirurgie and the Deutsche Gesellschaft für Thorax-, Herz- und Gefäßchirurgie, Maastricht, the Netherlands—November 7–9, 1991

For information on this meeting, contact Organizing Secretariat, Conference Agency Limburg, Mrs Brigitte Bessems, c/o University Hospital Maastricht, PO Box 1918, 6201 BX Maastricht, the Netherlands; or telephone 31 (0)43 258344 (facsimile: 31 (0)43 258355).

■ Sixth World Conference on Lung Cancer, Melbourne, Australia—November 10–14, 1991

For information on this meeting, contact Ms Jane Willis, MCS Convention Services, PO Box 335, Heidelberg, Vic 3079, Australia; or telephone (613) 499-6722 (facsimile: (613) 499-7137).

■ Sixty-fourth Scientific Session of the American Heart Association, Anaheim, California—November 11–14, 1991

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 750-5300.

■ Circulatory Support 1991, San Francisco, California—November 15–17, 1991

For information on this meeting, contact The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

■ Twenty-eighth Annual Meeting of The Society of Thoracic Surgeons, Orlando, Florida—February 3–5, 1992

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

■ Forty-first Annual Meeting of the American College of Cardiology, Dallas, Texas—April 12–16, 1992

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

■ Seventy-second Annual Meeting of the American Association for Thoracic Surgery, Los Angeles, California—April 27–29, 1992

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Chicago, Illinois—June 8–10, 1992

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Eighteenth Annual Meeting of The Western Thoracic Surgical Association, Kauai, Hawaii—June 25–27, 1992

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

CLASSIFIED ADVERTISEMENTS

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SITUATIONS AVAILABLE

Cardiovascular surgeon, board certified/eligible, wanted to join two surgeons in an established cardiovascular program in the southeastern USA. Must have experience in adult cardiac, thoracic, and vascular surgical procedures. Salary/full partnership negotiable. Please send resume.

Please respond to Box 398. 398C/I

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Possible academic affiliation. Please respond with CV.

Please respond to Box 404. 404I/C

Cardiovascular and thoracic surgeon, BC/BE, recently trained, to join an established 3-man private practice group in Southeastern coastal capital city and two-university town. Great hunting, fishing, and sports. Submit CV and references.

Please respond to Box 519. 519D/I

Thoracic surgeon wanted: a busy general thoracic surgical practice (non–open-heart, nonvascular) 20 miles south of Boston seeks to add a third surgeon. Candidate must be board certified or eligible. This active practice operates from a multihospital referral base. The community is an ideal area for family living, with excellent schools and nearby Boston and Cape Cod cultural and recreational facilities. Competitive salary, malpractice insurance, and excellent benefits package. Please respond with CV.

Please respond to Box 531. 531E/J

Cardiothoracic surgeon needed for rapidly expanding program located in a coastal area of the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Respond with curriculum vitae and names of references.

Please respond to Box 533. 533E/J

Associate director for heart transplantation is urgently needed for rapidly expanding heart transplantation program in California. Excellent benefits package. Please respond with curriculum vitae and outline of surgical experience.

Please respond to Box 534. 534E/J

Cardiovascular and thoracic surgeon: excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join a large cardiac surgery group in southern California. Must have a minimum of 1 year experience in pediatric and neonate cardiac surgery (including fellowship). Excellent fringe benefits. Please reply with curriculum vitae and outline of surgical experience.

Please respond to Box 535. 535E/J

Cardiovascular and thoracic surgeon experienced in adult and pediatric cardiac surgery as well as general thoracic surgery needed to join the practice of an established cardiovascular and thoracic surgeon. Send curriculum vitae and details of experience.

Please respond to Box 541. 541F/K

Cardiothoracic and vascular surgeon, board eligible or certified, to join 2-man private practice in Ft. Lauderdale, Florida. Send CV.

Please respond to Box 542. 542F/K

Cardiothoracic surgeon, BC/BE, to join rapidly expanding single-hospital practice in Chicago area. Practice is 90% adult cardiac, 10% vascular with potential for general thoracic procedures. Generous salary leading to partnership. Send curriculum vitae.

Please respond to Box 545. 545F/K

Cardiothoracic/vascular surgeon: board certified in general and thoracic surgery. Minimum of 2 years of practice experience. Very active private multispecialty practice. Located in western Pennsylvania. Salary leading to full partnership. Excellent benefits. Detailed CV must accompany response. All replies treated confidentially.

Please respond to Box 546. 546F/K

Assistant surgeon sought for busy adult cardiac surgery practice. Responsibility to assist senior surgeons in the OR and management of ward patients. BC/BE in general surgery is necessary, some vascular experience and/or thoracic surgery is desirable. Must be licensed in California or able to obtain license. Generous salary and fringes. Position is available immediately and could be permanent. Ideal location in very pleasant small city near major metropolitan centers.

Please respond with CV to Michael N. Wood, MD, 1060 E Foothill Blvd, #201, Upland, CA 91786. 553G/I

Cardiovascular and thoracic surgeon, BE/BC, wanted to join an established expanding private practice in the Northeast with an academic affiliation. Must have experience in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership.

Please respond to Box 554. 554G/L

Cardiovascular surgeon, board certified or board eligible in thoracic surgery. Large surgical practice in western Pennsylvania. Excellent compensation and benefits. Please send CV and references.

Please respond to Box 555. 555G/L

Assistant surgeon: cardiac, thoracic, or general surgeon with cardiac experience; to join well-established three-man group in San Diego. Group does about 400 cardiac cases and the same number of thoracic/vascular cases per year. Position is for surgical assistance, ideal for physician who wants to live in a beautiful location and have low-stress practice. Very generous salary/benefits.

Respond with CV and three letters of reference to Kenneth D. Stahl, MD, Cardiovascular and Thoracic Surgeons, 6719 Alvarado Rd, Suite 303, San Diego, CA 92120. 556G/I

Residency in cardiovascular and thoracic surgery: opening available in July 1992 for three-year training program in busy adult (600) and pediatric (150) cardiac surgery program including arrhythmia surgery, heart and heart/lung transplantation, and vascular and thoracic surgery. Outstanding facilities and fully accredited program at a major Canadian university. ABS certification or eligibility required.

Please reply to Patricia A. Penkoske, MD, Department of Surgery, Room 2D2.09 Walter MacKenzie Center, University of Alberta, Edmonton, Alberta T6G 2R7, Canada; telephone: (403) 492-8057. 558G/J

University of Arizona. Expanding university program creates exciting opportunities for new faculty, both academic (tenure) and clinical tracks. Applicants must be interested in academic career. Interested particularly in cardiac, obstetric, ICU, and pediatric subspecialties. An AA/EOE.

Contact Burnell R. Brown, Jr, MD, PhD, Department of Anesthesiology, Arizona Health Sciences Center, Tucson, AZ 85724. Closing date: 03/31/92 or until position is filled. 560G/L

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join 2-man surgical group in the Southeast. 1–2 years of experience preferred. Please respond with curriculum vitae.

Please respond to Box 562. 562G/L

Cardiothoracic surgeon, recent graduate or no more than 2 years out of training, to join established group in a major Texas city. Practice includes adult and some pediatric cardiac, electrophysiologic, thoracic, and peripheral vascular surgery. Please respond with CV.

Please respond to Box 564. 564G/L

Adjunct staff surgeon—the Department of Surgery of the Deborah Health and Lung Center invites applications for the position of adjunct staff surgeon for the Division of Cardiac Surgery. This opportunity is appropriate for a surgeon having completed his/her cardiac surgery training and desiring further operative experience in the setting of a busy and varied clinical practice.

Please send curriculum vitae to Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart and Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015. 567G/J

BC/BE adult cardiothoracic surgeon wanted to join established private practice in Long Island, New York. Please send CV and references.

Please respond to Box 569. 569H/J

BC/BE cardiovascular and thoracic surgeon wanted to join busy two-member group in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308 [(305) 561-9781 after 9:00 PM]. 570H/A

Cardiac surgeon needed immediately for position on East Coast. Prefer 1–3 years' experience in all aspects of adult cardiac surgery. Salary with early partnership. Reply with curriculum vitae.

Please respond to Box 572. 572H/J

Cardiovascular and thoracic surgeon, BE/BC, to join busy, young CV surgeon with growing practice on Lake Michigan, 90 miles from Chicago. 200 pump cases/year. Strength in vascular surgery desired with large vascular practice available and director of medical center laboratory. Excellent salary and lifestyle, full benefits, room to grow, and early partnership.

Send CV to Meredith Schmidt, RN, Physician Services Representative, Mercy Memorial Medical Center, 1234 Napier, St. Joseph, MI 49085; (616) 927-5131. 574H/J

Cardiovascular and thoracic surgeon needed immediately for adult cardiovascular and thoracic surgery practice. Prefer recent graduate but will consider all qualified. BC/BE. Salary leading to partnership.

Submit CV with references to R. Michael Kennerly, MD, 445 Biltmore Ave., Suite 103, Asheville, NC 28801; (704) 258-9446. 576H/J

Cardiovascular and thoracic surgeon to join established 3-man private practice in mid-South. Practice includes thoracic, cardiac, and vascular surgery. Must be board certified or eligible in thoracic and cardiovascular surgery. Prefer candidate who has recently completed his/her training. Please respond with curriculum vitae.

Please respond to Box 580. 580H/A

Board certified/eligible cardiothoracic surgeon for busy 1-physician practice in Atlanta. Transplant experience. Future partnership.

Send resume to Georgia Cardiac Surgical Associates, PC, PO Box 134, College Park, GA 30237. 583H/I

Ohio—thoracic and cardiovascular surgeon: Position available for responsible board certified/eligible PA. Your responsibilities will include assisting in the operating room and care of preoperative and postoperative cardiothoracic and vascular patients. Excellent salary and benefits.

Please send resume to PO Box 4037, Dayton, OH 45402. 584I

Cardiothoracic surgeon, BE/BC, to join private practice in small community on west coast of Florida. Recent graduate preferred. Florida licence a plus. Send CV and references.

Please respond to Box 586. 586I/J

General thoracic surgeon, mature, BC, with considerable experience in all aspects of general thoracic surgery with particular expertise in esophageal function and surgery. Please respond with CV.

Please respond to Box 587. 587I

Wanted—thoracic surgeon, BE/BC, to join a board-certified surgeon for noncardiac thoracic surgery and peripheral vascular surgery in a growing medical center in upstate New York.

Please respond to Box 588. 588I/K

Cardiothoracic/vascular surgeon, BC/BE, wanted to join established private practice in Philadelphia area. Practice includes all aspects of adult cardiac, thoracic, and vascular surgery.

Send CV with experience and references to Maryann Dwyer, Cardiothoracic Associates, 201 North 8th St, Room 325, Philadelphia, PA 19106. 589I

Cardiothoracic surgeon—metropolitan Kansas City area: An exceptional opportunity exists for BC/BE cardiothoracic surgeon in a large, expanding, independent multispecialty group. The practice is conveniently located close to hospitals, affordable luxury housing, and nationally recognized school systems. There is a strong, diversified mix of patients with an excellent referral base. A very attractive incentive package is available including a guaranteed income. Malpractice, marketing, and relocation assistance is also available.

Please send curriculum vitae to John Storm, 51 N 12th St, Kansas City, KS 66102; (913) 281-7775, FAX (913) 281-8494. 591I/L

Cardiovascular/thoracic surgeon: A progressive major regional multispecialty group in Texas Gulf Coast area is seeking a BC/BE cardiovascular /thoracic surgeon. Applicant should be technically strong in all phases of adult cardiac surgery. Cardiology department has three cardiologists. Practice is located in subtropical climate in an upward bound economic environment. Guaranteed salary with bonus plan. Shareholder status available 12–18 months. Malpractice insurance, interview, and relocation expenses included in benefits package. Location is attractive to those who enjoy outdoor sports such as fishing, golfing, and sailing. Look into our great team!

Contact Amanda Fuhro, Valley Diagnostic Medical and Surgical Clinic, PA, 2200 Haine Dr, Harlingen, TX 78550; (512) 421-5199. 592I/L

Cardiovascular/thoracic surgeon(s), BC/BE, needed to expand cardiovascular program in the upper midwest. Major referral center with medical school affiliation. Join four BC surgeons in providing cardiac and vascular services in this family community of 100,000.

Please respond to Box 593. 593I/J

Cardiothoracic surgeon wanted for January 1992: Expanding private practice in Philadelphia. Adult cardiac and thoracic surgery. Academic appointment. Eventual partnership.

Send CV and References to PO Box 1295, Philadelphia, PA 19105. 594I/K

Cardiovascular-thoracic surgeon, Eligible or board certified, wanted to join established private practice in south Texas. Primary adult cardiac surgery, thoracic and vascular in modern hospital facilities. Please send CV, references, and clinical experience.

Please respond to Box 596. 596I/J

The University of Medicine and Dentistry of New Jersey—New Jersey Medical School and United Hospitals Medical Center–Children's Hospital of New Jersey are opening their search for a Division Director of Pediatric Cardiovascular Surgery. The Medical School and Children's Hospital are seeking a skilled and experienced neonatal and infant cardiac surgeon who is also experienced in performing cardiac surgery on children and adolescents. This well-qualified individual should be able to provide the necessary leadership in patient care, research, and education. Candidates must be board certified or equivalent.

To apply, please send your curriculum vitae to Linda C. Reich, Office of the Dean, UMDNJ-New Jersey Medical School, MSB-C662, University Heights, 185 South Orange Ave, Newark, NJ 07103-2714. The UMDNJ is an affirmative action/equal employment opportunity employer, m/f/h/v, and a member of the University Health System of New Jersey. 597I

Cardiothoracic and vascular surgeon, American graduate, BE/BC, needed to join an established practice in the Southwest, specializing in adult cardiac, thoracic, and vascular surgery. Salary and fringe benefits leading to partnership. Send curriculum vitae.

Please respond to Box 598. 598I/J

SITUATIONS WANTED

Cardiovascular and thoracic surgeon, extensive experience, ABS, ABTS, university trained. CABG with IMA and vein, mitral reconstruction and valve replacement, extensive pulmonary and vascular experience. Solid research interest with 90 published papers. Wishes to join active cardiovascular surgery practice. Curriculum vitae and references on request.

Please respond to Box 571. 571H/J

Board-certified cardiovascular surgeon, 48, university trained, wishes to relocate. Interested in group practice. CV and references upon request.

Please respond to Box 573. 573H/A

CCP perfusionist looking for expanding program or new program in western half of the US. Have perfusion equipment and supplies to outfit one OR room. Will consider all areas and economic considerations. Have experience in starting new programs.

Please respond to Box 581. 581H/I

American-born cardiothoracic surgeon (ABTS 1980; recertified 1990) with strong training seeks new challenges in program development or private practice. Broad CVT experience with low mortality, solid work history. CV, caselog, references available.

Please respond to Box 585. 585I/J

Thoracic and vascular surgeon, university trained, 37, ABS, ABTS wishes to relocate. Prefer southeastern US. CV and references upon request.

Please respond to Box 590. 590I/K

FELLOWSHIPS

Fellowship in vascular surgery—the Deborah Heart and Lung Center is currently seeking qualified applicants for a vascular fellow position.

Please contact Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart and Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, Ext 359. 566G/J

Surgical residents/fellows—clinical residency/fellowship positions available in adult and pediatric cardiothoracic surgery. Large volume of cases. New Jersey license required. Excellent salary and benefits.

Please contact Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart and Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, Ext 359. 568G/J

Fellowship in Cardiothoracic Surgery and Transplantation: A 1- to 2-year fellowship in cardiothoracic surgery with primary involvement in heart and lung transplantation. Opportunity for training in circulatory support devices. Experience in cardiac surgery desirable. Competitive salary and benefits.

Please respond with curriculum vitae and references to Mohsin G. Hakim, MD, Director of Heart and Lung Transplantation, Willis-Knighton Medical Center, PO Box 32600, Shreveport, LA 71103; telephone: (318) 632-2508. 595I

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GUTHRIE

Guthrie Clinic

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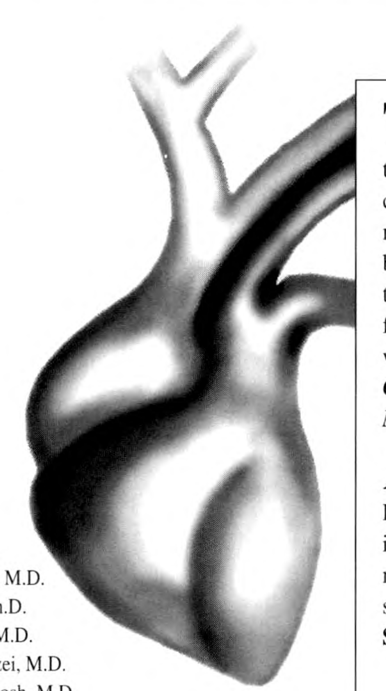

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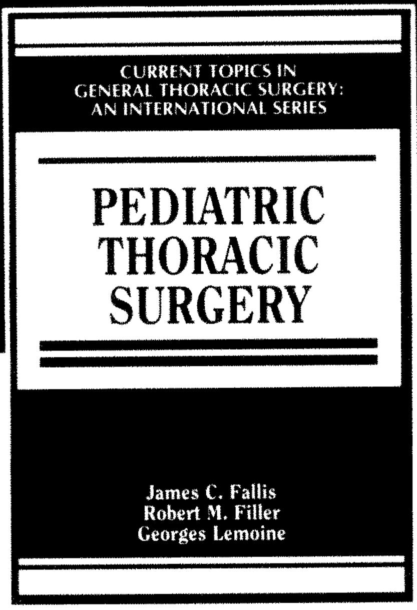
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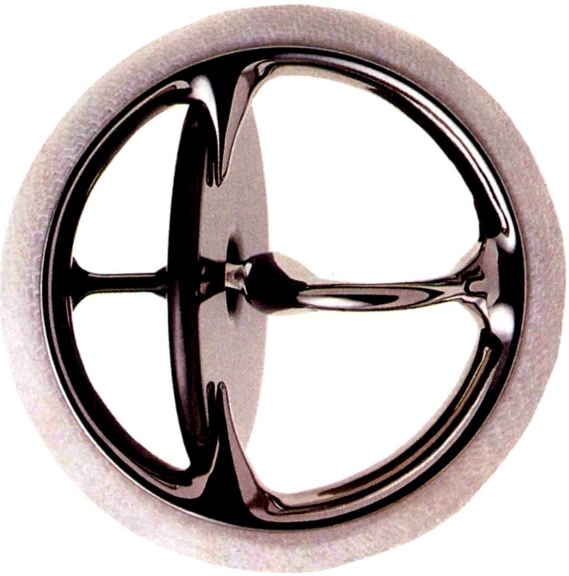

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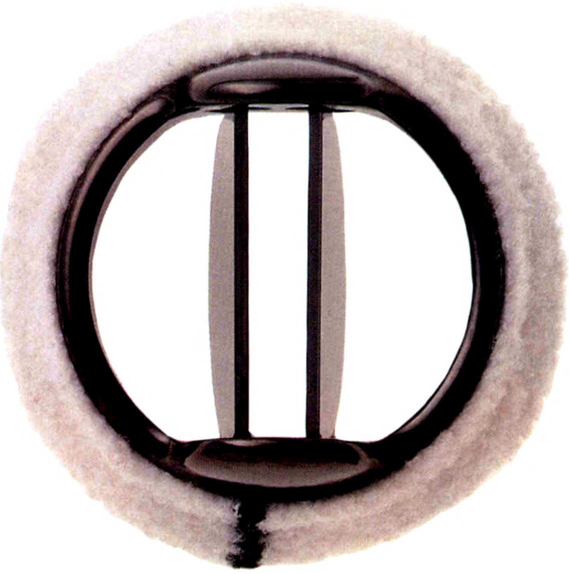

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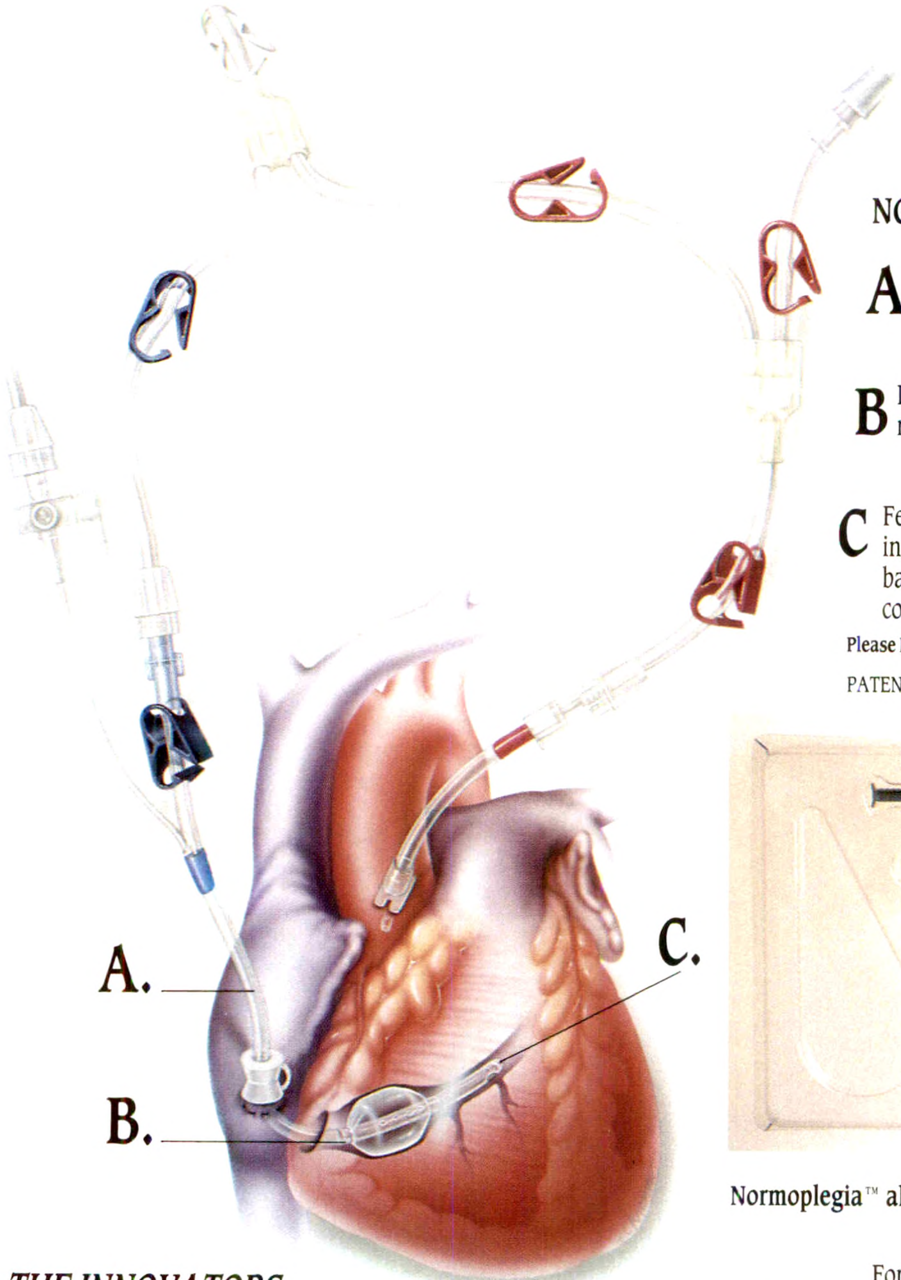

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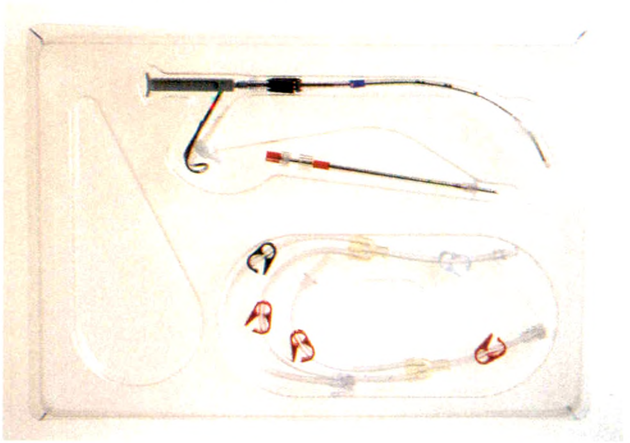

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November 1991

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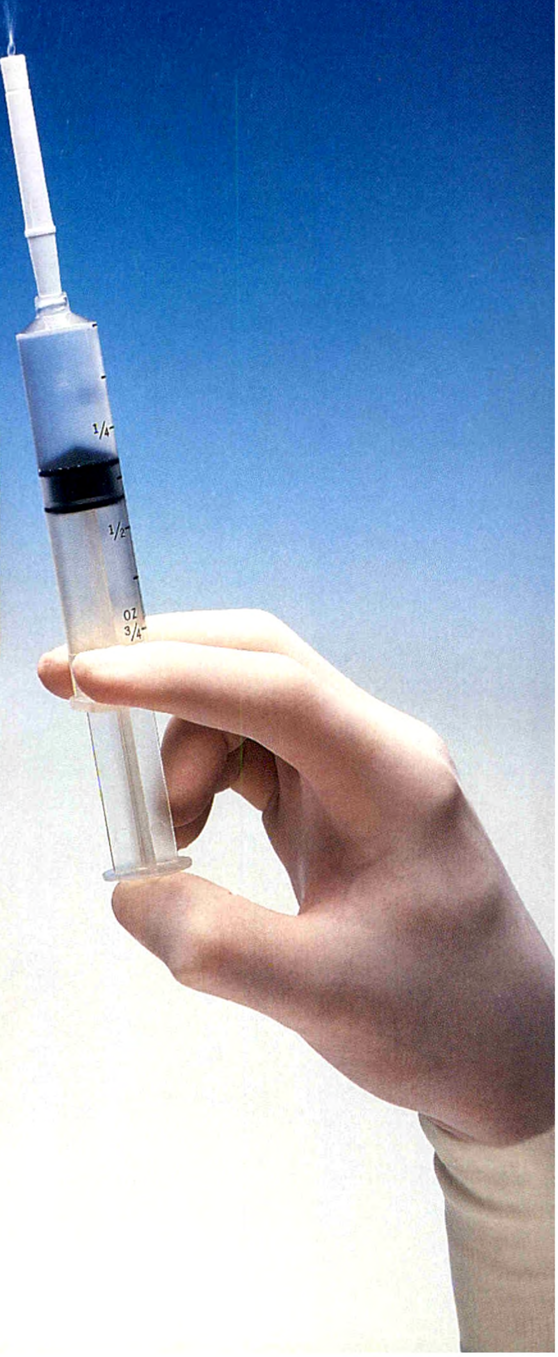

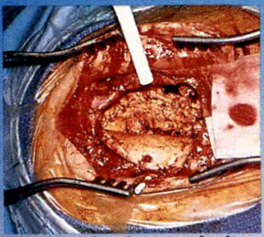

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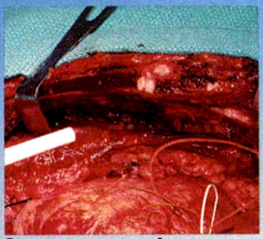

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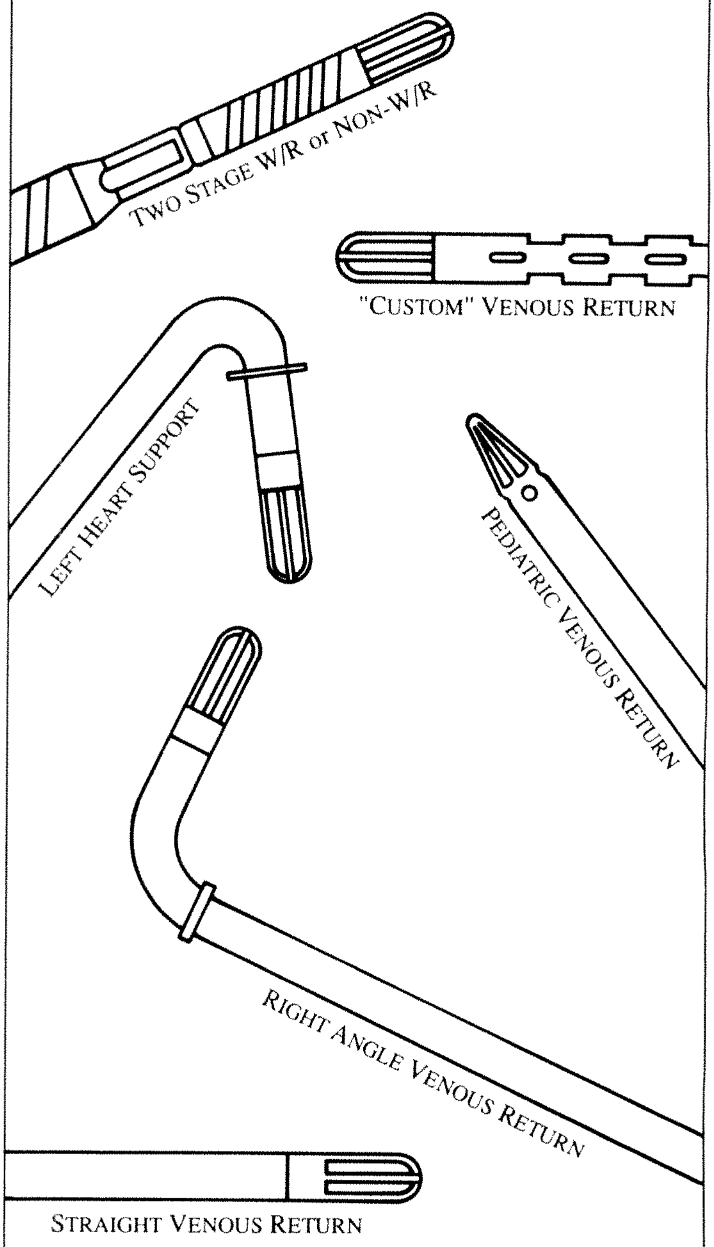

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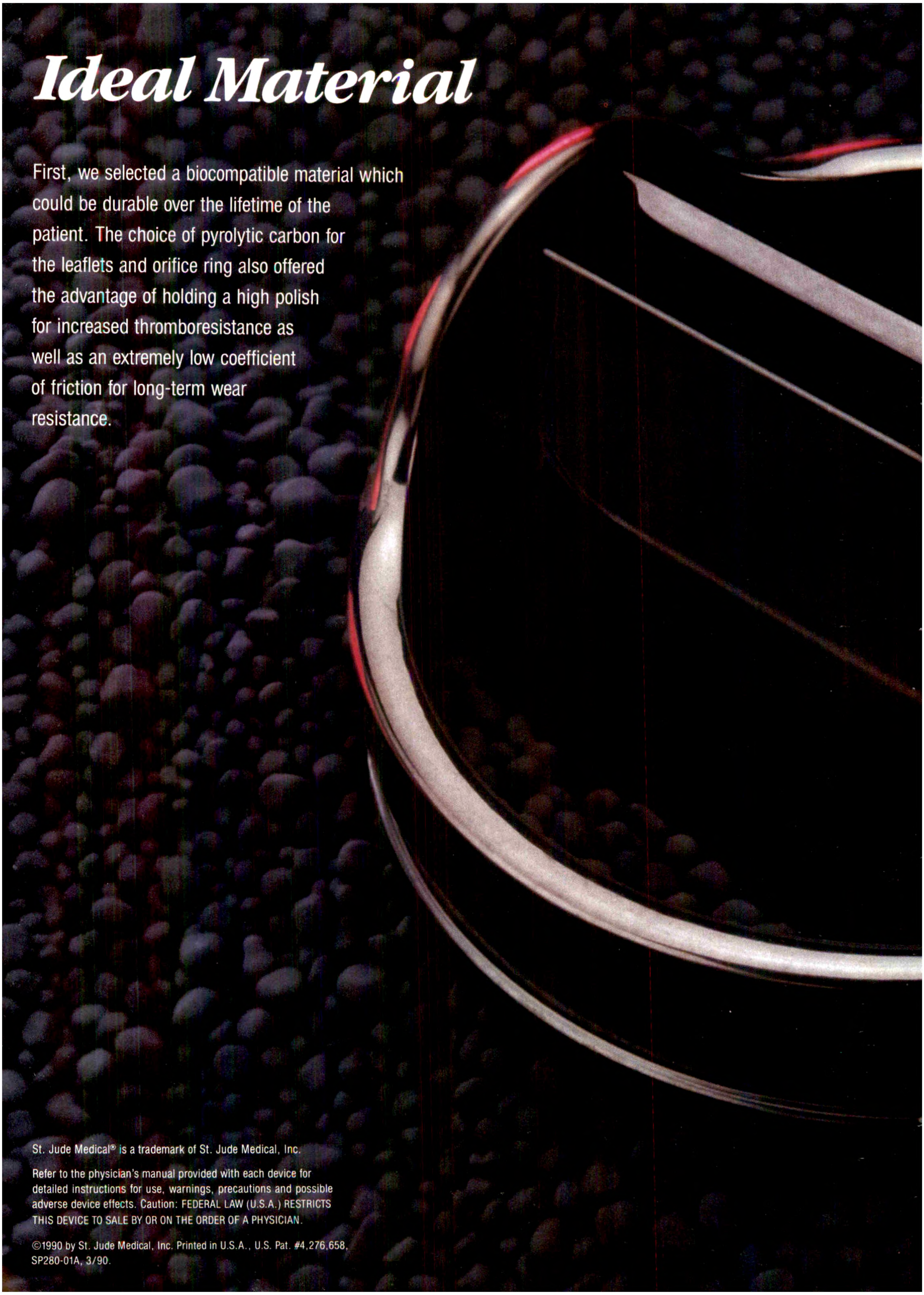

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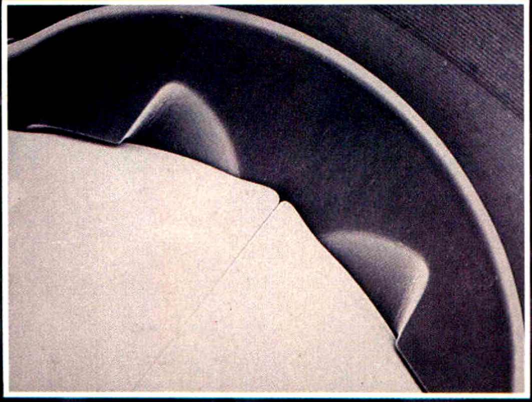

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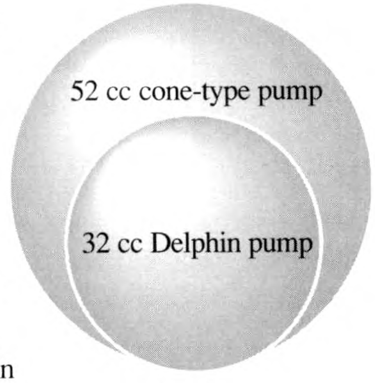
Air handling and centrifugal blood pumps

By far the most popular reason given for using centrifugal pumps is safety.[1] Certainly, the inability to pump gross air (a large bolus of air) is a significant safety advantage of centrifugal pumps. Yet not all centrifugal pumps perform equally well when it comes to air handling.

Protection against gross air

A large bolus of air will deprime a centrifugal pumphead. Once the pump is deprimed, the flow rate drops to 0 and the air is held up in the pumphead, though the pump continues to spin. Roller pumps, on the other hand, will continue to force air on towards the patient.

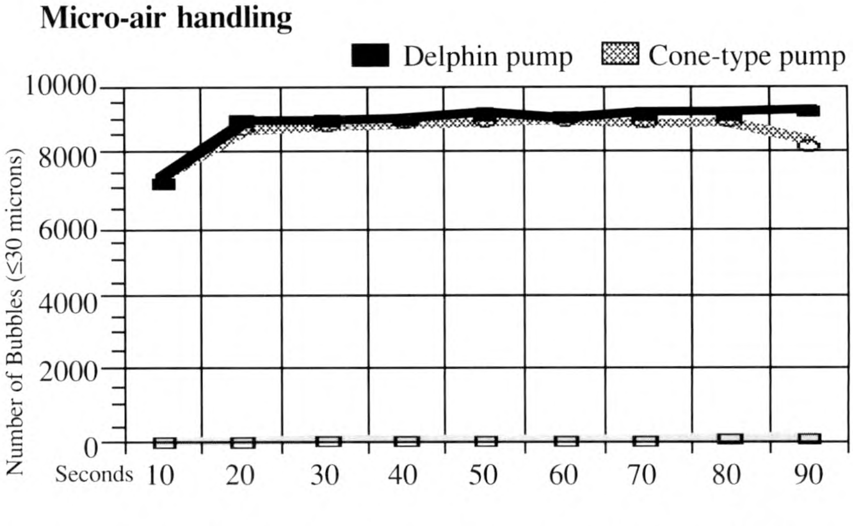
Just 32 cc of air will deprime a Delphin pumphead. Approximately 52 cc are required to deprime a cone-type centrifugal pump.[2] The Delphin pumphead protects patients by depriming with a relatively small amount of air.



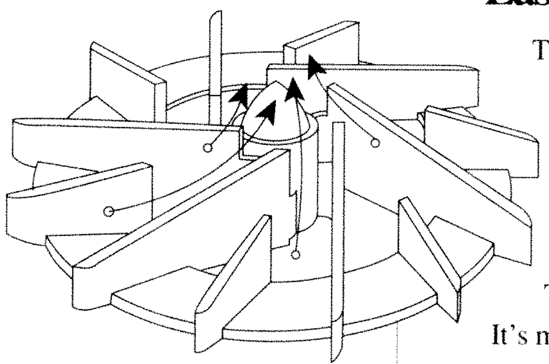
Air to deprime

Micro-air handling

All arterial pumps, including centrifugal pumps, will pump micro-air to some extent.[3] Under similar operating conditions, centrifugal pumps are essentially equivalent in their micro-air handling abilities.[4]



Although micro-air handling is essentially equivalent for all centrifugal pumps (as shown on this graph), the Delphin system deprimes with only 32 cc of air.

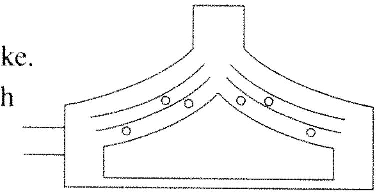


The open impeller design of the Delphin pumphead facilitates debubbling.

Easy debubbling

The open impeller design of the Delphin pumphead lends itself to convenient debubbling. Bubbles trapped beneath the cones are difficult to remove during priming. In an effort to debubble, many perfusionists resort to vigorously tapping the cone-type pumphead, a practice one manufacturer warns "may cause device malfunction."

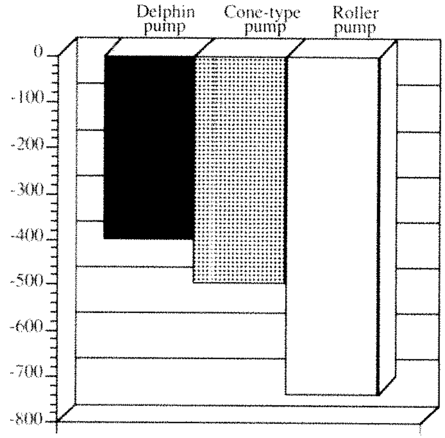
Tap the Delphin pumphead if you like. It's made of durable polycarbonate which has twice the impact strength of the acrylic used in some pumpheads[5].



The constrained design of cone-type pumpheads tends to trap bubbles during priming.

What about cavitation?

Cavitation, the sudden formation of micro-bubbles in solution, is caused by negative pressure. The greater the negative pressure potential at the pump inlet, the greater the risk of cavitation. Conversely, the lower the negative pressure, the greater the protection against cavitation. The Delphin system provides protection against cavitation by generating low levels of negative pressure. Should the inlet be occluded, the Delphin system produces less negative pressure than other pumping systems (for equivalent flow rates and pressure readings).



Negative pressure generated
(inlet occluded at 4L/min and 400 mm Hg)

The Delphin pump protects against cavitation by generating low levels of negative pressure.

For more information about centrifugal pumps, or a free, no-obligation clinical evaluation of the Delphin centrifugal system, call **800/521-2818.** Or write Sarns, 3M Health Care, 6200 Jackson Road, Ann Arbor, MI 48103.

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Footnotes

[1] Based on a poll of *Heart-To-Heart* readers conducted in June, 1990. Almost three out of four respondents were already using a centrifugal pump for arterial perfusion (67%) or planned to (7%). A solid 26% did not use centrifugal pumps at all, and 15% were infrequent users.

[2] Pumps were flowing at 3.5 L/min with a pump outlet pressure of 300 mm Hg, a static pressure head of 30 mm Hg, and a mean arterial pressure of 60 mm Hg. To obtain these flow rates and pressures, the Delphin pump was operating at 2,350 RPMs, and the cone-type pump at 2,800 RPMs.

[3] Reed, Charles C. and Stafford, Trudi B. *Cardiopulmonary Bypass.* Houston: Texas Medical Press, Inc., 1985, p. 377.

[4] Numbers of 30 micron micro-bubbles were counted using a TLL Micro-bubble Activity Monitor attached near the centrifugal pump outlets following the introduction of 10 cc of air into each pumphead.

[5] Based on Izod impact of plastics testing for polycarbonate and acrylic using ASTM test method D256.

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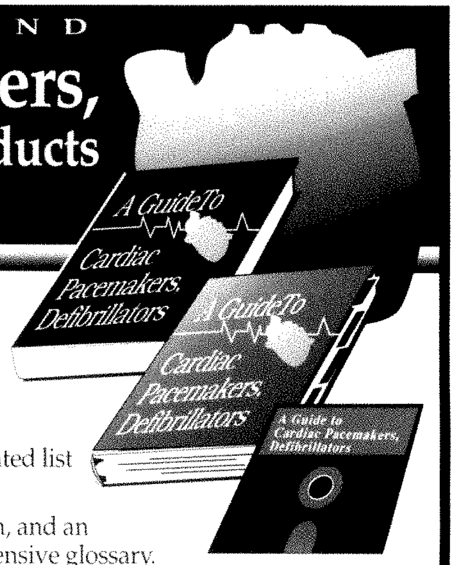

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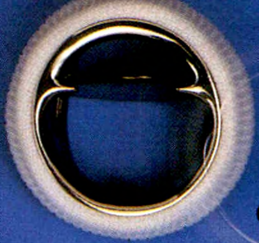

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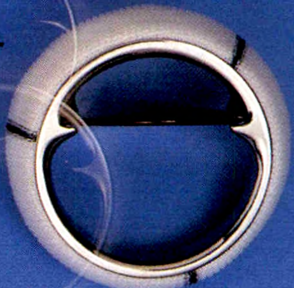

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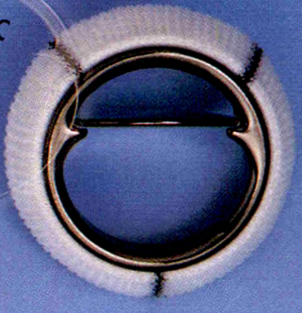

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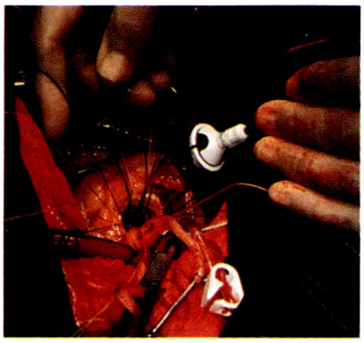

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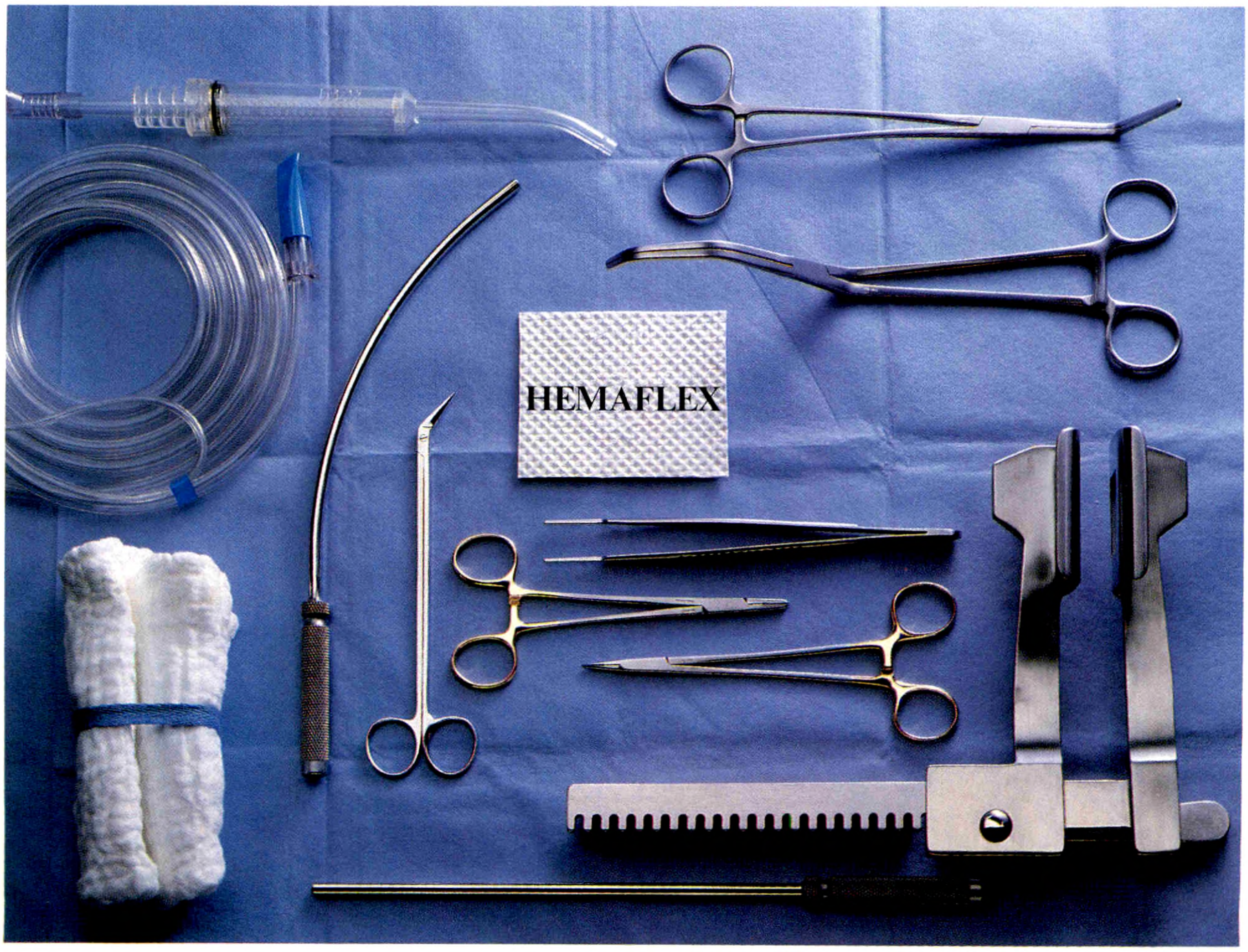

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The frame is designed to be flexible at the orifice as well as at the commissures. The compliance of the stent's commissure supports is intended to reduce the loading shock at the valve commissures and free margin of the leaflets. The flexibility of the orifice is intended to reduce the loading shock at the base of the leaflet. The flexible orifice concept is based on the physiology and mechanics of natural heart valves and reported experience with implantation of unstented homografts.

The lightweight frame is made of Elgiloy,* a corrosion-resistant alloy, chosen because of its superior spring efficiency and fatigue-resistant characteristics.

The metal frame is covered with a porous, knitted polytetrafluoroethylene cloth to facilitate tissue ingrowth and encapsulation. The inflow orifice is contoured to support the right coronary cusp of the porcine valve. The commissure supports are not equidistant to conform to the anatomical configuration of the porcine aortic valve. The suture ring has a soft, silicone-rubber insert that is covered with a porous, seamless polytetrafluoroethylene cloth. The aortic sewing ring has been scalloped to conform to the natural aortic root. The compliant nature of the suture ring facilitates coaptation between the valve and an often irregular or calcific tissue bed.

The porcine aortic valves are fixed in buffered glutaraldehyde, and then mounted on cloth-covered frames. After mounting, the valves are tested in vitro to ascertain their competency and flow characteristics. After final inspection, the valves are sterilized, rinsed, and then transferred into fresh glutaraldehyde and packaged under aseptic conditions.
Indications
Bioprostheses are indicated for use in patients suffering from valvular heart disease. Aortic and mitral valvular heart disease are conditions involving any of the following: obstructions of the heart valves, or stenosis; leakage of the valves, known as regurgitation or insufficiency; and combinations of the two, sometimes referred to as mixed disease or combined lesions.

Valvular heart disease may be caused by any number of factors, including congenital abnormalities, infection by various microorganisms, degenerative calcification, rheumatic heart disease and rupture or dysfunction of the mitral subvalvular mechanism secondary to myocardial ischemia.

Bioprostheses are used particularly in those patients for whom long-term anticoagulation is contraindicated or who may be difficult to maintain on anticoagulation therapy.

The Carpentier-Edwards Bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of th[illegible]al valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis.
Warnings
For Single Patient Use Only
In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, and in patients who are on maintenance hemodialysis.

The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient and consideration of alternative methods of treatment. Overall durability, especially long-term, has not been established for bioprostheses.

Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery.

Note: Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis (see Clinical Experience).

Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed.

Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection.

Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombus. The appropriate anticoagulation therapy must be determined by the physician on an individual basis.

Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc., should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result.

Carpentier-Edwards Bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.

Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards Bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided.

Both the valve and glutaraldehyde storage solution are sterile. The outside of the jar is not sterile and must not be placed in the sterile field. Furthermore, the valve and container should never be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation.

Storage between 10°C and 25°C (50-77°F) is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue.

Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with the eyes, seek medical attention.
Surgical Precautions
1. When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result (Ref. 50). The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient.
2. Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts.
3. Left ventricular perforation or ulceration has been reported (Ref. 16). Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage.
4. The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15 mm, produced a pursestring effect, causing compression of the valve orifice.
5. When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures.
6. Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue.
7. As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs, which would interfere with proper valvular function.
8. The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence.
   Note: The contrasting suture material should not be removed from the prosthesis.
9. In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia.
10. A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar and implantation data card; if any differences are noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal.
11. Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation.

Complications
As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days), necessitating reoperation and replacement of the prosthetic device.

Complications associated with the use of Carpentier-Edwards Bioprostheses compiled from the literature and from reports received through the complaint handling system in accordance with the United States (Federal) regulations establishing Good Manufacturing Practices, 21 CFR Section 820.198, include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, degeneration, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrhythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct.

Note: Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies (Ref. 10) show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes a potential relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the post operative period, and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards Bioprostheses to calcification as compared to other commercially available porcine valves.
Clinical Experience
Clinical data, available on 288 patients requiring isolated aortic valve replacement (AVR) and on 241 patients requiring isolated mitral valve replacement (MVR) with Models 2625 and 6625 Carpentier-Edwards Bioprostheses, respectively (mean follow-up of 2.6 and 2.2 years, respectively), indicate operative (< 30 days) and postoperative (> 30 days) survival rates of 97.2% and 95.0% per patient year, respectively, for the isolated AVR population, and 94.9% and 93.2% per patient year, respectively, for the isolated MVR population.

In the isolated AVR population there were a total of 208 (72.2%) males and 80 (27.8%) females with a mean age (± standard deviation) of 65.2 (± 10.1) years and a range of 24-88 years. The indications for valve replacement were stenosis (52.4%), regurgitation (13.5%), mixed disease (18.4%), and previous prosthetic mitral valve dysfunction (2.1%). For 13.5% of the patients, the diagnosis for replacement is unknown.

In the isolated MVR population there were a total of 79 (32.8%) males and 162 (67.2%) females with a mean age of 63.3 (± 10.5) years and a range of 30-84 years. The indications for valve replacement were stenosis (27.0%), regurgitation (36.1%), mixed disease (19.9%), and previous prosthetic aortic valve dysfunction (7.5%). For 9.5% of the patients, the diagnosis for replacement is unknown.

The follow-up methods used at each clinic included hospital visits, office visits, and contact by telephone or letter with either the patient himself, his family, or his local doctor.

These data were compiled from a multicenter clinical trial conducted by Edwards CVS Division.

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- ☐ The *text* for case reports should be *no more* than 4 double-spaced typewritten pages. If tables or illustrations are included, the text must be reduced. The text for a "how to do it" article should be even briefer, but with detailed illustrative material. Case reports and "how to do it" articles that exceed the allowable length will not be considered for publication.
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8. Wilkins EW Jr, Grillo HC, Scannell JG, Moncure AC, Mathisen DJ. Role of staging in prognosis and management of thymoma. Ann Thorac Surg 1991;51:888–92.

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12. Katz PO. Achalasia. In: Castell DO, Richter JE, Dalton CB, eds. Esophageal motility testing. New York: Elsevier Science Publishing Company, 1987:107.

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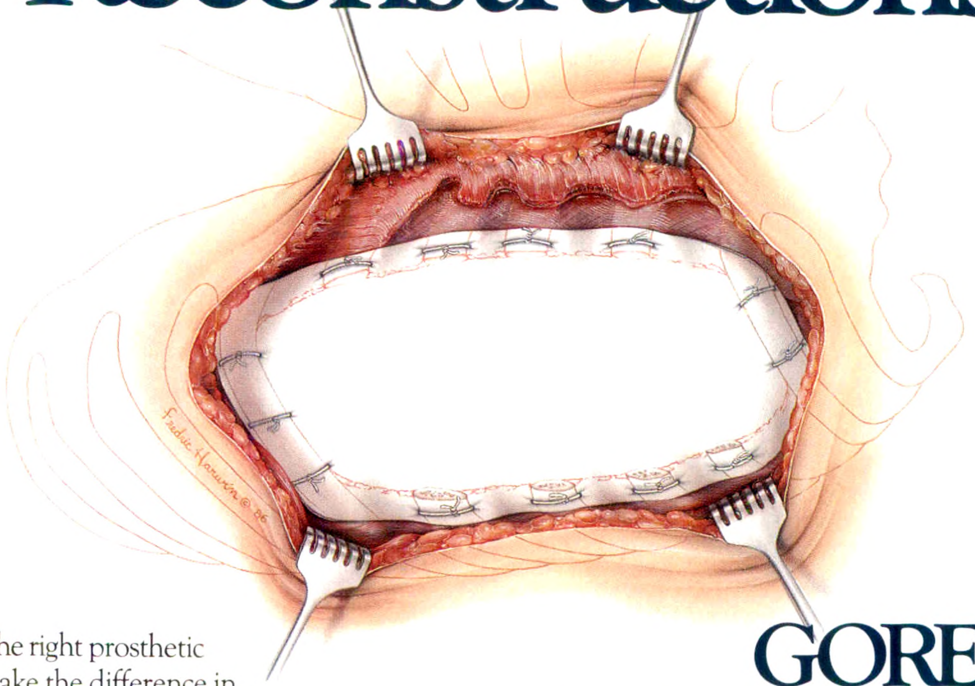

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Damus-Stansel-Kaye Procedure: Personal Observations

Gordon K. Danielson, MD

Mayo Clinic/Foundation, Rochester, Minnesota

Dr di Carlo and associates [1] have presented a comprehensive review of their experience in infants with the ingenious operation known as the Damus-Stansel-Kaye (DSK) procedure [2, 3], among other names. The operation achieves a functional great artery switch in patients with complete transposition of the great arteries or other complex anomalies with transposition hemodynamics without the need to relocate the coronary arteries. The main pulmonary artery is transected near the bifurcation and anastomosed to the posterolateral aspect of the ascending aorta. Any ventricular septal defect is closed, and an extracardiac conduit establishes right ventricle to pulmonary artery continuity. If present, pulmonary artery narrowing from a pulmonary artery band and valvular or subvalvular pulmonary stenosis must be relieved to allow unrestricted outflow from the left ventricle to the aorta. This arrangement leaves an anatomical double-outlet right ventricle, but the aortic valve remains closed throughout the cardiac cycle because the pressure in the aorta is higher than the pressure in the right ventricle. The history of development of this concept is a fascinating one; the appearance of multiple independent letters and reports within a short period of time probably represents another example of "an idea whose time has come."

See also page 1148.

The first reference to this concept was in a letter to Dr Dwight C. McGoon from Paul S. Damus, a West Coast resident in general surgery, dated September 30, 1972, which described and illustrated a new operation for transposition [4, 5]. A manuscript was subsequently submitted to *The Annals of Thoracic Surgery* in December 1973, but for uncertain reasons, perhaps because there was no report of carrying out the procedure on cadaver hearts, the manuscript was not accepted [4, 6]. Subsequently, in May 1975, Dr H. C. Stansel, Jr, independently proposed the operation and described experimental procedures on postmortem specimens that confirmed that, with suitable tailoring, anastomosis of the pulmonary artery to the aorta was feasible [7]. Later in the same year, also independently, Dr Michael P. Kaye from Rochester, MN, published the same concept [8], and Dr F. Alvarez from Madrid, Spain, described the operation in the Spanish literature [9].

Doctor McGoon was the first to perform the operation clinically (in 1975, but the patient did not survive) and the first to achieve an operative survivor (a 5-month-old infant on February 12, 1976) (McGoon DC; personal communication). The patient lived 2 years before dying of pulmonary hypertension. The first patient to survive to the present time was a 3-year-old who was operated on July 22, 1976, the first of 2 patients on whom the first hemodynamic and angiographic data related to the DSK operation were published [10]. By 1982, the DSK procedure had been performed at our institution on 20 patients with complete transposition and on 4 patients with double-outlet right ventricle and subpulmonary ventricular septal defect [2]. We [11] and others [12–14] have expanded the application of this concept to include patients having univentricular heart with subaortic stenosis and patients having other complex anomalies.

Although the DSK concept has been employed now for 15 years, controversy still exists regarding its role in the treatment of various cardiac anomalies. The major advantage of the operation is that the coronary arteries do not need to be transferred from the aorta to the pulmonary artery. This is particularly advantageous when there are dense adhesions at the base of the heart from a previous operation, which make coronary artery transfer hazardous, or when there are coronary artery anomalies such as an intramural coronary artery, which make coronary artery transfer difficult. Additionally, the operation is advantageous when the great artery relationships preclude coronary artery transfer.

The major disadvantage of the DSK procedure is the need for a right ventriculotomy and an extracardiac conduit, which becomes more of a problem in infancy, as reported by di Carlo and associates in their series [1]. Hospital mortality was high in infants in the early experience of all centers, but has declined substantially in recent years concomitant with increasing experience in repair of cardiac anomalies of all types in infants. In our opinion, the DSK operation is similar to operation for truncus arteriosus in infancy and also in older ages with respect to mortality, morbidity, and need for future conduit replacement.

There are several technical pitfalls that must be avoided to have a successful result with the DSK procedure. Because the pulmonary valve becomes the new "aortic" valve, great care must be taken not to distort the pulmonary valve when the pulmonary artery is anastomosed to the ascending aorta. Even minor tension or distortion can produce serious valvular regurgitation. This is especially important when the main pulmonary artery is short or when a pulmonary artery band is present; the surgeon should be prepared to augment the main pulmonary

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artery if there is any concern about distortion of the pulmonary valve.

Another consideration is to avoid compression of the conduit between the heart and sternum. We have found it helpful to place the extracardiac conduit either to the right or left of the ascending aorta, as indicated, to minimize the length of conduit lying beneath the sternum [10].

Durability of the pulmonary valve as the new "aortic" valve has the same limitations as in the arterial switch procedure. Late insufficiency of the neoaortic valve was reported by di Carlo and associates [1] in 1 patient, and there is 1 recent patient in our series who has required neoaortic valve replacement because of progressive valvular insufficiency (Puga FJ; personal communication).

The question of whether to close the aortic valve/subaortic area remains controversial [3]. Closure of the aortic valve is not necessary to prevent the valve from opening if cardiac hemodynamics are satisfactory. In 1 of our early patients who had severe pulmonary hypertension, episodic increases in pulmonary vascular resistance caused the aortic valve to open, resulting in severe cyanotic episodes. Reoperation was performed to close the aortic valve; this prevented cyanotic episodes but did not prevent profound low cardiac output when pulmonary vascular resistance increased. It was not until the pulmonary hypertension was controlled with vasodilators, sedation, and hyperventilation that the patient improved, and it seems likely closure of the valve might have been obviated by earlier control of the pulmonary hypertension.

The principal reason given for closure of the native aortic valve is to avoid development of aortic valve insufficiency [3]. Early onset aortic insufficiency is probably related to distortion of the aorta by the anastomosis. The reason for late onset of this complication is not known, but it appears to be a definite problem: Di Carlo and associates [1] report 2 cases in their series and we have encountered 1 patient in our experience who required late (9 years after repair) reoperation for aortic valve insufficiency. Suture of the aortic valve at the time of initial repair may not be an answer; 1 of the patients of di Carlo and associates required a second procedure for treatment of aortic valve insufficiency after valve suture. In my practice, the aortic valve/subaortic area is not closed at the time the DSK procedure is performed because of concern that the sutures, pledgets, or patches may promote stasis and thrombosis in the aortic root and to simplify the operation and shorten the duration of aortic cross-clamping. An additional advantage of leaving the aortic valve unclosed is that, should conduit obstruction develop and not be recognized, exertional cyanosis will signal the need for conduit replacement. Further long-term evaluation of all patients undergoing this procedure should yield an improved perspective on this controversial issue.

The DSK principle has been employed successfully by us [11] and others [12–14] to relieve subaortic stenosis in various forms of univentricular heart, especially double-inlet left ventricle with rudimentary right ventricle (outlet chamber) giving origin to the aorta. However, we have generally abandoned the DSK approach in favor of direct resection of the subaortic stenosis either through the aorta and retracted aortic valve or through the outlet chamber [15]. In a personal series of 27 patients who underwent subaortic resection in relation to a modified Fontan procedure, there were only 3 deaths (cardiac failure, infection, and pulmonary embolism) and no instances of complete heart block, results that are notably better than our previous experience with the DSK procedure in relation to a modified Fontan procedure.

Dr di Carlo and colleagues have employed a previously described modification of the DSK operation [14] in which the ventricular septal defect patch is carried to the right of the aortic valve (similar to the Rastelli principle), with the thought that the aortic sinuses would be washed out with antegrade blood flow through the aortic valve. Whether the hoped for advantages of this modification merit the increased time needed for placement of the baffle and the increased risk of residual ventricular septal defect (the incidence of residual ventricular septal defect was higher in our Rastelli series than for ventricular septal defect closure in other cardiac anomalies requiring a less extended baffle) remains to be determined by further experience.

The DSK operation has been largely supplanted in the treatment of transposition of the great arteries and double-outlet right ventricle with subpulmonary ventricular septal defect by direct arterial switch procedures and in the relief of subaortic stenosis in univentricular heart anomalies by direct resection of the obstructing tissue. However, Dr di Carlo and colleagues have confirmed that the DSK operation does provide an excellent solution to certain difficult anatomical situations where other procedures are not feasible or are hazardous, and it should remain in the cardiac surgeon's armamentarium, to be recalled when the occasion merits.

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Therapeutic Thoracoscopy: New Horizons for an Established Procedure

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The development of endoscopic video capability and corresponding instrumentation has opened new horizons in the fields of thoracic, orthopedic, general, gynecological, and urological surgery. This is most exemplified by laparoscopic cholecystectomy, which was an unknown procedure 2 years ago and now is accepted as a common procedure in the community hospital.

Thoracoscopy was originally described by Jacobaeus [1] in 1922, and then not used again until the 1950s. The procedure was then dormant until the early 1970s, when it was reinstituted by me and others [2]. There are now greater than 160 articles in the literature on diagnostic thoracoscopy. My personal experience with diagnostic thoracoscopy is now greater than 250 cases. It is used in our institution frequently, and has been taught to our residents since 1974.

See also page 1176.

The development of endoscopic video systems and instrumentation has allowed *therapeutic* thoracoscopy for peripheral lung resection, pleural biopsy, open lung biopsy, closure of leaking blebs, parietal pleurodesis, and pericardiectomy and biopsy. Without the technical equipment and instrumentation, this would not be possible.

Landreneau and associates [3] present in this issue of *The Annals* an excellent description of a new technique that has been infrequently reported. As stated, it is a technique that the thoracic surgeon should have in his or her surgical armamentarium [3]. Their description of the surgical technique is well presented and illustrated. Their recommendations that this procedure should be limited to the high-risk or medically compromised patient should be well heeded. It should be noted that their patient was at an increased risk, but not an excessive pulmonary risk, with forced expiratory volume in 1 second of 1.4 L. Perhaps an open procedure should have been considered, with a more extensive resection, ie, extended wedge or segmentectomy, with a better long-term 5-year survival. In addition, they extended the incision to 3 cm, which would have allowed introduction of a 30-mm flexible rotator stapler. This would have decreased the operative time considerably and reduced air leaks postoperatively.

With the introduction of thoracoscopic stapling, the same resection can be accomplished by using two ports 1 cm in length. The introduction of 30-mm stapling devices, dissectors, coagulators, and autotying sutures has facilitated this new development in therapeutics.

The application of the neodymium:yttrium-aluminum garnet (Nd:YAG) laser for this technique was originally reported by LoCicero and co-workers [4]. The application is similar to that of electrocautery resection of small pulmonary neoplasms, initially described by Pearlman and later amplified by Cooper and colleagues [5]. More flexibility with the laser could be obtained if it was used through a fiberoptic bronchoscope inserted through the thoracoscope. The quartz fiber of the Nd:Yag laser inserted through a rigid endoscopic system has a limited rotational movement. I doubt that the Nd:YAG laser will have wide acceptability in thoracoscopy with the advent of new instrumentation. Laser sclerosis of the parietal pleura is innovative, but will probably have limited use, as sponge pleurodesis can be easily accomplished with the newer instruments. In addition, the persistence of air leaks from the pulmonary parenchyma after laser resection can be substantial and require considerable time for closure.

While we thoracic surgeons must continue to be innovative in our approach to problems, we must remember that our goal in the surgical treatment of non–small cell carcinoma of the lung is to cure the patient while preserving lung tissue. The use of epidural anesthesia in thoracic surgery, combined with limited thoracotomy and limited resection, has resulted in cure rates of 33% to 35% in severely compromised patients with a forced expiratory volume in 1 second less than 1 L and an FEV_{25-75} less than 0.6 L [6].

The local recurrence rate with a 1-cm margin is greater than 20%. If this technique is to be applied to pulmonary resection, then consideration should be given to postoperative coned-down radiation therapy.

The use of double-lumen endotracheal anesthesia greatly facilitates the procedure, as pointed out by Landreneau and associates.

This editorial is not written as a criticism of this well-written report, but is a reminder that thoracic surgeons should use this application of the Nd:YAG laser in the treatment of lung cancer infrequently, as other methods may provide for greater safety, control, decreased operative time for the patient, and better 5-year survival.

The next year will bring wide application of therapeutic thoracoscopy to open lung biopsy, mediastinal node biopsy, closure of pneumothoraces, and pericardial bi-

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opsy and drainage. New horizons have been made possible for an established procedure.

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Introduction

Robert L. Replogle, MD

Michael Reese Hospital, Chicago, Illinois

Fig 1. Dr Ralph D. Alley

The first Ralph D. Alley lectureship was planned to serve two functions. The first was to recognize the organization of the Thoracic Surgery Research & Education Foundation. This new endeavor, committed to the improvement of our specialty, was initiated in December 1988 in large part through a substantial gift from Mr David Sheridan, long-time friend and co-worker of Dr Alley. We are pleased that Dave is in the audience today.

This named lecture commemorates the contributions of Ralph D. Alley (Fig 1) to thoracic surgery and thoracic surgeons. He was born in Bulson, India, of missionary parents, is fluent in Hindi (a fact well known to those attending the discussions of scientific papers), and was a graduate of Yale Medical School (where he was able to pay his expenses by illustrating the famous book on child development of Gesell and Amatrade) and an intern in Surgery at Yale-New Haven Hospital. Immediately after his 6 months as a surgical intern, he was called to active duty in the US Navy, and 5 months later he found himself on an LST, receiving casualties a few hundred yards off the coast of Normandy on D-Day, June 5, 1944. Ralph won the war and returned to New Haven to be a Winchester resident in thoracic surgery. He moved to Albany in 1952, where he has spent his entire professional career.

Ralph has been intimately involved in the origins of The Society of Thoracic Surgeons and was named to the Council at the Founding Members meeting October 31, 1963. He was the Secretary of the STS during the formative years, has served on the American Board of Thoracic Surgery, has been a Regent of the American College of Surgeons, and was elected President of the STS in 1975. He was recognized by the STS for his achievements by receiving in 1977 one of only six distinguished service awards given by The Society.

This lecture is a tribute to Dr Ralph D. Alley, raconteur, wit, spell-binding story teller, and friend of thoracic surgeons and thoracic surgery.

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The Future of American Thoracic Surgery

Harry Schwartz, PhD

Columnist for *The American Medical News*

Introduction of Dr Schwartz by President W. Gerald Rainer

This is an historic moment, with the first Ralph D. Alley lectureship. We are extremely pleased to have Dr Alley with us with his wife, two of his daughters, and Dave Sheridan, one of his close friends. The selection of the speaker to deliver this first Ralph Alley Lecture as one of the requirements Dr Replogle just mentioned was not very difficult indeed. I have long been an admirer of Harry Schwartz. Dr Schwartz obtained his PhD from Columbia University. He is Distinguished Professor Emeritus of State University of New York, and he has been notably prominent in his editorials and writings of many articles in such publications as the Wall Street Journal, New York Times, *and* USA Today; *where we see them most commonly, I think, is in* The American Medical News. *I have had the opportunity to hear Dr Schwartz many times over the last few years and have always been impressed with the different perspective that he brings to our profession. His talks always contain provocative thoughts of some of the ways that we should approach our future. Dr Schwartz has chosen as his topic today, "The Future of American Thoracic Surgery." Dr Schwartz, we are delighted to have you.*

Dr Rainer, Dr Alley and your family, ladies and gentlemen, it is obviously a very high privilege and honor for me to give the first Ralph Alley lecture, and I hope I live up to the responsibilities that are concurrent with that honor.

Let me begin by doing something that is very unfashionable. I want to say a word or two in praise of American medicine. Now, all of you know that American medicine when it is discussed is usually criticized. It is too expensive, millions of people do not have health insurance, etc, etc, etc. Yet, it always seems to be overlooked that American medicine today is more effective than ever in history, is doing more good things for more Americans than ever before in history, and is responsible both for many of the good things in the United States and many of the problems.

And let me just very briefly point out that first of all, the thing American medicine is usually most jumped on about is that supposedly our infant mortality in this country is a disgrace. You have all heard that; you have all read it. But, if you bother to look at the data, you will see that our infant mortality in this country has declined steadily year after year after year, at least for the 30 years I have gone back, so that every year it is the lowest in history. At the moment it looks as though for 1991, our infant mortality will be at the level of about 9.5 deaths per thousand births, which means that significantly less than one baby in a hundred in the United States will die. I might add that when I was born infant mortality was 90 deaths per thousand live births. Just in my lifetime—and I am neither the youngest nor the oldest person in this room—infant mortality has gone down by 90%.

It is true of course that there are populations, notably the black population, that have a higher infant mortality. What is never discussed is the fact that there are terrible, grievous social problems in the black population that contribute a great deal to that. Two-thirds of all black children are born to unmarried mothers. A significant fraction of them are born to mothers under the age of 18. This plague of births to young girls who are unready for the burden of motherhood is a terrible social problem. Something needs to be done about it, but I cannot see that American medicine is responsible.

What I do see is that although there are increasing numbers of deaths from things like acquired immunodeficiency syndrome (AIDS)—babies dying in the first year of life because of AIDS, an ailment that was not known 10 years ago—nevertheless, our infant mortality continues to go down and down.

And just by contrast, let us look at the Soviet Union, the great land of socialism. Until recently, some people were pointing to the USSR as an example for us. But, in the Soviet Union, the infant mortality rate now is about 25 deaths per thousand births—two and a half times the American rate. But you never read about the Soviet rate in the American denunciations of what happens here.

Second, let's look at the length of life as measured by the life expectation of a baby born this year. This measure is a trick of the statisticians. What they are really talking about when they talk about the life expectancy of a baby born this year is the average length of life of the people who die this year. The length of life of Americans has been increasing steadily again. And that is a source of a lot of our problems, because now with the number of people over 80 and over 90 and over 100 years of age increasing, they bring costs with them.

Presented at the Twenty-seventh Annual Meeting of The Society of Thoracic Surgeons, San Francisco, CA, Feb 18–20, 1991.

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And I was told yesterday by one thoracic surgeon that thoracic surgery on a man or woman who is 80 years old is by no means unheard of—in some places, apparently fairly routine. You can see, if that man or woman had not lived to be 80, he or she would not have to be operated on. So, success—the success of medicine—creates problems and costs, but I think we would all rather have the problems.

Let me add again that there is one group of Americans for whom the average length of life has not been increasing and has actually decreased, and that is black males, which obviously is a serious problem. Why has the life expectancy of black males been declining? By far the largest single reason is homicides. It is the fact of many young black males murdering other young black males. And once again, we have a sign of the really terrible social, economic, psychological morass in which that portion of our black population, which is really a third-world population, now lives.

Obviously it is something we need to do something about, but there is nothing there that doctors are going to be able to solve. Doctors are not going to be able to solve the fact that in virtually every black ghetto of the United States, drugs are rampant, wars between drug gangs are rampant, and young black males kill each other wholesale in the fight for better places to sell drugs. Those of you who live in Washington, DC, or in New York or Los Angeles know what I am talking about. The remarkable fact that I would point out, however, is that despite these terrible problems, for the nation as a whole, medicine continues to give longer, healthier, and better quality life for the great majority of our people. And it is something we ought to be proud of, but that again is usually not discussed.

Now, let me return to my topic. If we are going to talk about the future of thoracic surgery, let me get two easy predictions out of the way, then go to the hard part perhaps. One prediction is that the meeting of the STS in the year 2001 will have many more women and many more black, Hispanic, and Asian thoracic surgeons than are present now. I know that there are women, black, Hispanic, and Asian thoracic surgeons present here today, but there will be many more of you, simply because the demographic trends in the United States are such that the present minority elements of our population are increasing in number. There are an increasing number of them who go to college, who go to medical school, and now go on to specialize. So that is an easy prediction to make.

The second prediction to make is a more sorrowful one. My expectation is that in the year 2001, a patient entering a doctor's office will see prominently displayed the doctor's latest monthly human immunodeficiency virus (HIV) examination test result to certify that the doctor does not have HIV. Particularly, if it is going to be an operation, the doctor's first question to the patient is likely to be, "May I see your latest HIV examination result?" And I suppose that one form of competition among doctors will be competition among doctors who advertise that they display their weekly, maybe their daily HIV result. And we know that it takes a while between receiving the HIV in your blood stream and the time it manifests itself.

All I am really saying here is that I unfortunately suspect that AIDS is going to proliferate, to continue to expand, that we will not have a means of either preventing it or curing it within the next decade, and that it is going to have a tremendous impact upon medical practice. And I am sure that all of you have read about the furor in the wake of the patients of this Florida dentist who apparently had AIDS transmitted to them because this dentist was dying of AIDS and did not inform his patients.

Let me go, however, to a more general mode—I am an economist, and the reason that an economist worries about this sort of thing is that economists above all are students of scarcity. Scarcity is born of two facts: First, all human resources are limited, are finite. Second, human wants tend to be infinite. Unfortunately, the reality that all human resources are finite is one that is denied every day.

The most interesting example of that denial, and one that is very apt considering where we are and the time we meet, is the whole question of water for California. Those of you who are familiar with California history have known that California has, until very recently, worked on the assumption that water was an infinite resource, which could always be increased to become available. And so, for many years, California agriculture was based on the assumption that water could be supplied at a very, very low cost, virtually free of charge. And all of you have either been to Palm Springs or have seen television reports of golf classics at Palm Springs and have seen how beautifully green the greens are in the Palm Springs golf clubs; that is absolutely pure desert.

All of a sudden, the state of California has realized in the last few weeks that water is a finite resource, moreover, a finite resource that shows signs of running out. There has not been much rain in California now for 4 or 5 years. And so now you have all kinds of revolutionary moves being taken such as the declaration that different water authorities are cutting their water allocations to agriculture by 75% or 80% or 50%. But it has taken 5 years of drought for the state of California to face the reality that it is running out of water.

Now, it is this assumption that resources are infinite that guides American policy in many ways and, as we will see in a moment, has guided American policy in the field of health care as well. And, sooner or later, we are going to have to pay a price equivalent to that which the farmers in California are now paying. But let me say that there are two basic laws of health care. If you get nothing else out of this lecture, if you are just aware of these two laws and apply them in considering problems of health care policy, you will have made an advance.

The first one was enunciated by an English politician named V. Enoch Powell, after he had spent 3 years as head of the National Health Service in England. After he left the post, he sat down and wrote a little book which stated what I call Powell's law. What it simply says is this: The potential demand for free medical care is infinite.

And, once you realize that, you begin to realize that whenever anybody approaches trying to supply free medical care to a large population that law will throw in.

This is evident even here in the United States, where without national health insurance, we have given the great majority of our population very good health insurance. Characteristically people here talk mainly about the individuals who do not have health insurance. They rarely talk about the fact that 85% of the United States' population has health insurance and, usually, pretty good health insurance.

If you want the explanation for the tremendous explosion of costs in the health care field in the United States over the past 20 to 25 years, you have it essentially in Powell's law. By trying to give the great majority of Americans free or almost free medicine, we generated such a demand that the main screaming now is about the huge cost of medical care. I am old enough to remember, as I am sure many of you are, the days in which the demand for medical care was constrained because most people did not have medical insurance. Now, with Medicare, Medicaid, God knows how many private schemes, HMOs, PPOs, etc, etc, the complaint is that the nation cannot afford the present cost of health care. And, as you may know, in 1989, the American health care bill came to $600 billion, which at the going rate of increase means that for 1991, health care in the United States is going to cost nearly $700 billion, and that is quite a lot.

Now, the second law of health economics, and it is another unpleasant law that politicians particularly do not like to face, is that in health care, the ultimate economy is death. The patient who has died does not cost anything any more because autopsies are out of fashion here. But while the patient is alive, you can have more tests, more consultants, more x-rays, maybe more operations, etc. So, if you remember those two fundamental laws, you will realize that in making health care policy, we are really between a rock and a hard place. And there are no easy alternatives. These two laws apply everywhere.

Now, one more fundamental point I would like to make about the future of health care and thoracic surgeons is this: Unfortunately, there is a tremendous amount of antagonism toward physicians as a group in the United States. Now what is interesting is that if you ask people about their own doctor, they usually like their own doctor. He or she is a great person, and they are full of praise. But if you ask them about doctors as a group, they tend not to like them. And the reason they do not like them usually is, if you sort of summarize lots of answers from lots of surveys, that doctors are regarded as too rich, and, moreover, they are regarded as having gotten rich out of profiteering on human misery.

Now, mind you, that is quite different from the attitude toward the much greater earnings of football stars, baseball stars, basketball stars, or for that matter, television stars, because people in athletics or entertainment are people whom we enjoy watching. Therefore, we feel they have made a real contribution. So what if they make $5 million a year as some baseball players now do, or $19 million a year as Bill Cosby is supposed to have collected in a recent year, or whatever. They are doing God's work—they are making us feel happy.

But, if a doctor has a median income of $130,000, which is what *Medical Economics* reported last September, that is too much. Interestingly, one consequence of this feeling is that almost every day, if you read the papers carefully, you can find one or another real insult to doctors. I would just like to cite two from the very recent past. My first is from yesterday's *San Francisco Chronicle*. If you still have it, it is on the last page of the first selection and in the bottom right-hand corner article. It is a story about a speech that the commander of an American battalion in the Gulf made to his troops to prepare them for the ground battle that seems to be ahead. Let me just quote the speech. " 'You will be afraid,' the colonel told his troops. 'If you are not afraid, there is something wrong with you. Don't be afraid of fear. Rather, understand it, and grapple with it and cope with it. Like I told you before, this is not an Izod polo shirt, a Weejuns loafer crowd, not a whole lot of kids here have dads who are anesthesiologists or justices of the supreme court.' "

Interesting? A lieutenant colonel preparing his men for battle tells them, "Not a whole lot of kids here have dads who are anesthesiologists or justices of the supreme court." Then he continues, "We're the poor white middle class and the poor black kids from the block and the Hispanics from the barrio." Now, obviously, when he talked about anesthesiologists, he meant there are very few kids here whose dads are doctors. Anesthesiologists were the surrogates for doctors. Well, I have no idea how many doctors' kids are there, but I do know that there are thousands of doctors in the Gulf, including thoracic surgeons, ready to take care of soldiers who are wounded. That was not on that colonel's mind. What his mind said was that when it comes to fighting, perhaps dying, you do not find doctors' kids in the ranks. Doctors, in his mind, belong to the oppressor class.

The other example is in this morning's *New York Times*, on the op ed page. In the bottom center of the page, there is a piece about what is wrong with testing AIDS medicine. The man who wrote it, who, by the way, is a senior writer for *Business Week*, states that what is wrong with testing AIDS medicine is, in effect, that the doctors who do clinical testing of AIDS medicine have been corrupted and so they focus on testing AZT. Presumably the charge is that Burroughs Wellcome is doing the corrupting and doctors are easily corruptible.

As I say, these are just two examples, but you have to understand the weight of this really dreadful dislike, loathing of doctors. And you have to understand that there is very much involved in the making of policy. If you have been annoyed at what has happened to your fees for Medicare, just remember that many people who set those and other fees consider doctors or very large numbers of doctors as just simply crooks. This is why there is this tremendous epidemic of second opinions, precertification, all of the hassle factors that you people find in your daily practices, which I am sure infuriate you.

Now, the point of all this is simply to join these factors I have talked about with something broader that is not usually discussed, certainly not at medical meetings. And I notice that Dr Rainer in his presidential address yesterday said that one of the requirements for the leaders of tomorrow is a much wider understanding of the world, and of the economical and political factors that determine the world.

Now, let me just bring what is to my mind the most important economic factor. The most important economic factor is that to a considerable extent, the United States government is bankrupt. Now, it does not look that way. We have all been living very nicely here in San Francisco. There is still a lot of prosperity here, but what very few Americans understand is how shaky the underpinnings are of our prosperity. For example, the national debt is now in excess of $3 *trillion* —not million, not billion, but $3 trillion.

You know, every Monday afternoon the Treasury sells 3-month and 6-month Treasury bills, and the nightmare of the Treasury officials who run that is that one day, the Japanese bidders will not show up, and the interest rate for those bills may go from about 6%, which is where they are now, to who knows, 14%, 18% overnight. That is, to a very considerable extent, our destiny is now in the hands of others. These others are mainly the Japanese because they are the indispensable swing element that makes it possible for us to continue selling our bonds and our Treasury notes and our Treasury bills. And notice what is so nice, we can still sell these bonds and notes and bills denominated in American dollars. But have you noticed what has been happening to the American dollar lately? Over the past year or two the value of the American dollar has been deteriorating. The American dollar is now lower in value relative to most important currencies than it has been for many years.

I can remember that 7 or 8 years ago my wife and I went to London and we bought pounds there. A British pound was then worth one dollar and three cents. If you go to London now, you will be paying approximately two dollars for a pound, which means that vis-à-vis the British pound, the United States dollar has deteriorated about 50%. I might say that vis-à-vis the German mark and the Japanese yen, the drop has even been more extreme.

A dollar is coming to be worth less and less, and at some point, it may be that we will not be able to sell our federal debt instruments for dollars. We will have to do what second class countries have to do, that is, we may have to issue debt instruments denominated in currencies people are willing to hold, say, yens or marks.

And I might add that this $3 trillion national debt is not declining. As a matter of fact, we will probably have a record addition to the national debt this year. The general estimate now is that the debt of the United States government for the current fiscal year will increase by $400 billion. $400 billion! Now you see $400 billion, just the net increase in the national debt, is getting within some kind of hailing distance of the total medical bill of the United States.

I repeat, we have been able to live a normal life because foreigners have been willing to buy our debt and buy it denominated in American dollars. How long that will continue, I do not know, and neither does anybody else. There is another consideration. The oil companies still continue to be willing to sell oil for dollars. The world price for oil is in dollars. Well, that means that with the dollar declining in value, the real price of oil declines much more sharply. Oil is much cheaper now than it was in 1973 when the Arabs started raising the price of oil.

Well, at the moment, Saudi Arabia does not have much choice. But move yourself ahead a year or two. Iraq has been defeated. Saudi Arabia is feeling fine. And we have brought our troops back from the Gulf. And there is finally a meeting of OPEC, in which they agree, well, we do not want this depreciating American dollar, which is worth less every week. Let's announce we will only sell oil for marks or yen. What happens then?

You may tell me, well, they cannot do that to us after what we have been doing for them. You have all heard the crack, I am sure, about the current national anthem of Saudi Arabia as "Onward Christian Solders." But remember that gratitude is the expectation of future favors. It has nothing to do with the past.

So, we are in very deep economic trouble in this country. Now, politicians do not like to talk about this. Politicians get elected by promising more and more and more. What has happened to this country is that the political promises that our system is loaded with have simply exceeded our capability of ever paying for. Now there is one way to get rid of all this. It is very easy. If we have a furious inflation in this country—if your *New York Times*, which you pay 75 cents for here today at a hotel goes up to $10, which it might—the debt can be cut to manageable size. Historically, that is what countries weighed down by an unmanageable amount of debt have usually done. Inflation is the easy way to get rid of this thing. You rob your people by inflation.

Now what has all this got to do with the future of American medicine, the future of thoracic surgery? More and more, your fees and your incomes are being set by third party payers. I noticed yesterday that in the introduction to Dr Rainer, much was made of the fact that he has been fighting very hard for a new survey of what you people do and what it is worth before the people who are in charge of the resource-based relative value scale.

Well, more and more, therefore, what the government is willing to pay you is going to determine your life. And as you people know, the government has been most anxious to cut your fees. The resource-based relative value scale is essentially a scheme to cut your fees. Now the internists thought that there are going to be a scheme to cut the surgeons' fees and give the money saved to the internists and the family physicians. They are only now waking up to the fact that Congress is just interested in cutting what the government pays.

And so the internists are absolutely appalled now because the last Congress passed a law that, among other things, prohibits, beginning next year, payment of fees for what it called "routine electrocardiogram readings." The electrocardiogram is now being folded into the office visit. Well, that is really what I am getting at —the core of what I am trying to tell you—the pressure on the American government, and by extension, on American business as well, is now so enormous that the only thing that counts is cutting what they pay for medical care. And the general impression among the policy makers is that doctors have been making out like bandits.

And by the way, for most Americans, that impression will not be weakened by reading the latest issue of *Medical Economics* and the data there on earnings of various surgical specialties. Cardiovascular surgeons, according to *Medical Economics*, had a median income of almost $300,000 last year. What they call thoracic surgeons—although how they separate them from cardiovascular surgeons is not entirely clear—were the only group among the surgeons whose income actually went down in 1989. It went down to $188,000 from $196,000.

For the ordinary American, the most meaningful data are net practice earnings: 21% of cardiovascular surgeons earned $600,000 or more in 1989. What they call thoracic surgeons had only 6% in that category. There were not enough general surgeons making more than $600,000 for *Medical Economics* to give any figure. The highest figure for which they given anything is that there were 1% of general surgeons who made between $400,000 and $450,000.

So, from the point of view of the legislators and the people who work for insurance companies, doctors generally and surgeons more particularly are an overpaid group. Moreover, there is a widespread impression that much of what you people do is unnecessary. The media has been exceedingly successful in spreading that notion. So the number one imperative before medical policy makers—government medical policy makers—is how to cut the bill. And of course, it is ironic because Dr Gail Wilensky, who is the head of the Health Care Financing Administration, is herself the wife of a surgeon, but a plastic surgeon. I guess a good many procedures of plastic surgeons are paid for by people out of pocket. They are not covered by insurance.

Now, everything possible is going to be done to compress your earnings. You know, of course, about the first experiment in selective contracting in a number of areas in the United States. The Health Care Financing Administration has just contracted with institutions to do all the cardiovascular surgery on Medicare patients in that area, and the assumption is that they will get a much lower fee per operation than they would if they left it to the free market. And of course, if we have major inflation—and I repeat that we have, I would say, a 40% or 50% chance of major rampant inflation within the next half decade—then it becomes very easy. Prices go up 20%, 30%, or 40% a year, and they do not raise your rates.

It is amazing how quickly you people or people in your position can get cut down to size, so I would say that the outlook—the financial outlook for your specialty and for all surgical specialties—is not good. You are now at war with the payers, all of whom have one item on their agenda, namely, how to keep you (and not just you, the hospitals also, the pharmaceutical makers, and so on) in check. What can you do about it? Given the enormous pressures on the government policy makers, I do not know that there is really a great deal you can do.

Obviously you should try. I think the most important element you ought to try is better communications with your patients. Does anybody here suppose that more than one in a hundred or more than one in a thousand patients out there knows about Medicare's soon to be inaugurated volume controls in which the more procedures you do, the less you are going to be paid?

I would say the great sin of American medicine, the great failing of American medicine, is that American medicine has not managed to enlist its patients in defense of their rights. If you people are stopped from doing more than, say, 200,000 cardiac bypass operations a year, you may suffer, but the people who needed those operations and who will not get them because of this cap will be hurt even more.

Now, American medicine's big failure has been in failing to communicate with the patients. And by contrast, let's look at Great Britain, where the British Medical Association has communicated with patients. The British government is planning this year to revolutionize the British National Health Service and turn it in effect into one great big HMO—as one of my friends there said, make Kaiser Permanente the entire National Health Service. The British Medical Association did not like that. Because the British Medical Association did communicate with British patients, they made the Thatcher reform so unpopular that now there is a new Prime Minister, Mr Major. He has made it plain to his associates that come April 1, when the new scheme is intended to go into effect, he wants as little change as possible. So the British Medical Association has shown that by cooperation with patients, one can have an effect.

But, on the other hand, obviously the scientific progress in your field is going to continue. You are going to be able to do much more for your patients 10 years from now than you can do today. The only question is, "Will you be allowed to?" I suppose the kernel of my message is that you cannot assume that in the year 2001, thoracic surgeons or cardiovascular surgeons or any other kind of surgeons will be allowed to do everything to their patients that they are scientifically able to do. As a potential patient, I regret that.

The Thoracic Surgery Directors Association (TSDA) Resident Research Award, sponsored by Medtronic, Inc, was established in 1990 to encourage resident research in cardiothoracic surgery. Abstracts submitted to The Society of Thoracic Surgeons (STS) Program Committee representing research performed by residents were forwarded to the TSDA to be considered for this award. The abstracts were reviewed by the TSDA Executive Committee consisting of: William A. Gay, Jr, President; Martin F. McKneally, President-Elect; Gordon F. Murray, Secretary/Treasurer; Stanton P. Nolan, Councillor-at-Large; and Bruce A. Reitz, Councillor-at-Large.

The first TSDA Resident Research Award was given to Dr Todd L. Demmy, resident in training at Allegheny General Hospital, Pittsburgh, Pennsylvania, who received a sum of $2,500 and had his expenses paid to the STS meeting.

The TSDA, with support by Medtronic, Inc, will make this award annually, using the above selection procedure. The resident author of the selected study will be recognized at the STS meeting.

Resuscitation of Injured Myocardium With Adenosine and Biventricular Assist

Todd L. Demmy, MD, James A. Magovern, MD, Race L. Kao, PhD, and George J. Magovern, MD

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Recovery of energy metabolism and contractility in stunned myocardium requires several days, even when mechanical circulatory support is employed. This double-blind study was undertaken to determine if myocardial recovery could be accelerated by intracoronary infusion of adenosine during reperfusion. Ten mongrel dogs were subjected to 45 minutes of global normothermic ischemia while on biventricular support with centrifugal pumps. During initial reperfusion, 20 minutes later, and at hourly intervals for 4 hours, dogs received 100 mL/min of unaltered blood or blood enriched with adenosine (0.2 mmol/L) into the coronary arteries for 5 minutes. Circulatory support was discontinued after 4 hours or sooner if the first time derivative of left ventricular pressure exceeded 2,000 mm Hg/s. Animals that received adenosine were weaned sooner (72 ± 27 versus 216 ± 54 minutes) and had higher systolic pressure (110 ± 21 versus 57 ± 36 mm Hg), lower left ventricular end-diastolic pressure (23.8 ± 4.8 versus 34.0 ± 7.2 mm Hg), and higher first time derivative of left ventricular pressure (3,407 ± 812 versus 1,510 ± 1376 mm Hg/s) than controls at the completion of the experiment ($p < 0.05$). Final myocardium adenosine triphosphate levels were higher in the adenosine group (20.0 ± 3.6 versus 14.2 ± 4.0 μmol/g protein; $p < 0.05$). Determination of infusion and coronary sinus blood concentrations demonstrated a 90% uptake of adenosine. All adenosine animals survived, but 2 of 5 control animals died within 1 hour of weaning. Reperfusion with adenosine-enriched blood accelerated recovery of ischemic myocardium and should be considered for patients requiring mechanical circulatory support after a heart operation.

(Ann Thorac Surg 1991;52:1044–51)

Low cardiac output can occur after a heart operation or other causes of reversible myocardial injury. Death in many instances can be avoided by supporting the stunned myocardium with inotropic drugs, intraaortic balloon counterpulsation, or mechanical circulatory assist until hemodynamic function returns. Unfortunately, up to a week of support may be necessary during which time renal, pulmonary, neurologic, or infectious complications can develop that limit survival [1]. Shortening the time for hemodynamic recovery of injured myocardium may prevent such complications and thereby improve the prognosis for this group of patients.

Adenosine-enriched cardioplegic solutions have been

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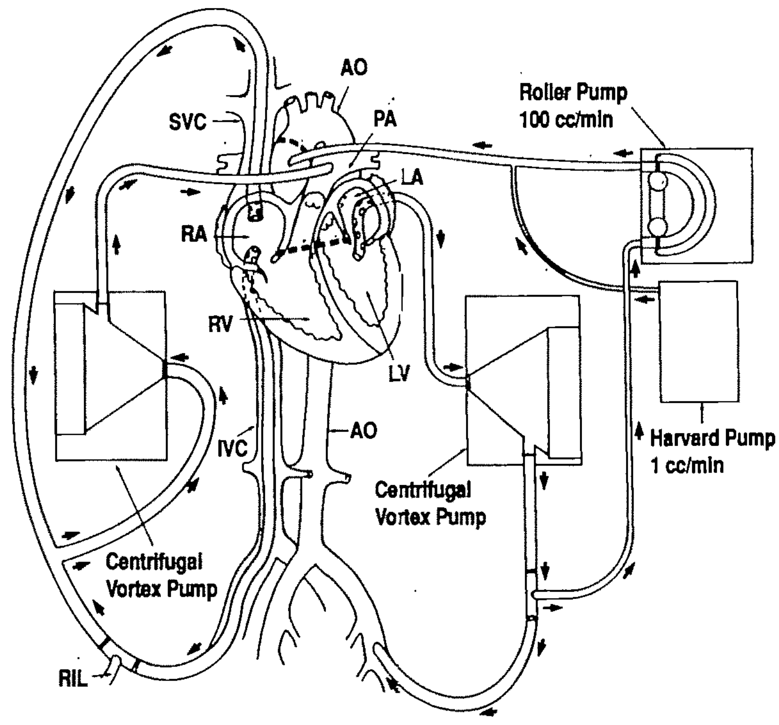


Fig 1. Experimental model of biventricular cardiac assist. Heart is placed on biventricular assist by cannulation of femoral artery, left atrial appendage, jugular and femoral veins, and main pulmonary artery. (AO = *aorta;* IVC = *inferior vena cava;* LA = *left atrium;* LV = *left ventricle;* PA = *pulmonary artery;* RA = *right atrium;* RIL = *rapid infusion line;* RV = *right ventricle;* SVC = *superior vena cava;* IVC = *inferior vena cava;* heavy dashed lines = *sites of cross-clamping;* → = *direction of flow.)*

shown to provide excellent myocardial preservation [2–8]. Preliminary studies from our laboratory have suggested that reperfusion solutions containing adenosine may also accelerate the recovery of stunned myocardium [9]. This study was undertaken to determine the effect of intracoronary adenosine administration after global normothermic ischemia in a canine model.

Material and Methods

Male mongrel dogs weighing 25 to 30 kg were anesthetized with 1% inhaled enflurane after induction with intravenous sodium thiopental (25 mg/kg) and pancuronium bromide (0.1 mg/kg). The animals were positioned in a right lateral decubitus position. The right common femoral vein, left common femoral artery, and left internal jugular vein were exposed for later cannulation. An 18-gauge plastic catheter was inserted into the right femoral artery and a 7F pulmonary artery catheter was introduced through the left common femoral vein. A left anterolateral thoracotomy was performed through the fifth intercostal space. A Tru-Cut biopsy needle (Travenol Labs, Deerfield, IL) was used to obtain a baseline full-thickness left ventricular specimen for measurement of myocardial high-energy phosphates. A second catheter was inserted through the cardiac apex to measure left ventricular pressure. Hemodynamic variables including aortic pressures, pulmonary artery pressures, cardiac output, intraventricular developed pressure (peak systolic minus end-diastolic pressure), and its first derivative (maximal positive dP/dt) were measured using an HP 8890B system (Hewlett-Packard Company, Andover, MA).

One thousand units of heparin were given intravenously before initiation of left-sided assistance. Blood was drained from the left heart with a modified 26F two-stage cannula (inserted through the left atrial appendage) and returned to the left common femoral artery through a 16F arterial cannula. The two-stage cannula was modified by excising most of the distal stage and adding additional side holes. Positioning of the cannula across the mitral valve resulted in excellent drainage with consistent left ventricular decompression. Right-sided venous drainage was achieved using two 20F cannulas, one inserted from the left jugular and advanced into the superior vena cava and one inserted from the right femoral vein and advanced into the inferior vena cava. Blood was returned to the pulmonary artery through a 20F arterial cannula. Two Bio-Medicus (Eden Prairie, MN) Bio-pumps (BP-80, see Fig 1) were used for the biventricular assist (BVA).

The assist circuits were modified by inserting a ⅜-inch–⅜-inch connector, which contained a Luer-lock port, into the tubing. On the right side this port was used for infusion of blood and crystalloid solutions. On the left side, the port supplied oxygenated blood from the circuit to serve as the carrier fluid for infusion of the adenosine or control solution after the ischemic period.

Ischemia was caused by occluding the ascending aorta with a vascular clamp. To prevent any blood not captured by the left heart drainage from entering the coronary arteries, a second cross-clamp was passed through the transverse sinus and applied across both ventricular outflow tracts below the aortic valve. A rapid mechanical cardiac arrest consistently ensued in less than 5 minutes. After the heart was arrested, a 22G catheter was introduced into the coronary sinus through a coronary vein. Myocardial temperature was maintained between 36° and

37°C by irrigation of the pericardial cavity with warm saline solution.

After 45 minutes of ischemia, the clamp across the ventricular outflow tract was removed, but the ascending aortic clamp remained in place. Left ventricular biopsy specimens were obtained. Arterial blood was drained from the left assist circuit and infused at a rate of 100 mL/min into the aortic root through a 12-gauge aortic root catheter. A Harvard infusion pump was used to infuse 1 mL/min of either the adenosine (20 mmol/L) or the control solution (0.9% saline solution) into the roller pump circuit. Infusing 1 mL/min of 20 mmol/L adenosine into 100 mL/min of blood resulted in a 0.2 mmol/L adenosine solution, which was infused directly into the coronary circulation for 5 minutes. During the infusion period, blood samples were obtained from the infusate and the coronary sinus to measure levels of adenosine and its metabolites. After the 5 minutes of reperfusion, the ascending aortic clamp was removed and the roller pump circuit was flushed and stopped. Hemodynamic readings were then made with the assist pumps at minimal flow (<1 L/min). The cycle of myocardial biopsy, ascending aortic occlusion, and 5 minutes of aortic root perfusion was repeated at 20, 60, 120, 180, and 240 minutes after ischemia.

No inotropic drugs were used during the experiment except small doses of metaraminol to support blood pressure. Lidocaine (2 mg/kg bolus) was given if ventricular ectopy developed, and injections of furosemide (1 mg/kg) were given if urine output was less than 0.5 mL $\cdot$ kg^{-1} $\cdot$ min^{-1}. All animals received a continuous infusion of nitroglycerin (16 mg/h) and mannitol (50 mL/h of 10% mannitol) to lower pulmonary vascular resistance and maintain steady urine output. Lactated Ringer's solution and 6% hetastarch were used for volume expansion. Blood transfusion was no more than 1,000 mL in any animal during the experiment. An attempt at weaning was made after 4 hours of BVA or when the dP/dt exceeded 2,000 mm Hg/s. Animals that could not be weaned after 4 hours were killed, and animals that could be weaned were killed 1 hour after decannulation. Final hemodynamic readings were made just before the animals were killed. The final readings in the control group animals that could not be weaned were made on mechanical assist support. The assignment of animals to adenosine or control infusions was made randomly, and the operative team was blinded to the identity of the solution.

Myocardial glycogen and lactate concentrations were determined using standard enzymatic assays. Myocardial water content was determined using the formula: percent water = (wet weight − dry weight)/(wet weight) × 100%. Adenine nucleotides were measured by high-performance liquid chromatography techniques adapted from published procedures [10, 11]. Blood samples for measurement of adenosine and its degradative products were immediately mixed with cold perchloric acid to stop continued degradation of adenosine. The high-performance liquid chromatographic determination of the whole blood was adjusted to reflect serum concentration by using the hematocrit. Background levels of adenosine or its degradative products were also measured at each time interval in the control animals. These values were subtracted from the values obtained in the adenosine-enriched group to yield the amount of adenosine degradation and extraction. Data analysis was performed using BMDP statistical software (Berkeley, CA). The Fisher exact test, analysis of variance, and t test were used to establish statistical significance. Results were expressed as mean ± standard deviation.

All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" (NIH publication No. 85-23, revised 1985).

Results

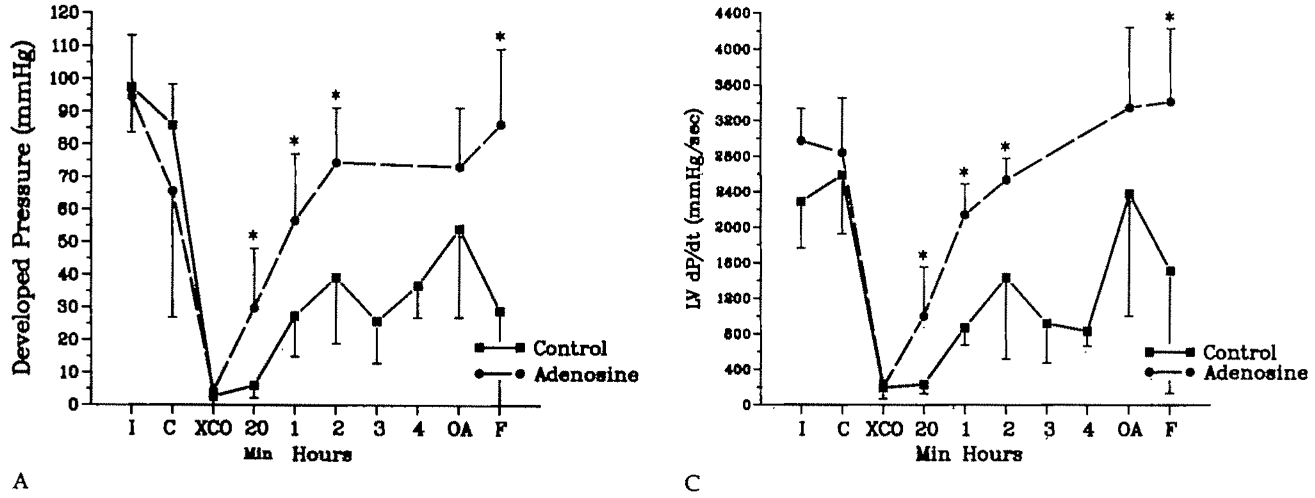
Ventricular Function

There were no differences in hemodynamic function between the control group and the adenosine group before ischemia. Flow rates for right and left cardiac assist averaged 3.3 ± 2.3 and 3.5 ± 2.5 L/min, respectively. There were no differences between the two groups in flow rate during the ischemic period or the 4-hour circulatory support period.

Animals treated with adenosine after ischemia had faster recovery of left ventricular function than the control animals. Figure 2 shows significant improvements in left ventricular developed pressure, compliance, and contractility as early as 20 minutes after the end of ischemia. This allowed a much shorter period of circulatory support (Table 1). Only 1 control dog could be weaned before the end of the 4 hours, but all of the adenosine-treated animals had weaning initiated early ($p = 0.02$, Fisher exact test). Weaning was prolonged in 1 adenosine-treated animal because of right ventricular dysfunction (Fig 3). There was 100% survival among adenosine-treated dogs, but only 60% of the control animals could survive for 1 hour after discontinuing BVA. However, the number of animals was insufficient to establish statistical significance for survival.

Even though adenosine-treated animals were supported for a shorter time, they had superior hemodynamic function after weaning from circulatory support. One hour after discontinuing BVA, animals in the adenosine group had higher left ventricular peak systolic pressure (110 ± 21 versus 57 ± 36 mm Hg), greater dP/dt (3,407 ± 812 versus 1,510 ± 1376 mm Hg/s), and lower left ventricular end-diastolic pressure (23.8 ± 4.8 versus 34.0 ± 7.2 mm Hg) than the control group (see Fig 2). The percent recovery of left ventricular stroke work index (78% ± 38% versus 48% ± 43%) and the cardiac output (4.01 ± 1.21 versus 2.39 ± 1.42 L/min) were higher in the adenosine group, but the differences did not reach statistical significance.

Right ventricular function was not different between the groups either before or after ischemia. Systemic mean arterial pressures were slightly higher in the control group in the postinfusion period (76.9 ± 14.9 versus 69.4 ± 16.7 mm Hg; $p = 0.08$) but the difference was not statistically significant.



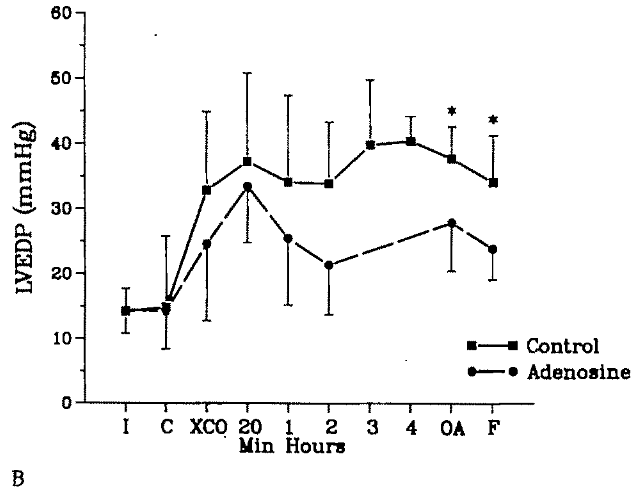


Fig 2. Recovery of ventricular function: relationship of left ventricular (LV) developed pressure, compliance (EDP, end-diastolic pressure), and contractility (dP/dt) versus time for adenosine and control groups. Developed pressure was calculated by systolic minus diastolic ventricular pressure. For each variable there was no significant difference between the groups before ischemia. Aggregate postischemic performance for the adenosine group was significantly better than control in each comparison (p < 0.01). Statistically significant differences at discrete time intervals between groups are indicated by asterisks (p < 0.05). Analysis for individual 3- and 4-hour time points was not performed because only 1 adenosine-treated animal was still on assist. By 2 hours the adenosine group had recovered function not statistically different from its starting values, whereas the control group remained statistically different from initial values (p < 0.05). (C = cannulated, assist started; F = final readings; I = initial; OA = off assist; XCO = cross-clamp off.)

Myocardial Adenine Nucleotides, Lactate, Glycogen, and Water

The results of biochemical assays favored the adenosine group. Figure 4 shows significantly greater recovery of adenosine triphosphate levels in the adenosine group after similar starting values. Adenosine triphosphate recovery correlated significantly with hemodynamic recovery (compared with data shown in Figure 2: left ventricular developed pressure, $r = 0.91$; left ventricular dP/dt, $r = 0.89$; $p < 0.05$). By the end of the experiment, lactate content was significantly lower in the adenosine-treated hearts (14.1 ± 6.8 versus 34.0 ± 19.5 μmol/g protein; $p < 0.001$) and glycogen content was similar (81 ± 24 versus 83 ± 30 μmol/g protein; p = not significant). Myocardial edema as measured by percent water content showed favorably lower values in the adenosine group as well (80.1% ± 1.9% versus 81.1% ± 1.4%; $p = 0.09$).

Table 1. Requirements for Biventricular Assist After Warm Ischemia[a]

Variable	Adenosine	Control	p Value
Met weaning criteria			
Time (min)	72 ± 27	216 ± 54	0.001
LV dP/dt (mm Hg/s)	2,221 ± 304	1,119 ± 1000	<0.05
Decannulation			
Time (min)	145 ± 52	232 ± 70	0.05

[a] Animals met weaning criteria when contractility (LV dP/dt) exceeded 2,000 mm Hg/s or when 4 hours of biventricular assist had been completed. Termination of assist (decannulation) was performed when hemodynamic function remained stable on minimal circulatory support.

Adenosine Metabolism

Coronary sinus samples taken during adenosine coronary artery infusions showed differences in the concentration of adenosine and its metabolites. Table 2 presents these values as well as the calculations for adenosine uptake and degradation. These data indicate that as the time from the cross-clamp release increased, more adenosine was removed from the coronary circulation and less was degraded. This extraction approached 18 μmol/min or 90% of the calculated infusion rate.

Comment

Adenosine is a biologically ubiquitous and highly dynamic substance [12]. Its effects on various organ systems depend on its relative concentration and the presence of

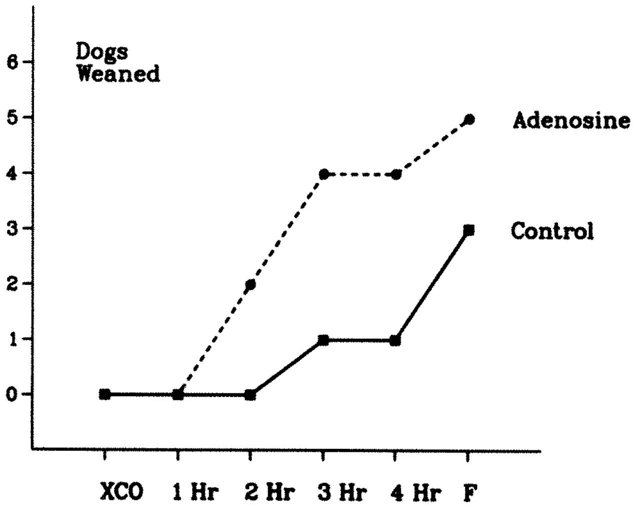


Fig 3. Aggregate number of animals from adenosine and control groups (each n = 5) that had been weaned from biventricular assist before the start of each time period. For example, 4 adenosine-treated animals were able to be weaned before 3 hours versus 1 from the control group. (F = survivors; XCO = cross-clamp off.)

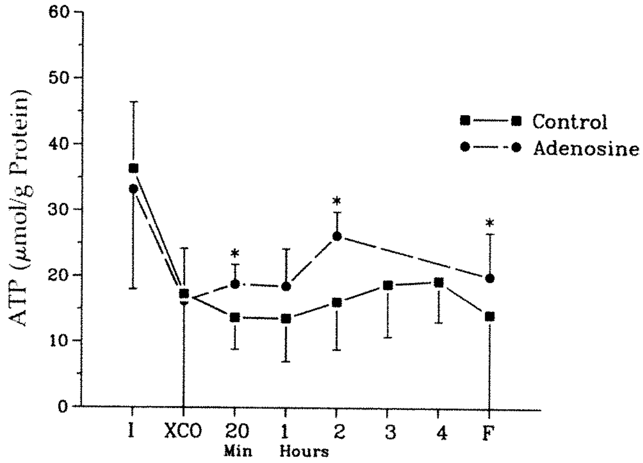


Fig 4. Recovery of adenosine triphosphate (ATP): relationship of myocardial ATP versus time for the adenosine and control groups. There was no significant difference between the groups at the start of the experiment or at the end of ischemia. Aggregate postischemic biopsy results in the adenosine group were significantly better than control ($p < 0.03$). Adenosine group samples at 3 and 4 hours were not compared ($n = 1$). Individual analysis of the 20-minute, 2-hour, and final specimens showed significant differences between the groups. Control ATP value remained less than initial values ($p < 0.05$), whereas final biopsies of adenosine-treated animals were not significantly different. (F = final biopsies; I = initial; XCO = end of 45 minutes ischemic period.)

inhibitory (A1) or stimulatory (A2) cell surface receptors. The functional state of the tissue to which it is being exposed is also important.

Administration of adenosine to healthy animals does not change ventricular performance except by lowering afterload [13, 14]. Ischemic hearts, however, may react differently because of inability to maintain adenosine triphosphate levels by phosphorylation. Adenosine triphosphate is degradated ultimately to adenosine, inosine, hypoxanthine, and xanthine, all of which can diffuse out of the cell. After reperfusion, adenosine triphosphate levels may remain low despite the presence of adequate mitochondrial phosphorylation capacity because the precursors have been lost [5]. Rapid return of intracellular adenine nucleotides and adenosine levels can be hindered by its low blood concentration (0.1 to 1 μmol/L) and short blood half-life (1 to 7 seconds) [12]. Therefore, infusing adenosine at supraphysiologic levels may increase myocyte adenosine uptake during reperfusion by simple and facilitated diffusion.

The concentration of adenosine used in this experiment was found to be optimal in our earlier work and has been reproduced elsewhere [2, 5, 9, 15]. This concentration caused profound systemic vasodilation when given intravenously, but this effect was minimal when adenosine

Table 2. Infusate and Coronary Sinus Blood Levels of Adenosine and its Metabolites[a]

	Infusate			Coronary Sinus				
	Adenosine		Control	Adenosine		Control	Calculated	
Time	ADO (1)	ADO + DP (2)	ADO + DP (3)	ADO (4)	ADO + DP (5)	ADO + DP (6)	ADO Uptake (7)	ADO Degraded (8)
X-C Off	235 ± 59	278 ± 89	2.6 ± 0.7	10.5 ± 66	731 ± 103	566 ± 513	110	126
20 Minutes	208 ± 48	254 ± 51	2.2 ± 0.7	7.9 ± 2.3	155 ± 71	60 ± 46	157	43
1 Hour	192 ± 89	237 ± 76	1.8 ± 1.2	4.5 ± 3.4	76 ± 43	18 ± 25	177	11
2 Hours	184 ± 32	208 ± 39	2.4 ± 0.5	2.3 ± 0.9	45 ± 32	10 ± 9	171	11
3 Hours	189	217	1.6 ± 0.5	2.0	41	8 ± 22	182	1
4 Hours	189	222	0.9 ± 1.0	3.4	48	6 ± 3	180	5

[a] All values represent μmol/L. Measurements were made simultaneously at midinfusion. The control values were assumed to represent the background levels of these substances so that infused adenosine utilization could be calculated. Adenosine uptake was calculated by the difference between ADO + DP in the infusate and ADO + DP in the coronary sinus after correcting for background levels [(column 2 − column 3) − (column 5 − column 6)]. Adenosine degradation during infusion was determined as the amount not undergoing uptake or passing through the coronary circulation unchanged (column 1 − column 4 − column 7).

ADO = adenosine; DP = degradation product (inosine, hypoxanthine, xanthine).

was given directly into the coronary arteries. However, intracoronary delivery causes transient, but potent depression of cardiac contractility and heart rate, which makes circulatory support desirable during infusions of this high concentration.

Biventricular assist was chosen as the form of support because of our earlier finding that it yielded survival after otherwise fatal global normothermic ischemia [16]. The present experimental model resulted in an ischemic reduction of adenosine triphosphate nearly equal to that of the model used in that earlier study. However, this study had 30% higher BVA flow and consistently good ventricular decompression, which was probably related to the use of the modified left heart cannula [16]. Good BVA prevented further injury by minimizing ventricular distention and cardiac work requirements and led to the salvage of 3 control dogs. Therefore, it is clear that BVA contributed to the postischemic recovery of the control and the adenosine-treated animals.

Adenosine delivery to myocardium during ischemia has yielded favorable results in other studies [2–8]. Its use solely in reperfusion has been reported less often. In isolated rat hearts there was increased hemodynamic recovery and decreased lactate production when adenosine was given in the reperfusate [17–19]. In dogs, coronary ligation with subsequent reperfusion without circulatory support caused 42% mortality due to refractory arrhythmias [15]. Nevertheless, surviving animals that received adenosine had reductions in infarct size of 50%, improved regional blood flow, and reduced ultrastructural disruption [15, 20–22]. There were improved hemodynamic values in the adenosine group of one study, but only left ventricular end-diastolic pressure was significantly different [15]. Finally, one clinical trial demonstrated better ventricular compliance after ischemia in patients who received dipyridamole, which is an adenosine degradation inhibitor [23].

The beneficial effect of adenosine has been explained by theories other than precursor repletion. These include decreased neutrophil adherence, decreased neutrophil superoxide production, decreased platelet aggregation, decreased oxygen demand, increased glycolysis, decreased calcium flux, and preservation of the microcirculation by vasodilation [15, 24, 25]. These proposed mechanisms have evidence to support them but were not studied in this work because of the experimental design and the relatively elaborate nature of the required assays. Moreover, it was unclear what the time course of these deleterious postischemic processes were. Many could have been ongoing and therefore less likely to be altered by brief infusions of a substance that is as rapidly metabolized as adenosine.

The method used to determine coronary adenosine uptake yielded high extraction rates similar to those of other reports [24]. Yet, it was difficult to prove that the adenosine extracted reached the myocardial cell directly. Adenosine can be metabolized rapidly extravascularly as well as intravascularly [26, 27]. It is possible that some went to nonmyocyte cells or was interstitially metabolized and released during the postinfusion phase. Nevertheless, the observed serial improvements in myocardial adenosine triphosphate level support the repletion theory. Evidence that adenosine forms in periorganelles adjacent to capillaries in ischemic hearts has been reported as well [28]. Further study may yield additional data that link adenosine uptake to myocardial recovery.

Adenosine clearly accelerated left ventricular recovery and shortened time required for BVA after a uniform global ischemic injury. This study also demonstrated trends toward improved survival, cardiac output, and other hemodynamic indices. It appears that infused adenosine is extracted from the coronary circulation and is used to increase intracellular adenine nucleotides. Therefore, further study of adenosine infusions in patients who sustain major cardiac ischemic injury appears warranted. Those patients who require mechanical circulatory support after severe myocardial ischemia form a group in which such treatment could be useful. In these very sick patients it is unlikely that adenosine could cause much harm, and based on this work, there is great potential for accelerating hemodynamic recovery.

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DISCUSSION

DR HENRY M. SPOTNITZ (New York, NY): I enjoyed your paper very much. I would like to ask you about a possible alternative explanation that you did not mention. The conditions in the aortic root during reperfusion are critical. As you pointed out, adenosine is a powerful vasodilator. Was there a difference in aortic root pressure during the reperfusion period, and could part of the difference between the two groups be explained on the basis of injury due to high perfusion pressure in the control group?

DR DEMMY: Well, the pressure was not directly measured; however, this is a model that was used in our previous experiments that showed good ventricular recovery after 24 hours of biventricular support without aortic root infusions. This control group had the same reduction of adenosine triphosphate and recovery profile as the previous studies' animals, which were not subjected to a controlled coronary flow, but rather a normal mean aortic root pressure. I also think that the relatively short time of infusion mitigates against it being an infusion pressure injury.

DR RICHARD M. ENGELMAN (Springfield, MA): I certainly enjoyed the presentation and thought it was very nicely done. I wondered, what is the dynamic degradation of adenosine when it is administered systemically? You administered this directly essentially into the coronary arteries. Could you have administered this systemically and expected it to be effective?

DR DEMMY: I think the answer is no, and there are a lot of data to support that conclusion. First, the half-life in blood is about 1 to 7 seconds. So, there has been work with peripheral infusions of adenosine (low dose) and other more stable precursors, such as ribose, which has shown some promise that it may work through other pathways. Also, there would be no way that you could achieve the concentration that was administered to these hearts systemically without causing tremendous shock from systemic vasodilation as well as temporary depression of cardiac function. That is why the biventricular assist was so critical in this experiment.

The instability of adenosine needs to be emphasized. We repeated this experiment using the Gish reservoir, which many surgeons use for their blood cardioplegia. The aortic root infusion was done immediately after mixing the adenosine with the appropriate volume of blood in this reservoir, but 4 of the 5 adenosine-treated animals died by the end of the experiment with no change in the control results. We found that extracorporeal circulation accelerated adenosine metabolism and that we were infusing mostly inosine, a negative inotrope, by the end of the experiment. It is extremely critical to give the adenosine as close to the target circulation as possible for it to be effective.

DR ENGELMAN: I think that is quite an important point, because that has been shown by a lot of people and I fully agree with it. Thank you.

DR VERDI J. DISESA (Philadelphia, PA): That was a very nicely presented study. Although it is hard to argue with 100% survival versus 60% survival, and the time from weaning from bypass was clearly shorter in your control group, the design of the experiment really determined that the animals with the slower recovery of dP/dt would be weaned from bypass later. I wonder about your selection of dP/dt and developed pressure as indices of contractility, because both of those are clearly load-dependent; had you considered using other load-independent indices of contractility that have been so well described by many members of this audience?

DR DEMMY: Well, I think one has to choose some objective criterion for weaning, and we did not use some of the more sophisticated measurements, such as sonomicrometry. When you are dealing with results that are this divergent, it probably does not matter that much what is used. Frankly, from that earlier series of animals that recovered so slowly, there was probably some experimental bias on my part not to take an animal off assist quickly; perhaps that is why there was a delay in decannulating some of the animals in the adenosine group. For those who want to reproduce this work clinically or experimentally, another measurement of contractility would probably be valid but may not be essential to getting the same good results. Also, the dP/dt can be fairly helpful if you have equal flow rates and afterload pressures, as in this study.

DR DISESA: I think if you are going to use those measures, then at least you have to give us some indication of what the filling conditions of the ventricle were. I do not recall that you presented at what end-diastolic pressure, for instance, these measurements were made.

DR DEMMY: Figure 2 shows what the end-diastolic pressures were. The control group had a much higher filling pressure and much poorer compliance than the adenosine group, and the results were statistically significant by the end of the study.

DR L. HENRY EDMUNDS (Philadelphia, PA): Basically what you are recommending is a hot shot of adenosine instead of aspartate or glutamate. Adenosine diphosphate, as I understand it, increases cyclic adenosine monophosphate and I think also guanosine monophosphate and could possibly accelerate recovery from stunning. Stunning, as I understand it, probably causes a defect in the metabolism of the high-energy phosphates rather than depletion. The mode of delivery and your choice of drug are very interesting and may in fact improve myocardial contractility during recovery after a reversible ischemic event.

DR DEMMY: Yes, I agree. I think adenosine's greatest potential benefit is bringing stunned hearts back faster. I also think that we should continue substrate enhancement studies such as those done by Buckberg to prevent stunning. In fact, Buckberg recognized adenosine as a possible reperfusate component in some of his writings.

Effect of AICD Patch Electrodes on the Diastolic Pressure–Volume Curve in Pigs

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Although the automatic implantable cardioverter defibrillator (AICD) is effective against malignant ventricular arrhythmias, the effects of AICD patches on left ventricular diastolic properties have not been defined. Accordingly, extrapericardial (group E, n = 5) or intrapericardial (group I, n = 6) AICD patches were implanted through a median sternotomy in 11 anesthetized pigs. Six weeks later, using a left thoracotomy, the hearts were arrested with hypothermic cardioplegia. A balloon catheter was inserted into the left ventricle through the aortic root, and pressure–volume curves were measured before and after sequential removal of patches and pericardium. A dense intrapericardial fibrotic reaction in group I was not present in group E. Normalized left ventricular filling volumes in group E were significantly larger at pressures of 5.1 to 10, 15.1 to 20, and 20.1 to 28 mm Hg compared with group I ($p < 0.05$). We conclude that intrapericardial AICD patches adversely affect left ventricular diastolic pressure–volume relations and recommend that AICD patches be placed in the extrapericardial location clinically whenever possible.

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The automatic implantable cardioverter defibrillator (AICD) has been used successfully in the treatment of malignant ventricular arrhythmias; however, its effects on the diastolic properties of the heart are unknown [1, 2]. Implantation of the AICD involves the insertion of patch electrodes around the heart, and both the intrapericardial and extrapericardial positions have been used. Because the silicone-coated AICD patches could generate a fibrous reaction [3, 4], these patches may cause a change in the compliance of the left ventricle and other chambers. Although the AICD has been dramatically successful in reducing the incidence of sudden death to 1% to 2% per year in patients susceptible to ventricular arrhythmias, an annual mortality rate of 10% to 15% per year from congestive heart failure has also been reported in these patients [5, 6].

Because a decrease in left ventricular (LV) compliance could increase filling pressures or adversely affect systolic function in patients already prone to heart failure, the purpose of this study was to determine what effect AICD patch location has on the ventricular pressure–volume (P-V) relationship. Specifically, the positioning of the patch in the intrapericardial versus the extrapericardial location was investigated.

Material and Methods

Domestic pigs weighing 40 to 50 kg were anesthetized with intramuscular administration of xylazine (1.5 mg/kg), ketamine hydrochloride (15 mg/kg), and atropine sulfate (1 to 2 mg) and maintained with supplemental sodium pentobarbital as needed (5 mg/kg intravenously). Animals were intubated and mechanically ventilated with ambient air. Pigs were positioned in the supine position, and the chest was shaved, prepared, and draped in a sterile fashion. A median sternotomy was performed to expose the heart.

In 5 pigs (group E), large AICD patches (L-67; Cardiac Pacemakers Inc, St. Paul, MN) with the leads removed were secured in the extrapericardial location, outside the anterior and posterolateral aspects of the pericardium. In a second group of 6 pigs (group I), the pericardium was opened, and the patches were secured in the intrapericardial position, along the anterior and posterolateral aspects of the heart. The pericardium was loosely approximated in group I. The sternotomy was closed in layers. All animals were cared for in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Six weeks after the initial procedure, the animals were reanesthetized, and a left lateral thoracotomy was performed through the fifth intercostal space to expose the heart. The animal was heparinized (5,000 IU intravenously), the aorta was cross-clamped, and the inferior vena cava was transected to decompress the right atrium and right ventricle and to prevent volume within those chambers from decreasing LV compliance. The heart was arrested with hypothermic (4°C) cardioplegia (1 L of Stanford solution, 380 mOsm/L [STERIPHARM, Hawthorne, NY], plus 30 mEq of KCl) administered into the aortic root, and the left ventricle was then emptied through a supravalvular aortotomy.

A balloon catheter was threaded retrograde past the

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aortic valve into the left ventricle. A P-23ID pressure transducer (Statham Instruments Inc, Hata Rey, Puerto Rico) was used to monitor balloon pressures, recorded and calibrated on an optical oscillograph (DR-12; Electronics for Medicine Inc., White Plains, NY), while volume (normal saline solution with methylene blue, 4°C) was infused in 10-mL increments. The transducer was adjusted to the level of the LV balloon and zeroed at zero volume after complete evacuation of the LV balloon. Each P–V curve was repeated at least twice for reproducibility. If injected fluid did not match withdrawn volume or if methylene blue–stained fluid was detected, data were not used. Pressure effects secondary to balloon distention were minimized by using an oversized balloon, the unstretched capacity of which was larger than the volume infused.

Pressure–volume data were collected first in situ in the cold, arrested heart with patches and pericardium intact. In group I, the pericardium and the patches were then sequentially removed, and P–V curves were sequentially measured. In group E, the patches were removed first and then the pericardium. Finally, in both groups the heart was excised, and the balloon catheter was removed. The aortic valve was clamped, and the left atrium was clamped just above the mitral annulus. Pressure–volume relations were then measured by direct infusion of normal saline solution with methylene blue into the left ventricle while pressures were recorded by catheters in the LV apex. The excised heart was maintained in a 4°C Stanford solution bath during measurement of P–V curves. The integrity of the system was determined by withdrawing infused fluid, less than 5% volume discrepancy being acceptable. The left ventricle was then trimmed back to the free wall and septum and weighed (XA Analytic Balance; Fisher Scientific, Springfield, NJ).

Volume data were normalized for differences in LV weight using the relation

$$\text{Vn} = \text{observed volume} \times (175 \text{ g/actual LV weight}) \qquad (1)$$

The normalized volumes (Vn) were grouped into five pressure intervals: 0 to 5, 5.1 to 10, 10.1 to 15, 15.1 to 20, and 20.1 to 28 mm Hg. Volumes were averaged and statistically compared using a paired *t* test within groups and an unpaired *t* test between groups.

Stress–strain relations were calculated by assuming spherical geometry for the left ventricle, calculating radii from the relation

$$R = (0.238V)^{0.333} \qquad (2)$$

When V is LV volume, this relation gives Ri, the endocardial radius. When V is LV volume plus LV weight (assuming specific gravity of myocardium is 1.00), this relation gives Ro, the epicardial radius. Rm, midwall radius, is calculated as Rm = 0.5 × (Ro + Ri). Lagrangian strain was defined as

$$\epsilon = (\text{Rm} - \text{Rm}')/\text{Rm}' \qquad (3)$$

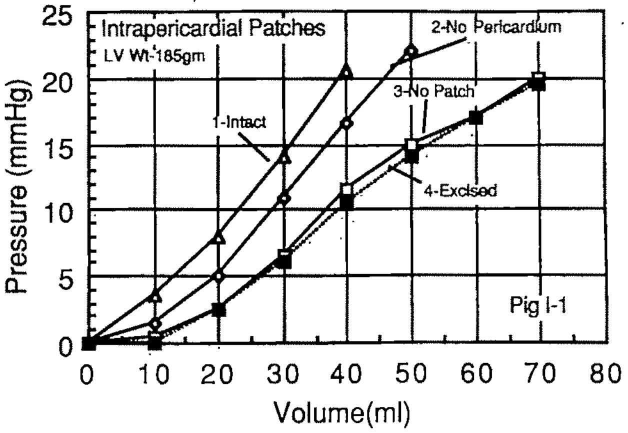

Fig 1. Representative pressure–volume curves for group I. Left ventricular (LV) pressure–volume curves shift to the right, increasing LV volume as pericardium (curve 2) and patches (curve 3) are removed. Excision of the heart has little effect (curve 4).

where ϵ is strain, Rm is the midwall radius at a given volume, and Rm′ is the midwall radius at an LV pressure of zero. Stress was calculated using the formula

$$\sigma = P \times Ri^2/(Ro^2 - Ri^2) \qquad (4)$$

where σ represents stress, and P is LV pressure in grams per square centimeter.

Elastic properties of the myocardium were described by fitting data to the following exponential equation relating stress and strain [7–9]:

$$\sigma = \alpha(e^{\beta\epsilon} - 1) \qquad (5)$$

where α and β are elastic constants, and β defines myocardial stiffness.

Curve fitting was performed using serial approximations of α and β and least squares analysis on a digital computer (Apple Macintosh IIci; Apple Computer, Cupertino, CA) with commercial software (Systat 5.0; SYSTAT, Inc, Evanston, IL). Results were averaged, and significance was determined by an unpaired *t* test. Mean stress–strain curves were generated by inserting the mean values for α and β into equation 5, calculating representative data, and fitting the results with a fifth-order polynomial.

Results

Dense fibrosis was noted about the patches, regardless of positioning. The fibrosis was intrapericardial in group I and extrapericardial in group E. The pericardial space in group E was minimally fibrosed, which was presumed to reflect postpericardiotomy syndrome.

Representative P–V curves from an animal in group I are illustrated in Figure 1. Volume introduced into the LV balloon is plotted on the horizontal axis, and resulting pressure is on the vertical axis. Curves 1 through 3 show increased LV balloon volume at a given ventricular pressure as the pericardium and patches are removed. No difference is noted between curve 3, data obtained using

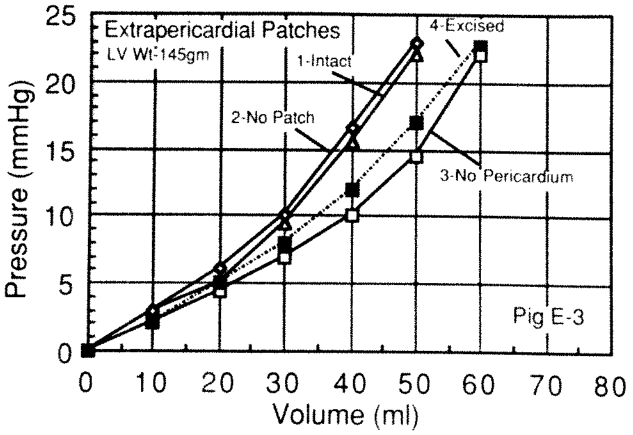

Fig 2. Representative pressure–volume curves for group E. Left ventricular (LV) pressure–volume curves shift to the right when the pericardium is removed (curve 3). Removal of the patches (curve 2) and excision of the heart (curve 4) have little effect.

an LV balloon, and curve 4, data measured using direct volume infusion.

Representative P–V curves from an animal in group E are illustrated in Figure 2. Curve 1, with the patches and pericardium intact, is similar to curve 2, data obtained after removal of the patches from the pericardium. Curve 3, measured after removal of the pericardium, demonstrates an increase in LV balloon volume from 45 to 55 mL at 20 mm Hg. Curve 4, obtained in the excised heart, demonstrates little effect of the removal of the balloon catheter.

Mean P–V data with the patches and pericardium intact for group I versus group E are presented in Figure 3 and Table 1. Left ventricular volume in group E was significantly larger in the 5.1 to 10, 15.1 to 20, and 20.1 to 28 mm Hg pressure intervals compared with group I ($p < 0.05$). Table 1 also includes LV weight, averaging 175 ± 42 (± standard deviation) for the series; this was the weight to which all LV volumes were normalized.

Figure 4 illustrates results of stress–strain analysis derived from data in Table 1, with patches and pericardium intact. Curves derived using equation 5 yielded correlation coefficients of r^2 greater than or equal to 0.99 for group I and r^2 greater than or equal to 0.98 for group E. Mean values for α were 0.043 ± 0.005 for group I and 0.031 ± 0.013 for group E (not significant). Mean values for β were 8.47 ± 0.27 for group I and 8.54 ± 0.42 for group E (not significant).

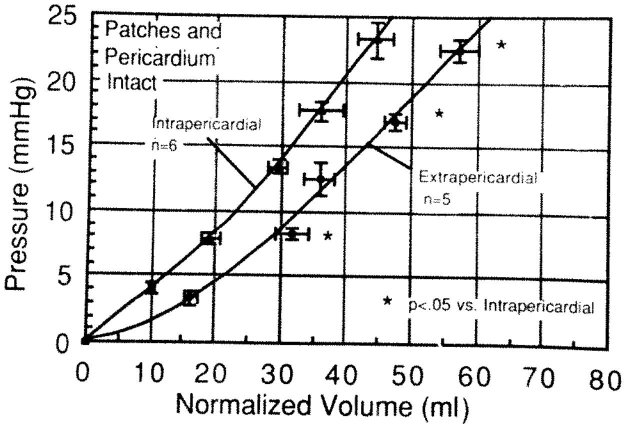

Fig 3. Mean left ventricular pressure–volume data for groups E and I with patches and pericardium intact.

Figure 5 illustrates P–V data for curves 1 and 3 in group E. Left ventricular volume was significantly increased in curve 3 in pressure intervals 10.1 to 15, 15.1 to 20, and 20.1 to 28 mm Hg ($p < 0.05$). Curve 3 could not be obtained in 1 of the 5 animals in group E because of balloon rupture.

Figure 6 examines the overall effect of the intraventricular balloon on P–V relations. Curves 3 and 4 were compared in all animals for which data were available irrespective of whether they were in group I or group E. There were no significant differences. Curve 4 was not obtained in 1 animal in group I and in 1 animal in group E because of fluid leaks from the left ventricle.

A comparison of mean P–V data for curve 4 (excised heart) is presented for groups I and E in Figure 7. Volumes were significantly larger in group E in pressure intervals 15.1 to 20 and 20.1 to 28 mm Hg ($p < 0.05$).

Comment

The present study was stimulated by early observations by clinical surgeons of marked fibrosis around AICD patch electrodes at reoperation. Our results demonstrate significant decreases in LV filling volume in group I at three levels of filling pressure measured by an intraventricular balloon with patches and pericardium intact (see Figure 3). When the patches and pericardium were removed, differences in volume were smaller, and significance was achieved at only two levels of filling pressure (see Figure 7). The data also show significant increases in volume related to excision of the pericardium and patches in group E (see Figure 5). Stress–strain analysis revealed no change in the ventricular stiffness constant (see Figure 4). Taken together, these results indicate a restrictive effect of the combination of patches and related fibrosis in group I, which results in decreased filling volume but no change in intrinsic myocardial stiffness. The background for this analysis is provided by previous studies of the LV P–V curve and ventricular compliance, including examination of the effect of the pericardium [7–17]. The effect of the pericardium on LV filling pressures in the arrested canine heart [15] produces small but significant decreases in LV volume over the entire range of pressures studied (0 to 20 mm Hg).

The present results do not address systolic function and do not indicate whether ventricular performance curves would be compromised as a result of the restrictive effects observed. However, the changes seen are quite similar to those observed with restrictive pericardial disease, in which compromise of systolic performance is well recognized. Furthermore, previous physiological studies have demonstrated effects of impaired ventricular filling on the

Table 1. Effect of Patch Location on Pressure–Volume Relations

Experiment No.	LV Weight^a (g)	Normalized Volumes^b				
		0–5	5.1–10	10.1–15	15.1–20	20.1–28
Group I: Intrapericardial Patches, Patches and Pericardium Intact (n = 6)						
I-1	185.0	9.5	18.9	28.4	NA	37.8
I-2	250.0	10.5	21.0	28.0	35.0	42.0
I-3	158.0	11.1	22.2	33.3	NA	44.3
I-4	137.0	NA	12.7	25.6	38.3	51.1
I-5	190.0	9.2	18.4	NA	37.7	36.9
I-6	163.0	10.7	21.5	32.2	42.9	53.7
Mean	180.5	10.2	19.1^c	29.5	35.975^c	44.3^c
SE	16.0	0.3	1.4	1.3	2.6	2.8
Group E: Extrapericardial Patches, Patches and Pericardium Intact (n = 5)						
E-1	110.0	15.9	NA	31.9	47.7	63.6
E-2	245.0	14.3	25.1	35.7	42.8	50.1
E-3	145.0	18.1	36.2	NA	48.3	60.3
E-4	175.0	20.0	30.0	40.0	NA	50.1
E-5	171.5	15.3	35.7	NA	51.0	61.3
Mean	169.3	16.7	31.75^c	35.9	47.5^c	57.08^c
SE	22.2	1.0	2.3	1.8	1.5	2.9

[a] This was measured weight of trimmed left ventricle. [b] These are volumes measured in the five pressure intervals and normalized for left ventricular weight. See text for details. [c] Significance: $p < 0.05$, group I versus group E.

LV = left ventricular; NA = no data points available in pressure interval; SE = standard error.

relation of LV systolic function to LV end-diastolic pressure.

This study also does not address whether diastolic properties in group E are normal. Our knowledge of P–V relations in isolated hearts in normal pigs suggests that P–V properties of group E are not completely normal, with small decreases in ventricular volume at any level of pressure studied. This could represent an artifact of the measuring techniques used, an effect of the minimal intrapericardial fibrosis observed in group E, or other factors. This issue cannot be resolved in the absence of a sham-operated group but is irrelevant to the question of advantages of the extrapericardial versus intrapericardial location for AICD patches. Pressure–volume data obtained in this study with the heart intact, without patches or pericardium, comparing the balloon catheter technique with direct volume infusion (see Fig 6) indicate that artifacts resulting from use of an intraventricular balloon were minimal.

Technical difficulty was introduced by the extensive fibrotic reaction caused by the AICD patches in the intrapericardial position. This dense intrapericardial fibrotic reaction was not noted in group E. The fibrosis caused difficulty in generating P–V curve 4 in 1 group I animal because trauma during removal of the patches resulted in irreversible leakage. In group E, rupture of the LV balloon also caused some loss of data.

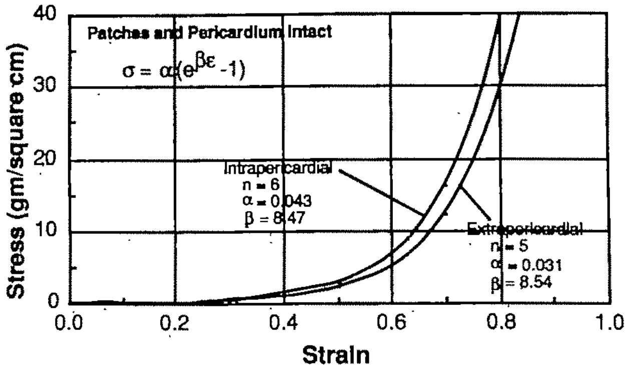

Fig 4. Effect of patch location on left ventricular stress–strain curves for groups E and I, calculated from mean values for α and β (patches and pericardium intact). See text discussion of equation 5 for details.

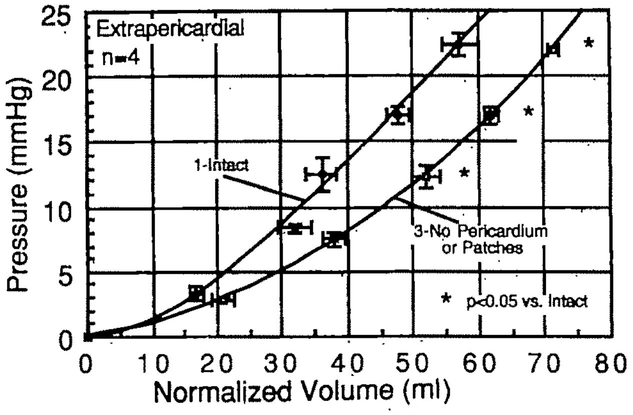

Fig 5. Mean left ventricular pressure–volume data before (curve 1) and after (curve 3) removal of patches and pericardium in group E. Volume increases when the left ventricle is freed from restraint of patches and pericardium.

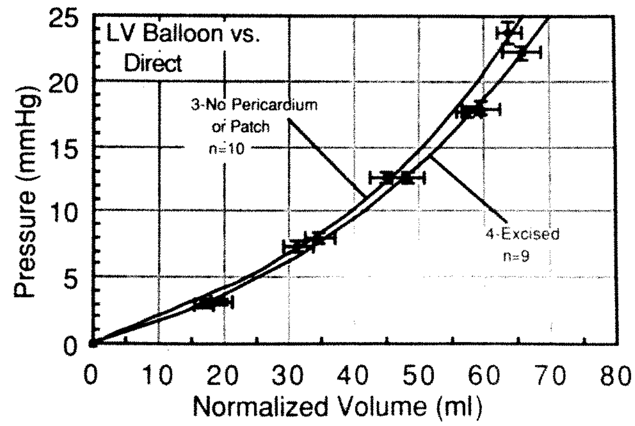

Fig 6. Averages of all available left ventricular (LV) pressure–volume data for groups E and I demonstrate no significant difference between measurement in situ obtained with an LV balloon (curve 3) and after excision of the heart obtained by direct volume infusion (curve 4).

In relation to the clinical setting, it is likely that our results exaggerate effects of intrapericardial patch electrodes on LV filling, as the pig heart is smaller than the human heart (eg, LV end-diastolic volume in pigs = 80 mL versus 125 mL in humans). The AICD patches have been inserted clinically in both the intrapericardial and extrapericardial positions [18]; several factors are involved in determining which position will most benefit the patient. Placement of the AICD patch in the intrapericardial position can result in lower acute defibrillation thresholds. In our clinical experience at Columbia–Presbyterian Medical Center, extrapericardial patches provided inadequate defibrillation thresholds in 6 of 41 patients. This was corrected by moving one or both patches inside the pericardium, which produced adequate thresholds and allowed successful AICD function. Moreover, placing patches in the extrapericardial position through a median sternotomy during a cardiac operation requires entry into at least one pleural space, which may have its own associated morbidity. In patients in highly debilitated conditions, subcostal and subxiphoid approaches, which necessitate intrapericardial patches, may be essential because of benefits related to reduced thoracotomy pain. On the other hand, the present study demonstrates that placement of the AICD patches in the extrapericardial position allows increased LV filling. This may have beneficial effects in a patient population at high risk of death due to heart failure. When other factors are equal, we believe positioning of AICD patches in the extrapericardial position is preferable.

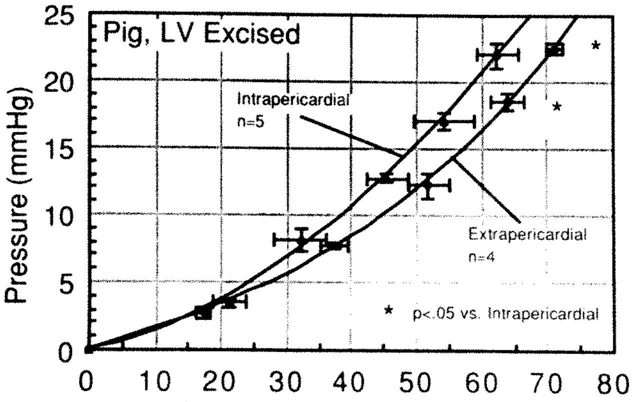

Fig 7. Mean left ventricular (LV) pressure–volume data for groups E and I after excision of patches and pericardium.

The AICD has become a familiar choice in the management of patients with malignant ventricular arrhythmias and poor LV function [19]. Previous studies [20] have shown no adverse effect of AICD insertion on intraoperative LV ejection fraction. The present study addresses the effect of AICD patch location on LV diastolic properties. The results suggest that diastolic filling may be impaired by intrapericardial patches, an observation that requires clinical confirmation. Because impaired diastolic filling could translate into LV functional abnormalities and contribute to symptoms of congestive heart failure, the surgical approach used for AICD insertion should be selected to minimize this effect whenever possible.

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DISCUSSION

DR ALEXANDER S. GEHA (Cleveland, OH): I congratulate Auteri and associates and the Columbia group for this important study. The setting is fairly artificial in that the pressure–volume relationships were examined in hearts that had been subjected to arrest and really were not in physiological shape.

A couple of points worry me. There is the effect of the induced scarring on myocardial function. The fact that the patch is implanted inside the pericardium causes concern about obliteration of anatomical landmarks should the need to operate on the coronary arteries arise in the future. Another concern is the effect of the patches on existing grafts when the patches are placed near them. Consequently, my colleagues and I have tried whenever possible to place the patches outside the pericardium if we get an acceptable defibrillation threshold. Sometimes, however, acceptable defibrillation thresholds cannot be obtained, and the patches may have to be placed in an intrapericardial position.

DR LARY A. ROBINSON (Omaha, NE): I applaud Auteri and associates for a very interesting laboratory approach to study the question of automatic implantable cardioverter-defibrillator (AICD) patch location. This is a problem that has been of clinical concern to my colleagues and myself. We are retrospectively reviewing the cases of our last 150 patients with AICD in terms of patch location and clinical result. We recently have changed our surgical technique to place all patches outside the pericardium when possible. It appears that in the perioperative period, an extrapericardial location reduces the incidence of atrial fibrillation and other arrhythmias and also decreases chest tube drainage and duration.

In the long term, I have had to perform coronary bypass grafting several years later on a few patients with intrapericardial patches and experienced considerable difficulty with obliteration of epicardial landmarks by hypertrophic scar. Other potential problems from intrapericardial patch location as demonstrated by the current research study also cause me concern and strengthen my resolve to place patches outside the pericardium. I strongly agree with the clinical observations of Dr Geha and his decision to place all patches in an extrapericardial position.

Again I commend Auteri and associates for an interesting and very important animal study about this timely clinical question.

Extended Vertical Transatrial Septal Approach to the Mitral Valve

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Optimal mitral valve operation requires adequate exposure without impairment of atrial physiology, namely sinus node and atrioventricular node function. We used an extended vertical transseptal atrial approach in 34 consecutive patients. The extended vertical transseptal approach combines two semicircular atrial incisions circumscribing the tricuspid and mitral annuli anteriorly and superiorly, allowing exposure of the mitral valve by deflecting the ventricular side using stay sutures. The right atrium is opened anteriorly along the atrioventricular sulcus. The atrial septum is incised vertically through the fossa ovalis. The right atriotomy is extended superiorly in the right coronary fossa between the right atrial appendage and the atrioventricular sulcus to meet the septal incision. The two joint incisions are extended onto the left atrial roof transversely. At this point, the two semicircular incisions are performed and joined, and mitral valve operation is performed. There were 18 women and 16 men. Five patients had ischemic mitral valve regurgitation, 18 had mitral valve prolapse, and 11 had rheumatic heart disease. The mitral valve was replaced in 17 patients and repaired in 17. There were no perioperative complications associated with the atriotomies, ie, no bleeding, no atrioventricular nodal dysfunction, and no sinus node dysfunction. The extended vertical transatrial septal approach provides good mitral valve exposure without inherent complications.

(Ann Thorac Surg 1991;52:1058–62)

Adequate exposure of the mitral valve is a critical factor of success for either replacement or repair of the mitral valve apparatus. The currently preferred approach is a left atrial incision within the right interatrial sulcus through a median sternotomy [1]. Variant approaches have been advocated to deal with complex cases, with special concern for a small left atrium or reoperations [2–7].

We have designed an approach to the mitral valve: the extended vertical transatrial septal approach. This approach was designed to include the following features: (1) routine use through a medial sternotomy; (2) absence of distortion of the mitral valve apparatus; (3) no impairment of atrial functional anatomy, namely, sinus node and atrioventricular node function; and (4) no inherent complications.

Material and Methods

Patient Population

Since December 1986, 34 consecutive patients had mitral valve operation using the extended vertical transseptal atrial approach. There were 18 women and 16 men. Their ages ranged from 26 to 79 years.

Twenty-eight patients had mitral valve regurgitation, which was associated with ischemic heart disease in 5, mitral valve prolapse in 18, and rheumatic heart disease in 5. Six patients had rheumatic mitral valve stenosis. Twelve patients had associated serious coronary artery disease, including the 5 patients with ischemic mitral valve regurgitation.

Twenty-four patients had a dilated left atrium on preoperative echocardiographic study (left atrial diameter greater than 40 mm), whereas 10 had normal left atrial dimension. The average left atrial diameter was 50 ± 14 mm (standard deviation) (range, 24 to 90 mm).

Twelve patients were in sinus rhythm preoperatively, whereas 20 were in permanent atrial fibrillation. Two patients had a permanent pacemaker implanted.

Surgical Technique

The heart was exposed through a median sternotomy in all patients. Cardiopulmonary bypass was attached to the patient using conventional aortic cannulation and double venous cannulation. The venous cannulas were introduced into the posterior segment of the lateral right atrial wall. Hypothermic cardiopulmonary bypass was started with rectal temperature lowered to 28°C. Tapes were snared around inferior and superior venae cavae.

The right atrium was opened longitudinally along the anterior segment of the atrioventricular fat pad (Fig 1). A pursestring suture was attached around the coronary sinus orifice. The aorta was cross-clamped, and cold blood cardioplegic solution was infused into the coronary sinus using a 16F Foley catheter [8]. The extended vertical transseptal approach to the mitral valve was completed as

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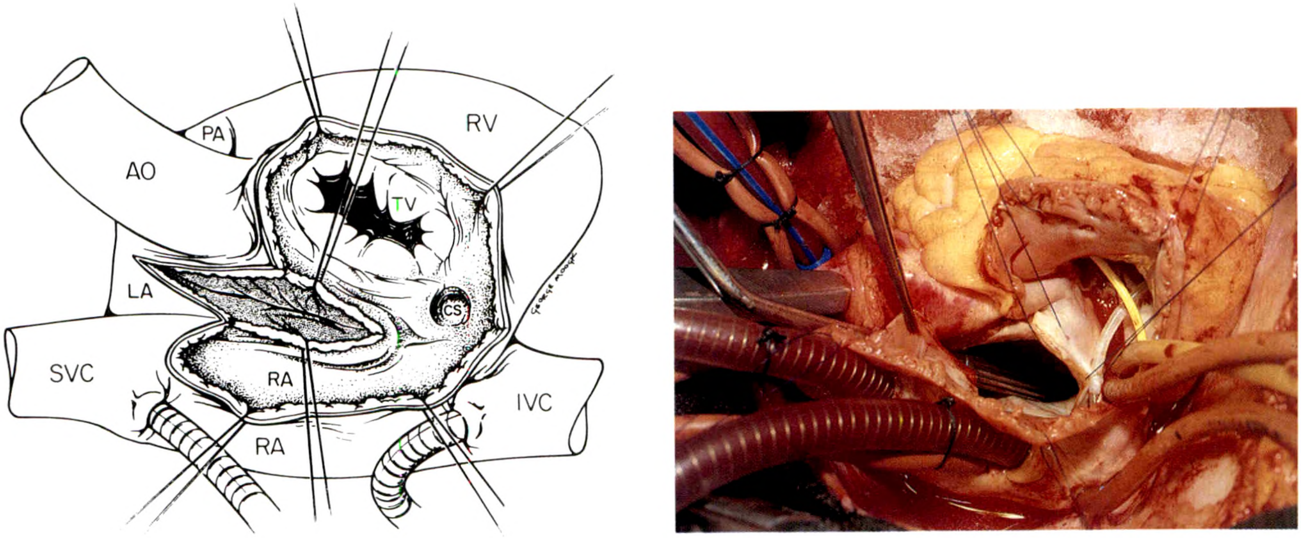


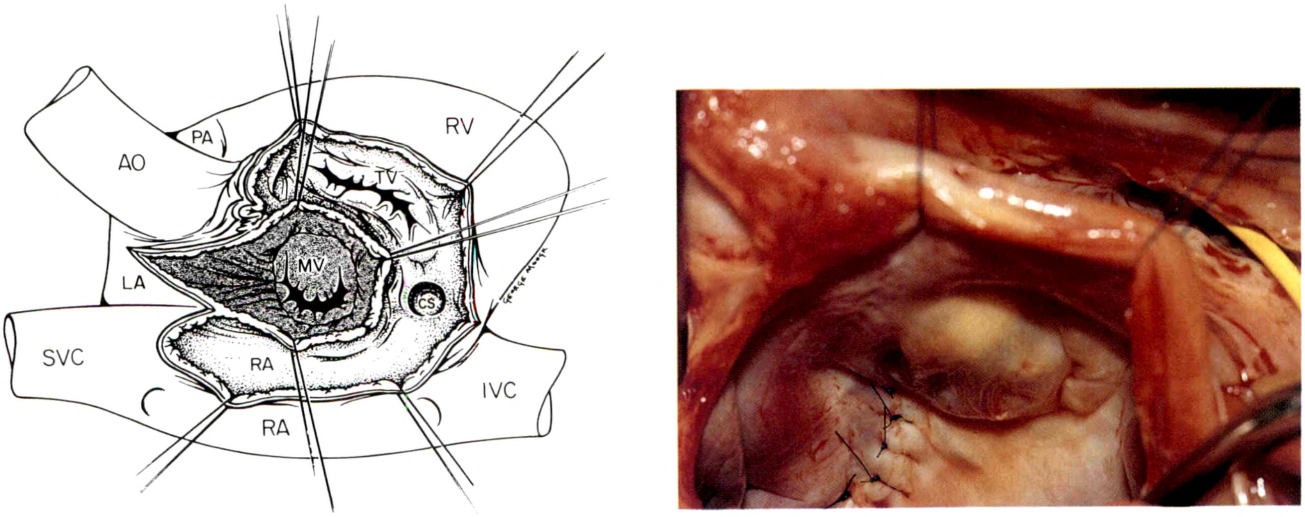
A

B

Fig 1. (A) Schematic operative view and (B) operative view of atrial incisions. The semicircumferential right atriotomy is widely opened to show the vertical septal incision extended into the left atrium. (AO = *aorta;* CS = *coronary sinus;* IVC = *inferior vena cava;* LA = *left atrium;* PA = *pulmonary artery;* RA = *right atrium;* RV = *right ventricle;* SVC = *superior vena cava;* TV = *tricuspid valve.)*

follows. The atrial septum was incised vertically through the fossa ovale. The atrial septal incision terminated inferiorly at the inferior pole of the fossa; it was prolonged superiorly by 1 to 2 cm into the atrial septum. The right atriotomy was prolonged superiorly into the right coronary fossa between the right atrial appendage and the atrioventricular sulcus to join the superior end of the septal incision. From where the two incisions met, the roof of the left atrium was incised. The left atriotomy was extended to the left over 3 to 5 cm, at a distance from the aortic root.

After completion of the atriotomies, two joint semicircular incisions were made circumscribing, at a distance, the tricuspid orifice anteriorly and superiorly and the mitral valve orifice anteriorly (septal incision) and superiorly. The exposure of the mitral valve apparatus was completed by deflecting the "ventricular" edge of the septal incision using stay sutures secured over the left



A

B

Fig 2. Similar views as depicted in Figure 1. The septal incision is deflected by stay sutures to expose the mitral valve. (MV = *mitral valve; other abbreviations as in Figure 1.)*

side of the sternotomy (Fig 2). A retractor was used for additional exposure. The mitral valve was readily exposed without dislocation or distortion.

Pathology and functional anatomy of the mitral valve were assessed, and operation on the valve could proceed using either repair or replacement. Concurrent cardiac operation was carried out before or after mitral valve operation according to the surgeon's choice.

Atriotomies were closed using 3-0 monofilament running sutures. The superior left atrial incision was closed, then the septal incision starting at the lower end upward. The two sutures were tied where they met together, and the right atrial incision was closed. The vascular clamp occluding the aorta was then removed.

Results

The mitral valve was replaced in 17 patients and repaired in 17 [9, 10]. Five patients died within 1 month postoperatively: 3 of low output failure and 2 of cerebrovascular accident.

Mean aortic cross-clamping time was 45 minutes for isolated mitral valve procedures and 60 minutes when concomitant procedures were carried out. There were no complications associated with closure of atriotomy; in particular, there was no retroaortic left atrial bleeding and no atrial septal defect. All 12 patients in sinus rhythm preoperatively remained in sinus rhythm postoperatively. There was no heart block postoperatively.

Comment

Exposure of the mitral valve apparatus has been satisfactory without the need for forceful retraction. In most cases only stay sutures were used. It was believed that mitral valve operation was made easier and shorter. Aortic cross-clamping time for mitral valve repair was 45 minutes, and there was no short-term or long-term relapse of mitral valve regurgitation.

Repair of atriotomy was not associated with bleeding or postoperative dehiscence such as an atrial septal defect. The septal segment was repaired by closing the edge of the fossa ovale, not the thin, friable membrane. There was no atrioventricular nodal dysfunction. This is consistent with the topography of the incisions, which are kept away from the atrioventricular nodal area. Conversely, the preservation of the sinus node function is more striking. The sinus node artery can be severed either by the right atriotomy in the right coronary fossa or by the left atrial incision [11]. However, blood supply through the sinus node artery does not seem necessary to sustain normal sinus node function, as suggested by experimental sinus node isolation [12], cardiac transplantation in which the recipient heart stays in sinus rhythm [13], dissection of the right coronary fossa for Wolff-Parkinson-White syndrome [14], and the superior approach to the mitral valve [6]. Recently, we have modified the technique to spare the sinus node artery arising from the right coronary artery using similar dissection as the epicardial approach to the anterior septal pathway. The fat pad in the right coronary fossa is partially mobilized to allow the right atriotomy to be carried out closer to the tricuspid annulus without involving the sinus node artery.

In conclusion, we have reported an approach to the mitral valve that is associated with adequate exposure and no inherent complications. Many approaches to the mitral valve have been described. Recently, Balasundaram and Duran [1] have reviewed the subject. They stated that their preferred approach is still the vertical left atriotomy through the interatrial groove exposed using a median sternotomy, although in selected cases, they transect the superior vena cava for adequate exposure [7]. We have used the extended vertical transseptal atrial approach routinely because it provided excellent exposure in every case, whatever the size and morphology of the heart.

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DISCUSSION

DR DELOS M. COSGROVE III (Cleveland, OH): Dr Guiraudon, what is the main impetus for doing this? Is it a problem with exposure through the standard left atrial approach?

DR GUIRAUDON: The main incentive was the shortage of residents and, of course, the need for adequate exposure. Expert assistants are required to handle a Cooley retractor, whereas the extended transatrial approach avoids the need for expert assistance.

DR TIRONE DAVID (Toronto, Ont, Canada): Why not use a self-retaining retractor? There are all kinds of self-retaining retractors available to replace the resident holding a Cooley's retractor. My main concern with your incision is the sinoatrial node artery. I am surprised that 12 patients had normal sinoatrial nodes after the operation. I have limited experience in dividing the sinoatrial node artery, but I have a number of patients in whom sick sinus syndrome developed.

I occasionally struggle to see the mitral valve to repair it, but all we do then is transect the superior vena cava a centimeter above the sinoatrial node and then carry the atriotomy toward the roof of the left atrium similar to the way you described. This approach gives excellent exposure.

DR GUIRAUDON: I have shared Dr David's concerns but had to admit, based on convincing evidence, that normal sinus node function is preserved after suppression of sinus node artery flow. Dr W. C. Sealy has reported normal function after total isolation of the sinus node. There is no report of sinus node dysfunction associated with the left superior approach to the mitral valve, although the sinus node artery should be divided in 25% to 30% of patients. I am aware of at least 300 patients who were operated on using the technique I just presented without associated sinus node dysfunction.

DR DAVID: Doesn't the sinoatrial node need blood supply?

DR GUIRAUDON: Atrial myocardial blood supply is complemented by substantial noncoronary flow, as well evidenced by the absence of atrial necrosis after heart transplantation, which abolishes coronary flow. The sinus node may exhibit normal function after heart transplantation. Atrial ischemia is a rare condition with few atrial acute infarctions reported.

I agree that the division of the superior vena cava provides an excellent exposure when combined with the classic left atrial incision. This approach is advocated by Selle, C. Duran, and many others. The extended vertical transseptal approach seems to provide identical excellent exposure with no inherent complications and without the need for vena cava repair.

DR ROBERT W. M. FRATER (Bronx, NY): I agree completely with Dr Guiraudon that this is a superb approach to the mitral valve. For about 600 or 700 cases I have used the transseptal approach from the right side of the coronary sinus back into the right superior pulmonary vein. That always leaves a bit of a ridge superiorly.

Having seen at the Video Surgery Conference in Italy last September the alternative of making a vertical septal approach into the left atrial dome, I tried it immediately. I found it the best exposure of the mitral valve without the need for more than couple of retraction sutures to create the exposure, and it has the same level of preservation, in the short term, of sinus function as I achieved with the older transseptal approach. I recommend all of you try it, extending a vertical transseptal incision to the right side of the coronary sinus so you do not get the conducting system, up between the vena cava and aorta into the dome; you will be astonished at the level of exposure. It is easy to do, it is easy to close, and it is truly without complications thus far in my experience. With Dr Guiraudon's presentation today, there are now at least four surgeons who are using it. It is clearly an idea that has suddenly hit everybody at the same time, and I think it is an idea that is on its way.

DR FREDDY VERMEULEN (Nieuwegein, the Netherlands): I support Dr Guiraudon with his approach; I learned it from him. We recently reviewed our 300 last mitral or mitral and tricuspid valve operations; in this group we used this approach at the surgeon's preference in about 100 cases. As one of the six surgeons of our group I really can support all of Dr Guiraudon's findings: no major bleeding, no rhythm disturbances, and no change thus far in patients previously in sinus rhythm.

The major advantage is the excellent exposure: I noted that our resident in training—and we have a different training program than in the States, as residents start very early in cardiothoracic surgery—really can watch how we do mitral valve operation; especially in mitral valve reconstruction it is a major advantage. At present, most of my associates are using this approach almost routinely.

There has been another paper presented at the last meeting of the European Association for Cardiothoracic Surgery by Dr Alfieri of Brescia, who reviewed his experience; he also learned this approach from Dr Guiraudon, and he supports all of the findings of those that have used it and as shown to us today by Dr Guiraudon.

DR CIRO CAMPANELLA (Edinburgh, Scotland): Did you explore, before embarking on your technique, the possibility of solving the problem of a poor approach by extending the standard longitudinal incision for routine mitral valve with a second one perpendicular to it like an inverted T? With this method, after assessing the mitral valve exposure using the standard longitudinal incision in front of the right superior pulmonary vein, if you believe the approach is inadequate you can choose to snare the cava and open perpendicularly the right atrium, the interatrial sulcus, and the interatrial septum. The exposure now is like in a book. Also, how long a cross-clamping time did your technique add to the overall length of the procedure?

DR GUIRAUDON: The average cross-clamping time was 45 minutes for mitral valve repair.

DR CAMPANELLA: I asked you because it seems to me a great deal of repair to do afterward.

DR GUIRAUDON: The closure can be done in a short time. The repair of the right atrium can be done after unclamping the aorta when the left atrium is securely closed. I know well the technique described by Charles DuBost (his name was misspelled Dubots in his original paper). Like many users of this technique, such as Doctor Piwnica sitting beside you, I found its repair difficult, frequently requiring an atrial patch to close.

DR JOACHIM LAAS (Hannover, Germany): I support Dr Guiraudon's view that the transection of the artery in the left atrial roof is not that important for maintenance of sinus rhythm. My colleagues and I have published a series of 100 mitral valves operated on consecutively using the superior approach. Thirty percent of these patients were in sinus rhythm before and after operation. So we believe that transection of the left atrial roof is not of importance with respect to sinus rhythm.

I am a little concerned about one of the intraoperative slides shown. The incision was quite close to the aortic root where the roof of the left atrium is very thin. Furthermore, in the same slide, the distance of the right coronary artery was very small. Dr Guiraudon, did you have any problems with bleeding after closing the left atrial incision, and was there any obstruction of the right coronary artery?

DR GUIRAUDON: I had no complications associated with the repair of the left atriotomy. I usually take large bites. The right coronary artery is easy to identify and spare.

DR ARMAND PIWNICA (Paris, France): We have to face more and more reoperations, second-time and third-time patients, with reconstructive operations or a bioprosthesis, and the approach described by Dr Guiraudon and associates is very important, because in some cases the left atrium becomes smaller and smaller. So we are very happy to use the transseptal approach.

DR GUIRAUDON: Thank you Dr Piwnica. When the extended vertical transseptal approach is used during a redo operation, a minimal dissection of adhesions is required to expose only the aorta and the right atrium.

REVIEW OF RECENT BOOKS

The Foundations of Cardiac Pacing, Part I. An Illustrated Practical Guide to Basic Pacing
By Richard Sutton and Ivan Bourgeois
Mount Kisco, NY, Futura, 1991
345 pp, illustrated, $65.00

Reviewed by James L. Nielsen, MD

The beneficiaries of the information in this book include not only cardiologists-in-training, but any physician who comes in contact with cardiac pacemakers on a routine basis. This is probably even more so for those dealing with pacers on an intermittent basis.

This book begins with explanations of basic cardiac electrophysiology and practical applications of clinical electrophysiology. The basic concepts and actual composition of a modern pacemaker are extensively discussed, as are pacemaker indications, selection, and implantation.

As the pacemaker patient is an ongoing concern, the authors have included chapters on extensive trouble-shooting and clinical follow-up to facilitate patient management. They also discuss the impact of pacemakers on the health care system here and abroad, and in later chapters prognosticate about the future of cardiac pacing.

Throughout the book, the text is well organized and concise, with clinical examples and clear illustrations used in a complimentary fashion. The chapters are arranged such that those readers who want to review certain topics can easily locate specific information.

As an introduction to pacemakers and their use, this well-written book is an excellent foundation.

San Antonio, Texas

Recovery of Oxygen Utilization Efficiency After Global Myocardial Ischemia

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The time required for myocardial oxygen utilization to recover from 12 minutes of warm ischemia was studied in 15 sheep. Five control animals had 42 minutes of cardiopulmonary bypass at 37°C with the heart beating and vented. In 10 experimental animals, the aorta was clamped for 12 minutes during bypass with the heart vented, and the animals were perfused 30 additional minutes after removal of the clamp. In both groups, measurements of left ventricular coronary arterial blood flow, oxygen consumption (LVO_2), peak systolic pressure, circumferential systolic stress integral (CSI), and pressure volume area (PVA) were made 30 minutes after and hourly for 5 more hours after cardiopulmonary bypass ended. Reversible ischemia significantly increases the relationship between LVO_2/PVA and LVO_2/CSI 1 hour after the clamp is released, but the relationships return to prebypass values by 2 hours. Ischemia significantly decreases end-systolic elastance, which remains depressed thereafter. Cardiopulmonary bypass without myocardial ischemia significantly decreases total ventricular mechanical energy output and oxygen utilization efficiency of the working sheep heart but does not significantly alter end-systolic elastance or the LVO_2/PVA and LVO_2/CSI relationships. Twelve minutes of warm ischemia impairs oxygen utilization efficiency in the reperfused sheep heart for more than 60 minutes but less than 120 minutes.

(Ann Thorac Surg 1991;52:1063–8)

Myocardial ischemia that does not cause necrosis ("stunned myocardium") decreases oxygen utilization efficiency and myocardial contractility [1]. The loss of contractility lasts much longer than the duration of ischemia [2]. In dogs, occlusion of the left anterior descending coronary artery for 5 minutes diminishes contractile function of the ischemic area for 6 hours [3]. The time required to regain myocardial oxygen utilization efficiency after reversible ischemia is not known.

These experiments test the hypothesis that recovery of oxygen utilization efficiency after reversible myocardial ischemia outlasts the duration of ischemia and coincides with recovery of contractile function.

Material and Methods

Thirty healthy Dorsett sheep (mean weight, 40.5 ± 6.5 kg) were studied in compliance with National Institutes of Health Publication No. 85-23 as revised in 1985. Anesthesia was induced with sodium pentothal (25 mg/kg intravenously), and the animals were intubated and mechanically ventilated during halothane anesthesia (0.5% to 1.5%). Ventilation was maintained at 10 to 20 cycles/min and 12 and 18 mL/kg with 100% oxygen (Model 607, Harvard Apparatus Co, Cambridge, MA).

Instrumentation

The heart was exposed through a clean left thoracotomy. One 4- to 6-mm flow probe (Transonic Inc, Ithaca, NY) was placed around the left main coronary artery. A 16- or 20-mm flow probe (Transonic Inc) was placed around the ascending aorta. A 6-mm hemispherical crystal (3 mHz; model H25-C; Sonotek Inc, Delmar, CA) was sutured to the base of the heart between the aorta and main pulmonary artery. A similar hemispherical crystal was placed on the apex. The anterior-posterior equatorial (minor) axis was measured with two hemispherical crystals (Model H25-C, Sonotek, Inc); the septal-free wall axis was not measured [4]. A plunge crystal (5 mHz; model 102 SL; Sonotek Inc) placed on the endocardial surface between the septum and anterior papillary muscle and one epicardial disk crystal (Model 100wt, Sonotek, Inc) placed perpendicular to the plunge crystal were used to measure wall thickness.

Micromanometer pressure transducers (model SPC-350; Millar Instruments Inc, Houston, TX) were placed in the aortic arch, superior vena cava, and the left ventricle. The hemiazygos vein was ligated and a fiberoptic oxygen saturation catheter (model P7110; Oximetrix Co, Mountainview, CA) was inserted into the coronary sinus. Pneumatic occluders (16 mm, In Vivo Metrics Inc, Healdsburg, CA) were placed around the arch of the aorta and innominate artery. The chest was partially closed.

Protocol

The animal was turned supine and the instruments were calibrated. Preischemic measurements of left ventricular

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mechanics and left ventricular oxygen consumption were obtained at different afterloads produced by partial inflation of the aortic arch occluder. With both occluders deflated, dimension, pressure, coronary blood flow, and coronary sinus oxygen saturation data were recorded for 20 seconds. During the next 5 to 8 seconds, a series of different ventricular volume–pressure loops at rapidly changing ventricular pressures produced by incremental inflations of both arch and innominate occluders were obtained for calculation of end-systolic elastance (Ees). The innominate arterial occluder was deflated, and the arch occluder was inflated to a constant volume and held for 90 to 120 seconds. Data were recorded at this constant increased afterload for 20 seconds; then the occluder was deflated. After 90 to 120 seconds data were recorded for 20 seconds followed by measurements for Ees. The arch occluder was inflated to a different volume. The sequence was continued until nine sets of data (five with the occluder deflated, four with partial inflation) and five measurements of Ees were obtained.

Animals were given 15,000 units of heparin and a two-stage venous cannula (32F; Sarns Inc, Ann Arbor, MI) was inserted into the right atrium from the right internal jugular vein. An arterial catheter (18F; USCI Inc, Glen Falls, NY) was placed in the left carotid artery. The sterile bypass circuit consisted of a venous reservoir and a 0.8-m^2 hollow fiber membrane oxygenator (Maxima) from Johnson and Johnson Cardiovascular, Anaheim, CA, and a centrifugal blood pump (model 520-c; Biomedicus Inc, Eden Prairie, MN). The circuit was primed with 1.5 L of 0.9 N saline solution.

After initiation of cardiopulmonary bypass at 37°C, the aorta was cross-clamped for 12 minutes through the left chest incision. Left main coronary arterial flow fell to zero. The left ventricle was vented through the left atrium and mitral valve during both the ischemic and reperfusion periods. The heart was reperfused on bypass for 30 minutes after the ischemic period. The heart was defibrillated, if necessary, after 3 to 5 minutes of reperfusion. Cardiopulmonary bypass was stopped, cannulas were removed and protamine (150 mg) was given. Calcium chloride was given to restore prebypass concentrations after bypass stopped; no other vasoactive drugs were given thereafter.

An identical protocol was used to record oxygen consumption and ventricular pressure–dimension data at different afterloads and measurements for calculation of Ees 1, 2, 3, 4, 5, and 6 hours after release of the aortic cross-clamp. After the last measurements, the heart was stopped with saturated KCl solution intravenously and rapidly excised. Crystal placement was verified and direct measurements were made of wall thicknesses of the anterior, posterior, and free walls, the apex, and the septum. The heart was trimmed and weighed; left ventricular volume was calibrated against sonomicrometric measurements using a latex ventricular balloon. The base wall thickness was calculated as 0.55 × anterior wall thickness [4].

Sham Controls

In 6 similarly instrumented sheep, the same protocol was followed except that the aorta was not cross-clamped and the heart did not receive any ischemic injury. Cardiopulmonary bypass was maintained for 42 minutes. A vent catheter was inserted and the heart continued to beat during bypass.

Data Analysis

Left ventricular volumes were calculated using measured longitudinal and anterior-posterior axes and an assumed septal–free wall axis dimension [4]. These calculated volumes were validated by postmortem sonomicrometry measurements of ventricular dimensions during simultaneous stepwise inflation and deflation of an intraventricular balloon. Other procedures and the time sequences to achieve steady-state conditions have been previously validated [5].

Three left ventricular dimension signals, left ventricular, aortic and right atrial pressure, aortic and left main coronary flow, coronary sinus oxygen saturation, and the electrocardiogram were simultaneously recorded on an analog chart recorder (Gould ES-1000B). Recorded data were digitally sampled every 10 milliseconds with a 12-bit, 16-channel analog-to-digital converter (Tecmar Lab Master, Solon, OH) in a Sperry IT personal computer.

Maximum Ees and ventricular volume at zero pressure were calculated from pressure–volume loops at rapidly varying afterloads using the Grossman equation as previously described. Circumferential wall stress was calculated using a prolate ellipsoid model [6–8]. Circumferential systolic stress integral (CSI) and pressure–volume area (PVA) were calculated as explained previously [1]. Because the ovine left ventricle is exclusively supplied by the left main coronary artery [9], we calculated left ventricular oxygen consumption and normalized these data for each beat per 100 g of left ventricular mass [1]. These data were related to pressure–volume area, which was divided into internal and external work, and oxygen utilization efficiency was calculated as described previously [1].

Statistics

Statistical analysis of preischemic and postischemic data was performed using commercially available software (BBN Software RS/1, Systat). Standard tests for normality and Student's t tests were employed. Correlation coefficients for the linear regression lines were also obtained with RS/1. A large-sample Z test was used for comparison of slopes.

Results

The experimental protocol was completed in 10 of 30 sheep and in 5 of 6 control sheep. Heart rate, measured as cardiac cycle length, did not vary significantly within either the control or experimental groups during the study (Table 1). One hour after ischemia, heart rate was significantly slower in postischemic sheep as compared with control animals.

Table 1. Metabolic and Hemodynamic Measurements

			After Ischemia					
Variable	Group	Before Ischemia	1 h	2 h	3 h	4 h	5 h	6 h
Cardiac cycle length (seconds ×10^{-3})	Con	594 ± 55	654 ± 59^{a}	648 ± 70.2	681 ± 79	638 ± 73	645 ± 51	663 ± 54
	Exp	545 ± 36	498 ± 19	605 ± 39	594 ± 46	592 ± 38	613 ± 38	559 ± 35
Left ventricular pressure (mm Hg)	Con	93 ± 4.0	67 ± 4.9^{c}	66 ± 2.7^{c}	65 ± 3.6^{c}	74 ± 4.9^{c}	73 ± 5.4^{c}	67 ± 3.6^{c}
	Exp	92 ± 4.4	65 ± 2.8^{c}	63 ± 2.2^{c}	62 ± 3.8^{c}	61 ± 3.5^{c}	65 ± 5.1^{c}	64 ± 5.7^{c}
Coronary artery flow (mL/min)	Con	66.3 ± 11.4	80.0 ± 16.1	80.6 ± 21.1	73.5 ± 22.1	83.6 ± 23.6	89.1 ± 23.7	79.8 ± 5.4
	Exp	76.0 ± 5.6	83.3 ± 14.1	68.6 ± 9.7	66.3 ± 9.4	75.9 ± 12.4	88.3 ± 15.9	105.7 ± 13.0
LV oxygen uptake (μL · 100 g^{-1} · beat^{-1})	Con	45.7 ± 4.4	52.3 ± 8.7	47.8 ± 8.5	48.3 ± 8.8	41.0 ± 4.6	51.7 ± 5.6	55.8 ± 3.4
	Exp	48.0 ± 6.2	50.6 ± 10.0	43.1 ± 6.5	44.8 ± 7.0	54.9 ± 10.9	69.0 ± 12.4	76.0 ± 10.4
Circumferential stress integral (mm Hg · s · 100 g^{-1} · beat^{-1})	Con	27.6 ± 1.7	17.6 ± 1.5^{b}	19.3 ± 2.0	19.8 ± 2.0	21.2 ± 2.2	22.8 ± 4.5	20.6 ± 4.5
	Exp	22.9 ± 2.8	14.3 ± 1.6^{c}	15.8 ± 1.7^{c}	16.6 ± 1.4^{b}	15.6 ± 2.1^{b}	17.1 ± 2.2^{b}	12.4 ± 2.0^{c}
Pressure–volume area (mm Hg · mL · 100 g^{-1} · beat^{-1})	Con	1778 ± 350	1093 ± 145^{b}	1065 ± 155^{b}	840 ± 181^{c}	1113 ± 114	1086 ± 150	987 ± 117
	Exp	1687 ± 208	847 ± 92^{c}	830 ± 78^{c}	739 ± 76^{c}	815 ± 132^{c}	851 ± 157^{c}	815 ± 178^{c}
Internal work (mm Hg · mL · 100 g^{-1} · beat^{-1})	Con	331 ± 102	214 ± 54	262 ± 83	263 ± 79	341 ± 95	371 ± 81	288 ± 91
	Exp	342 ± 50	157 ± 31^{c}	190 ± 33^{b}	216 ± 33^{b}	241 ± 50	259 ± 70	240 ± 64
External work (mm Hg · mL · 100 g^{-1} · beat^{-1})	Con	1446 ± 266	880 ± 108^{b}	803 ± 92^{b}	577 ± 112^{c}	772 ± 106	716 ± 183	699 ± 87
	Exp	1346 ± 170	689 ± 80^{b}	641 ± 58^{c}	523 ± 53^{c}	574 ± 102^{c}	592 ± 98^{c}	575 ± 108^{c}

For control animals, five measurements of each variable were made at each interval in 5 sheep except at 6 hours (4 sheep). Means of the five measurements were calculated and these means were averaged to determine the mean and standard deviation in the table. For the experimental animals, five measurements were also made at each interval in 10 sheep except at 5 hours (9 sheep) and 6 hours (8 sheep). The within-sheep means were averaged as in the control group to determine the tabulated data. (Con = control sheep; Exp = experimental (ischemic) sheep; LV = left ventricle ; ^{a} $p < 0.01$, unpaired Student's *t* test between groups; ^{b} $p < 0.05$ and ^{c} $p < 0.01$, paired Student's *t* test, before versus after bypass.)

After cessation of bypass, peak left ventricular pressure decreased in both groups. Peak left ventricular pressure remained constant thereafter, and the pressures between two groups did not differ (see Table 1).

Coronary arterial blood flow and left ventricular oxygen consumption did not vary significantly during the entire study in either control or experimental animals. Furthermore, measurements at each interval did not differ between control and experimental animals (see Table 1).

The integral of circumferential wall stress during systole decreased significantly after ischemia and remained depressed for the duration of the study. In the control group, CSI decreased significantly 1 hour after bypass ended but did not differ from the prebypass integral thereafter. There were no significant differences between groups at any interval.

Pressure–volume area, another index of mechanical energy production, decreased significantly after bypass in both groups. The decrease was more profound in the ischemic group, and the value in that group remained depressed for the duration of the study. The changes were largely due to changes in external work, although internal work also decreased significantly for the first 3 hours in the experimental group. Internal work did not change significantly at any time in control sheep. There were no significant differences at any interval between groups for PVA and internal or external work (see Table 1).

The slopes of left ventricular oxygen consumption (LVO_2) plotted against PVA (Fig 1) and CSI increased significantly ($p < 0.01$) 1 hour after ischemia but returned to preischemic values by 2 hours and remained unchanged thereafter (Table 2). The slopes after bypass did not change significantly from prebypass values in the control sheep. At 1 hour, the difference between groups in LVO_2/CSI slopes was significant ($p < 0.05$) and the difference between groups in LVO_2/PVA slopes nearly reached statistical significance ($p = 0.06$).

After ischemia, Ees decreased significantly in the experimental group and remained depressed for the duration of the study (see Table 2). End-systolic elastance did not decrease significantly in control animals until 3 hours after bypass.

Oxygen utilization efficiency was calculated after converting left ventricular oxygen consumption and pressure volume area to joules [10] (Table 3). Forty-two minutes of warm cardiopulmonary bypass of the beating heart significantly ($p < 0.05$) reduced oxygen utilization efficiency for 3 hours in the working sheep heart. Addition of 12 minutes of ischemia produced a similar decrease in total oxygen utilization efficiency which lasted for the duration of the study. Except for the first hour after ischemia, oxygen utilization efficiency for internal work did not change significantly; the decrease in external work was largely responsible for the significant reductions in oxygen utilization efficiency that occurred in both groups.

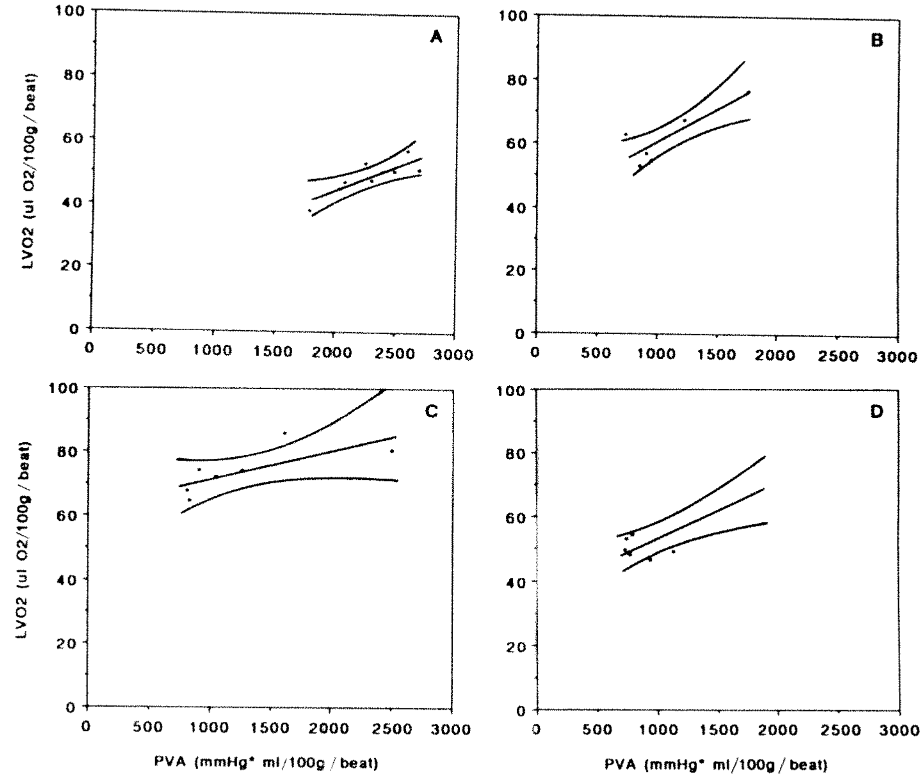

Fig 1. Relationship of left ventricular oxygen consumption (LVO2) to pressure-volume area (PVA) before (A), 1 hour after (B), 2 hours after (C), and 5 hours after (D) 12 minutes of warm (37°C) ischemia for 1 sheep. Each slope was calculated from nine measurements, four of which were made at increased afterload. (A: $Y = 1.43 \times 10^{-2} X + 17$, $r = 0.87$; B: $Y = 2.40 \times 10^{-2} X + 34$, $r = 0.91$; C: $Y = 0.98 \times 10^{-2} X + 62$, $r = 0.75$; D: $Y = 1.44 \times 10^{-2} X + 39$, $r = 0.88$.)

Comment

Twelve minutes of warm ischemia significantly increases the slope of the relationship between myocardial oxygen consumption and total ventricular mechanical energy output 1 hour after injury, but by 2 hours the slope is again normal. The impairment in oxygen utilization of the loaded ventricle is reversible but outlasts the period of ischemia by a factor between 5 and 10.

The effect of ischemia on LVO_2 is only apparent when total ventricular mechanical energy output is increased by increasing afterload. One hour after ischemia, left ventricular oxygen consumption per unit of PVA or CSI of the working ventricle without increased afterload does not differ between control and ischemic sheep. Figure 1 shows that at the ventricular mechanical energy outputs (PVAs) of the working ventricle without increased afterload, LVO_2 is similar in control and ischemic sheep. However, when afterload is increased, the slopes differ significantly.

Reversible ischemia also decreases myocardial contractility as measured by Ees. However, under the conditions of our study (eg, anesthesia, open chest), contractile force does not recover within 6 hours of the ischemic event. Therefore, we do not know if recovery of contractile force coincides with recovery of the LVO_2/PVA and LVO_2/CSI relationships.

The reduction in total ventricular mechanical energy output (PVA) after 42 minutes of cardiopulmonary bypass

Table 2. Oxygen Consumption and End-Systolic Elastance

Variable	Group	Before Ischemia	After Ischemia 1 h	2 h	3 h	4 h	5 h	6 h
LVO_2/PVA ([μL/mm Hg · ml] × 10^{-2})	Con	2.2 ± 0.9	2.7 ± 1.2[a]	2.2 ± 0.5	2.7 ± 1.4	1.1 ± 0.6[b]	2.0 ± 1.3	1.4 ± 0.7
	Exp	1.6 ± 0.8	5.8 ± 4.4[c]	1.8 ± 0.7	2.2 ± 1.1	2.9 ± 2.3	2.2 ± 1.3	5.0 ± 4.4
LVO_2/CSI (μL/mm Hg · s)	Con	1.76 ± 0.78	2.25 ± 0.41[b]	1.51 ± 0.41	2.05 ± 0.74	1.24 ± 0.88	2.42 ± 2.02	2.91 ± 2.56
	Exp	1.45 ± 0.59	4.39 ± 2.88[c]	1.80 ± 0.93	1.92 ± 1.27	2.04 ± 1.91	2.04 ± 1.72	1.89 ± 1.63
End systolic elastance (mm Hg/mL)	Con	3.57 ± 0.37	2.78 ± 0.67	2.51 ± 0.33	2.32 ± 0.35[d]	2.38 ± 0.32[c]	2.13 ± 0.34[c]	2.15 ± 0.25[c]
	Exp	4.48 ± 0.41	2.45 ± 0.26[c]	2.13 ± 0.20[c]	2.00 ± 0.29[c]	1.95 ± 0.14[c]	2.10 ± 0.27[c]	2.06 ± 0.21[c]

Nine measurements were made in each sheep at each time interval and regression slopes were calculated from these nine measurements. For each interval, the regression slopes were averaged for 5 control sheep and 10 experimental sheep (except as noted in Table 1). The table contains the mean slopes with standard deviations. (CSI = circumferential (systolic) stress integral; LVO_2 = left ventricular oxygen consumption; PVA = pressure–volume area. [a] $p = 0.062$, unpaired Student's *t* test between groups; [b] $p < 0.05$, unpaired Student's *t* test between groups. [c] $p < 0.05$ and [d] $p < 0.01$, paired Student's *t* test, before versus after bypass.)

Table 3.

Variable	Group	Before Ischemia	After Ischemia					
			1 h	2 h	3 h	4 h	5 h	6 h
PVA (joules × 10^{-1})	Con	2.36 ± 0.47	1.45 ± 0.19^{a}	1.42 ± 0.21^{a}	1.12 ± 0.24^{b}	1.48 ± 0.15	1.44 ± 0.19	1.31 ± 0.17
	Exp	2.24 ± 0.28	1.13 ± 0.12^{b}	1.10 ± 0.10^{b}	0.98 ± 0.10^{b}	1.08 ± 0.18^{b}	1.13 ± 0.21^{b}	1.09 ± 0.24^{b}
LVO_2 (joules × 10^{-1})	Con	9.14 ± 0.87	10.47 ± 1.74	9.56 ± 1.69	9.65 ± 2.03	8.20 ± 0.92	10.35 ± 1.12	11.15 ± 0.77
	Exp	9.59 ± 1.24	10.12 ± 1.99	8.61 ± 1.29	8.95 ± 1.41	10.08 ± 2.18^{b}	13.81 ± 2.62	15.20 ± 2.23
PVA/LVO_2 (%)	Con	27.8 ± 6.9	15.3 ± 3.2^{a}	17.2 ± 4.0^{a}	13.7 ± 3.5^{a}	18.5 ± 2.0	14.1 ± 1.7	12.2 ± 2.4
	Exp	25.5 ± 2.9	13.3 ± 1.5^{b}	14.7 ± 1.8^{b}	13.2 ± 2.2^{b}	12.2 ± 1.9^{b}	11.5 ± 3.1^{b}	8.9 ± 2.5^{b}
EW/LVO_2 (%)	Con	22.3 ± 5.0	12.1 ± 2.2^{a}	12.7 ± 2.5	9.5 ± 2.3^{a}	12.5 ± 0.9	8.9 ± 1.8	8.4 ± 1.2
	Exp	20.6 ± 2.4	10.9 ± 1.3^{b}	11.4 ± 1.5^{b}	9.3 ± 1.9^{b}	8.5 ± 1.6^{b}	7.8 ± 1.9^{b}	6.4 ± 1.7^{b}
IW/LVO_2 (%)	Con	5.5 ± 2.1	3.2 ± 1.1	4.5 ± 1.7	4.2 ± 1.4	6.0 ± 1.8	5.2 ± 1.2	3.8 ± 1.7
	Exp	5.0 ± 0.7	2.3 ± 0.4^{a}	3.3 ± 0.6	3.8 ± 0.6	3.8 ± 0.8	3.6 ± 1.5	2.6 ± 0.8^{a}

Pressure-volume area (PVA) and left ventricular oxygen consumption (LVO_2) have been converted to joules × 10^{-1}. Oxygen utilization efficiency is then calculated for the entire heart (PVA/LVO_2) and for external work (EW) and internal work (IW). None of the differences between groups is significant ($p > 0.05$). (^{a} $p < 0.05$ and ^{b} $p < 0.01$, paired Student's *t* test, before versus after bypass.)

is a surprising observation. The reduced energy output is primarily related to reduced left ventricular peak systolic pressures; prebypass and postbypass cardiac outputs as estimated by multiplying stroke volume and heart rate (data not tabulated) are similar. Pressure–volume area decreases significantly after cardiopulmonary bypass in both groups, but the LVO_2/PVA and LVO_2/CSI relationships only *increase* temporarily in the ischemic animals. As Novitzky [11] implies, cardiopulmonary bypass alone depresses total ventricular mechanical energy output but does not significantly decrease Ees. Addition of 12 minutes of ischemia significantly reduces contractility (as assessed by Ees) and increases the LVO_2/PVA relationship.

Only speculative explanations can be offered for the bypass-induced decrease in total ventricular energy output. Pulseless cardiopulmonary bypass may change the distribution of blood flow within the myocardium to deprive the subendocardium even though the ventricle is vented and total coronary blood flow is unchanged. The near-total absence of collateral vessels in the ovine coronary circulation [9] may produce tiny ischemic areas around the terminal branches of the coronary arteries. Neuroendocrine responses and generation of vasoactive peptides from platelets, endothelial cells, neutrophils, and other sources [12; Downing SW, Edmunds LH Jr, unpublished results] may directly impair myocardial oxygen utilization efficiency as well as increase capillary permeability and vasodilatation. In these experiments, bypass circuits were sterile, depth of anesthesia did not change, and all animals received calcium after protamine. Nevertheless, 42 minutes of warm bypass decreases total ventricular mechanical energy output but does not change the relationships between LVO_2/PVA and LVO_2/CSI nor decrease end-systolic elastance for several hours.

Both cardiopulmonary bypass and reversible ischemia plus bypass significantly decrease ventricular oxygen utilization efficiency (PVA/LVO_2). Unlike other indices of myocardial energy production, PVA is an energy term and can be converted to joules [13]. Myocardium consistently metabolizes 1 mL of oxygen into 20 J; therefore, measurement of PVA makes calculations of oxygen utilization efficiency possible [13]. As shown by the increased slope of the LVO_2/PVA relationship at 1 hour, ischemia clearly decreases oxygen utilization efficiency of the afterloaded ventricle. Although this observation is provocative, we suspect that more precise methodology than that which is possible in vivo may show an ischemic effect on oxygen utilization efficiency of the left ventricle working at low mechanical energy outputs also. However, in these studies, cardiopulmonary bypass is the primary cause of the decrease in oxygen utilization efficiency, which varies between 34% and 65% of prebypass values; there are no differences in total oxygen utilization efficiency or in either compartment between control and experimental groups.

Left ventricular oxygen consumption and coronary arterial blood flow do not change significantly at any time. The reactive hyperemia that follows reversible ischemia subsides within 1 hour. Bavaria and associates [2] observed a significant increase in left ventricular oxygen consumption of the working ventricle a mean of 94 minutes after 25 minutes of warm ischemia, but Furukawa and co-workers [1] did not. In the present study, cardiopulmonary bypass does not change left ventricular oxygen consumption of the resting heart but does reduce total ventricular mechanical energy output.

These studies show that 42 minutes of warm cardiopulmonary bypass significantly decreases total ventricular mechanical energy output and oxygen utilization efficiency of the working sheep heart but does not change left ventricular oxygen consumption. The addition of 12 minutes of ischemia reversibly increases the LVO_2/PVA and LVO_2/CSI relationships when afterload is increased and significantly decreases myocardial contractility, which remains depressed after the LVO_2/PVA and LVO_2/CSI slopes return to preischemic and prebypass values.

Addendum

We regret to point out some errors in table and figure headings in our previous article [1]. In Figure 2, the ordinate should read $LVVO_2$ (L O_2/100 g/beat) and the abscissa should read P-VA (mmHg · mL/100 g/beat). In Table 2, $LVVO_2$ (J/beat) should be multiplied by 10^{-4} and P-VA and EW should be multiplied by 10^{-3}. In Tables 3, 4, and 5, the $LVVO_2$ term for intercepts should read L O_2/100 g/beat (ie, a slash should replace the asterisk).

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DISCUSSION

DR RICHARD M. ENGELMAN (Springfield, MA): I enjoyed listening to your presentation, and I wonder, have you given any thought to extending your observations to include a preconditioned heart, a heart that has had, say, repeated stunning or simple preconditioning to see how it manifests changes in the oxygen utilization?

DR FURUKAWA: We have not done that, but it is an interesting thought.

DR MICHAEL GROSSO (Denver, CO): This is nice work. I had one question. Did you find the decreased oxygen efficiency utilization decrease just on cardiopulmonary bypass alone interesting? What do you think is going on there?

DR FURUKAWA: That was an unexpected finding, and we do not know the mechanism of that decrease. At this point we can only speculate as to whether or not cardiopulmonary bypass releases vasoactive peptides that affect the myocardium. However, this is just speculation, and I do not have answers to that question.

DR TIMOTHY J. GARDNER (Baltimore, MD): The phenomenon of myocardial stunning is a loss of contractile function or efficiency after an ischemic insult. It has been my understanding that this contractile loss persists far beyond the postischemic period in which changes in oxygen utilization are observed. I have assumed previously that the oxygen metabolism defect that occurs early after ischemia resolves far sooner than the contractile deficit or the ventricular stunning.

DR FURUKAWA: I think that is what we found, because the measurement of contractile function did not recover by the end of our observation period. However, the relationship of left ventricular oxygen consumption to pressure–volume area, which measures utilization efficiency, did recover somewhere between 1 hour and 2 hours after the ischemic period ended.

Cardiopulmonary Bypass, Profound Hypothermia, and Circulatory Arrest for Neurosurgery

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Six female and 4 male patients (age, 23 to 75 years) underwent operation for difficult intracranial lesions. Preoperative diagnoses included four giant intracranial aneurysms, three base of skull glomus jugulare tumors, two arteriovenous malformations, and one cerebellar hemangioblastoma. All lesions were inoperable or nearly so by standard neurosurgical techniques. All patients were placed on total bypass via groin cannulations. Bypass times ranged from 111 to 269 minutes (mean, 174 minutes) with cooling times of 26 to 83 minutes (mean, 48 minutes) and warming times of 68 to 110 minutes (mean, 83 minutes). Circulatory arrest times ranged from 1.25 to 60 minutes with 1 patient not requiring arrest. The lowest core temperatures recorded varied from 8.4° to 13.7°C. There was one postoperative death and one major complication, both in patients with arteriovenous malformations. Eight patients (80%) have achieved an excellent result. Profound hypothermia with the option of circulatory arrest and exsanguination has been an indispensable adjunct to the safe management of intracranial aneurysm, glomus jugulare tumor, and hemangioblastoma.

(Ann Thorac Surg 1991;52:1069–75)

Extracorporeal circulation, profound hypothermia, and circulatory arrest has been used in open heart surgery since 1959 [1]. The first reported use of this technique for the resection of an intracranial tumor was in 1960 [2]. It was then used at a number of neurosurgical centers [3–5]. This technique was generally abandoned because early techniques of hypothermia and extracorporeal bypass were cumbersome, added significant morbidity, and were time consuming. Also, the development of modern neuroanesthesia and microsurgical techniques has allowed safe operation on most intracranial aneurysms. However, even with modern neurosurgical techniques, some intracranial lesions are still judged to be inoperable. As a better understanding of hypothermic physiology and coagulation has developed, hypothermic circulatory arrest has been refined and standardized. It is now being applied safely and relatively conveniently to noncardiac complex surgical problems [6–8]. We report our experience with extracorporeal circulation and profound hypothermia with the option of circulatory arrest and exsanguination for the extirpation of intracranial lesions that would have been inoperable, or nearly so, with conventional techniques.

Material and Methods

Clinical Material

Six female and 4 male patients ranging in age from 23 to 75 years underwent operation for difficult intracranial lesions requiring cardiopulmonary bypass between 1983 and 1988 (Table 1). Preoperative diagnoses included four large intracranial aneurysms, three base of skull glomus jugulare tumors, two arteriovenous malformations (AVMs), and a cerebellar hemangioblastoma. Patient 1, who had a giant clinoid aneurysm of the left internal carotid artery with a broad-based neck and extension into the sella turcica, presented with frontal headache and third nerve palsy. A 1.6-cm aneurysm of the right middle cerebral artery presented with amaurosis fugax secondary to embolization from an internal carotid siphon atherosclerotic plaque (patient 8). Both vertebral aneurysms, located in the posterior fossa, presented with hearing loss. All 3 patients with glomus jugulare tumors presented with ipsilateral deafness, 2 of whom had ipsilateral twelfth nerve palsies. All three glomus jugulare tumors occluded the jugular bulb with invasion of the middle ear and base of skull. Two of these tumors had extension into the posterior fossa. Both patients with AVMs presented acutely with severe headaches from intracerebral hemorrhages. Patient 5, who had a 1.5-cm AVM located in the right cerebellar hemisphere also had right-sided paresis and cerebellar signs. The other AVM (in patient 7) was located posterior and superior to the right internal capsule with inflow from the posterior cerebral artery and superior cerebellar arteries and outflow through a large aneurysmal vein into the vein of Galen. The hemangioblastoma was located in the cerebellar vermis obstructing the aqueduct of Sylvius and presented with posterior midline headache and cerebellar signs (patient 4).

Concurrent illness was related to age, being present in all 6 patients over the age of 50 years (4 with intracranial aneurysms and 2 with glomus jugulare tumors) and in

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Table 1. Clinical Material

Patient No.	Age (y)	Sex	Diagnosis	Presentation	Concurrent Illness
1	57	M	Giant clinoid aneurysm of left internal carotid	Frontal headache, CN III palsy	COPD
2	60	F	Right glomus jugulare tumor, posterior fossa extension	Right deafness, right CN XII palsy	Hypertension, peptic ulcer disease, hypothyroidism, arthritis
3	24	F	Left glomus jugulare tumor, posterior fossa extension	Left deafness, left CN XII palsy, pulsatile tinnitus	None
4	28	M	Cerebellar hemangioblastoma	Posterior headache, cerebellar signs	None
5	24	F	Right cerebellar AVM	Subarachnoid hemorrhage, cerebellar signs, right paresis	None
6	53	M	Right glomus jugulare tumor	Right deafness, recurrent otitis	Parkinson's disease
7	23	F	Large right parietal AVM	Subarachnoid hemorrhage	None
8	61	M	1.6-cm aneurysm right middle cerebral artery	Amaurosis fugax	Femoropopliteal bypass, heavy smoker
9	71	F	Posterior fossa left vertebral aneurysm	Left deafness, blindness	Right vertebral artery occlusion with CVA
10	75	F	Posterior fossa right vertebral aneurysm	Right deafness	Chronic lymphocytic leukemia

AVM = arteriovenous malformation; CN = cranial nerve; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident.

none of the 4 other patients whose ages ranged from 23 to 28 years (2 with AVMs, 1 with a glomus jugulare tumor, and 1 with a cerebellar hemangioblastoma). Of the patients with intracranial aneurysms, 2 had peripheral vascular disease. A 61-year-old male smoker had a previous femoropopliteal bypass, and a 71-year-old woman had antecedent right vertebral artery occlusion with previous cerebrovascular accident. A 57-year-old man had mild chronic obstructive pulmonary disease, and a 75-year-old woman had chronic lymphocytic leukemia. Of the 2 older patients with glomus jugulare tumors, 1 had Parkinson's disease and the other suffered from hypertension, peptic ulcer disease, hypothyroidism, and arthritis.

Preparation

The diagnostic evaluation included computed tomography and cerebral angiography. The large aneurysms had feeding vessels located in such a way as to be unapproachable without collapse of the aneurysm using circulatory arrest. Profound hypothermia and circulatory arrest was also necessary for cerebral protection in the patient with the left vertebral artery aneurysm (patient 9) because of a contralateral vertebral artery occlusion. Two of the glomus jugulare tumors were amenable to preoperative embolization of the ascending pharyngeal artery, which decreased the vascularity of the tumors. After review of the preoperative studies, it was determined that extracorporeal circulation, profound hypothermia, and circulatory arrest was appropriate.

Surgical Technique

Anesthesia was induced with thiopental sodium and fentanyl. Muscle relaxation was accomplished with a combination of pancuronium and succinylcholine. Inhalation agents used were either isoflurane or enflurane. Radial arterial lines and central venous lines were inserted in each patient. In the last 7 patients, pulmonary artery catheters and end-tidal carbon dioxide monitoring were used. Routine cardiopulmonary bypass intraoperative monitoring included electrocardiography, direct radial artery pressure, central venous pressure, expiratory carbon dioxide, urine output, pulmonary artery pressure, and core temperature. Arterial blood gases, hematocrit, and cardiac output were measured intermittently. All patients received dexamethasone sodium, 8 to 12 mg initially, then 6 to 10 mg at 8 hours.

With the patient in the supine position, the groins were prepared and draped for cannulation and the chest was sterilely prepared for possible external cardiac defibrillation and possible median sternotomy. The head was elevated and was held in appropriate position using Mayfield tongs. A semisitting position was used as needed for suboccipital craniectomy. In 2 patients with evidence of increased intracranial pressure, lumbar spinal fluid drainage was initiated and intracranial pressure was monitored. The cerebellar hemangioblastoma (patient 4) obstructed the aqueduct of Sylvius necessitating a right ventriculostomy catheter.

Initial cooling was begun with a heating-cooling blanket and the neurosurgical dissection commenced. External cooling decreased the core temperature to 32°C by the time of bypass. The intracranial dissection was facilitated with the use of an operating microscope. Three hours 40 minutes to 8 hours 11 minutes were required to complete the neurosurgical dissection to the point where decompression of the vascular abnormality was needed.

Table 2. Perfusion Details and Results

Patient No.	Bypass (min)	Cooling (min)	Arrest (min)	Warming (min)	Total OR Time	Core Temp (°C)	Result
1	165	28	43	68	8 h 15 min	12	Excellent, minor complication
2	250	76	60	108	13 h 15 min	13.7	Excellent
3	156	50	27	79	12 h 5 min	8.4	Excellent
4	111	26	0	71	12 h 50 min	13.4	Excellent
5	177	83	14	75	11 h 25 min	10	Prolonged neurological deficit, major complications
6	152	42	25	85	11 h 30 min	13.2	Excellent, recurrent tumor 5 y postop
7	269	74	1.25	72	17 h 50 min	9.5	Died
8	119	25	3	77	12 h 45 min	11.8	Excellent
9	195	44	35	110	15 h	13.7	Prolonged neurological recovery
10	145	28	37	82	15 h 50 min	10.4	Excellent, minor complication

OR = operating room.

End-tidal CO_2 monitoring may have prevented a gas embolus in 1 patient. The end-tidal CO_2 precipitously fell when the dura was opened. Though there was no evidence of any openings into the sinuses, the end-tidal CO_2 normalized after we covered the undersurface of the occipital sinus and performed further bipolar cauterization of the lateral margins. The patient had no subsequent problems.

After systemic heparinization with 400 IU/kg both femoral veins were cannulated with large (24F to 30F) multiholed catheters. Degree of heparinization was monitored by activated clotting and heparin assay. One of the venous cannulas was directed into the right atrium and the other drained the inferior vena cava. One femoral artery was cannulated.

A bubble oxygenator was used in 8 patients and a membrane oxygenator in 2. Hemodilution prime solution consisted of 2 L of Normosol-R (pH 7.4), 10,000 IU beef lung heparin, 8 mEq of KCl, 50 mEq of sodium bicarbonate, 250 to 500 mL of 20% mannitol solution, and 25 g albumin. Pulsatile flow was used during cooling and rewarming. Gas flow was adjusted as needed based on venous and arterial saturations of oxygen and carbon dioxide (O_2/CO_2 mixture for the bubble oxygenator and O_2/air mixture for the membrane oxygenator). The bubble oxygenator had excellent heat exchange capability, whereas the membrane oxygenator required one to two additional heat exchangers. It was found that at low flows with only one exchanger in the venous line, the blood returning to the patient drifted toward ambient temperature. Rewarming time was shortened by placing a heat exchanger in the arterial line. Two units of autologous blood were removed at initiation and reinfused at conclusion of bypass. Initial bypass flows were 2.8 to 4.0 L/min with a goal of 40 to 50 $mL \cdot kg^{-1} \cdot min^{-1}$.

Perfusion details and results are presented in Table 2. Cooling was accomplished using a heat exchanger water temperature of 20°C initially, decreasing to 0°C. Cooling times ranged from 26 to 83 minutes (mean, 48 minutes). Ventricular fibrillation occurred between 23° and 28°C. Core temperature was monitored using a variety of techniques including mixed venous return (all 10 patients), esophageal (7 patients), bladder (4 patients), tympanic membrane (3 patients), rectal (2 patients), and nasopharyngeal (1 patient). Lowest core temperatures achieved varied from 8.9° to 13.7°C (esophageal) or 8.4° to 13.0°C (mixed venous return).

When a core temperature of 10° to 12°C occurred, the patient was exsanguinated into the pump oxygenator and cardiotomy reservoir as needed for collapse of the aneurysm or reduction of vascularity. This technique allowed minute to minute control of the blood supply to the area of dissection. Four patients required more than one period of circulatory arrest with intervening trickle flows (500 to 750 mL/min). Total circulatory arrest times ranged from 1.25 to 60 minutes. Resection of the cerebellar hemangioblastoma was facilitated by trickle flow and arrest was not required (patient 4). Circulatory arrest in the patient with the large AVM (patient 7) made delineation of the extent of the lesion more difficult, and several periods of trickle flow were used instead.

After completion of the neurosurgical procedure, bypass at low flows was resumed for a final hemostatic examination. Once hemostasis was complete, full bypass with rewarming began. Spontaneous defibrillation occurred at 23°C, 25°C, and 31°C in 3 patients. Seven patients required external countershock. Rewarming required 68 to 110 minutes (mean, 83 minutes).

Estimated blood loss varied from 500 to 4,000 mL. An autotransfusion device was used for red cell scavenge. At the conclusion of bypass all patients received the withdrawn autologous blood, scavenged blood, and five platelet packs. Homologous whole blood use varied from 2 to 12 units. Heparinization was reversed using protamine sulfate, 1.2 mg/100 IU heparin, based on heparin assay at the conclusion of bypass. Reversal of heparin was validated by the activated clotting time and heparin assay. Additional platelets, fresh frozen plasma, and protamine were given as needed depending on operative field hemostasis and coagulation variables. Total operating time varied from 8 hours 15 minutes to 17 hours 50 minutes.

Results

Results are presented in Table 2. Four patients had no postoperative complications and an average hospital stay of 13 days. Resection of a right glomus jugulare tumor through a base of skull and mastoidectomy approach required transposition of the facial nerve, which resulted in an expected temporary postoperative paralysis (patient 6). This tumor recurred 5 years postoperatively and was successfully resected through a translabyrinthine approach without bypass. There were two minor complications. Patient 1 required cranioplasty 13 months later for a free floating cranial flap, and patient 10 required 2 weeks of nasogastric tube feedings for postoperative dysphagia.

Patient 9, a 71-year-old obese woman with hypertension and residuals of right blindness, right deafness, and mild right hemiparesis from a previous right vertebral artery occlusion, had a left vertebral aneurysm resected through a mastoidectomy and suboccipital approach. A severe postoperative neurological deficit of blindness, dysarthria, and dysphagia required a gastrostomy tube. A subdural hematoma was drained 1 month postoperatively. Extensive prolonged rehabilitation was necessary.

Results in AVM resection were disappointing and caused the only death (patient 7). Embolization of this large right parietal AVM was thought to be risky and not definitive. The inoperability of this lesion using conventional techniques was apparent. Biparietal craniotomy was performed. Delineation of the margins of resection was difficult. At the conclusion of the neurosurgical dissection it was noted that the right parietal lobe was congested and tense. It was believed that the swelling was from compromised venous drainage as both a bridging vein and a major vein draining the AVM had thrombosed. A pentobarbital coma was maintained postoperatively and a ventriculostomy was required on postoperative day 2 for increased pressure from an intraventricular bleed. Angiography showed severely diminished flow to the brain and autopsy demonstrated hemorrhagic infarction and residual AVM.

The other patient with an AVM (patient 5) also had major complications from postoperative swelling and hemorrhage. Postoperative severe hydrocephalus required a ventriculostomy. The posterior fossa was reexplored for bleeding on postoperative day 3. Subsequent respiratory failure required tracheostomy. Complete neurological recovery was prolonged by dysconjugate gaze, slow mentation, cerebellar speech, and some spasticity of the right arm.

Overall, 8 of 10 patients had an eventual excellent result (see Table 2). There was no cardiac morbidity and no clinical indication of thromboembolism from catheterization of the femoral vessels. In 1 patient, inadequate venous return did limit bypass flows to less than ideal, but it did not cause any problems.

Comment

Extracorporeal circulation, profound hypothermia, and circulatory arrest was developed in the 1950s and used successfully in the repair of congenital cardiac defects in infants in 1959 [1]. The first reported successful use of this technique for neurosurgery was by Woodhall and associates in 1960 [2]. They cannulated the femoral artery and jugular vein and did a palliative extirpation of a brain metastasis using hypothermic cardic standstill for 44 minutes and with the pump shut off for 10 minutes. After successful experimentation in dogs, Patterson and Ray [3] used this technique to treat seven intracranial aneurysms (two postoperative deaths) using esophageal temperatures of 5°C and arrest times up to 45 minutes. They provided the experimental basis that heart could be kept adequately decompressed using peripheral cannulation and that thoracotomy was not needed.

The experience at the Mayo Clinic in the 1960s demonstrated the superiority of the closed chest method of peripheral cannulation over the open chest method of bypass for operation on difficult intracranial lesions [4]. In this series of 66 patients, coagulation abnormalities of bypass and hypothermia caused half of the 23% mortality [4]. In ten intracranial aneurysms operated on by Drake's group, the three postoperative deaths resulted directly from the use of extracorporeal circulation [5]. With this added morbidity and with the concurrent developement of microsurgery and neuroanesthesia, which made many of these aneurysms now operable, cardiac bypass in neurosurgery was largely abandoned. In 1964, Drake and associates [5] stated that the additional hazards created by the bypass do not warrant its use in cases where the aneurysm can be obliterated by more conventional means.

In 1974, McMurtry and co-workers [9] reported good results in ten of 12 basilar artery aneurysms using moderate hypothermia, sternotomy, and induced ventricular fibrillation without bypass. Sternotomy, cardiopulmonary bypass, profound hypothermia, and circulatory arrest was used by Gonski and associates [10] to operate on 40 giant or complicated intracranial aneurysms from 1974 through 1984. Postoperative hemorrhage caused four of ten deaths, and complications due to the bypass and sternotomy occurred in 23% [10]. Bypass was used less and less as previously inoperable anterior circulation aneurysms became approachable with the development of hypotensive neuroanesthesia and the use of the operating microscope.

However, certain intracranial lesions continued to be inoperable by standard techniques. Advances in the control of coagulation were making hypothermic bypass with circulatory arrest safer. Hypothermic bypass is now a routine technique for complex cardiovascular operations, and it has successfully been applied to difficult noncardiovascular operations [6–8]. In 1983, Baumgartner and colleagues [11] established extracorporeal circulation through groin cannulation, deep hypothermia, and circulatory arrest as safe for complex neurosurgical operations. Thirteen intracranial aneurysms and one medullary hemangioblastoma were judged to be inoperable by standard techniques. There were no deaths and 13 of 14 patients had a good result. The target core temperature was 20°C, and arrest times ranged from 5 to 51 minutes. Coagulation was achieved at the end of bypass by the routine administration of protamine, 6 U of platelets, 4 U of fresh frozen plasma, 1 to 2 g of calcium chloride, autologous blood, and a unit of factor IX concentrate; however, the incidence of deep venous thrombosis and pulmonary emboli was 29% [11].

The effect of hypothermic bypass on coagulation is complex and not completely understood. It continues to be the major disadvantage of this technique. Even a tiny ooze at the neurosurgical operative site can have disastrous consequences. Hypothermia produces thrombocytopenia, probably by sequestration [12]. Hypothermia has been shown to cause reversible platelet dysfunction and slows all enzyme-mediated steps in the coagulation cascade [11, 13]. The fibrinolytic system is activated, and a dilutional effect of coagulation factors I, II, V, VII, and XIII occurs [11, 14]. By using retransfusion of autologous blood, banked whole blood for any red cell deficit, accurate and documented reversal of heparin, routine platelet transfusion, fresh frozen plasma for any residual oozing or correction of coagulation variables, and continued exposure of the neurosurgical operative field until absolute hemostasis was obtained, we operated on four large intracranial aneurysms, three base of skull glomus jugulare tumors, and a cerebellar hemangioblastoma without any bleeding difficulties and no thromboembolism. Accurate reversal of heparin and platelet infusions are probably the two most important corrections. Fortunately the effect can be measured rapidly with the activated clotting time, heparin assay, and platelet counts.

The results in intracranial AVM resection were not as good. Hemostatic complications occurred in both patients with AVMs and caused the only death. This may be due to the nature of AVMs and not due to coagulation abnormalities. The vessels leading to and from a AVM are abnormal both anatomically and physiologically [15]. Autoregulation is lost in the surrounding area of the brain that has been chronically exposed to low perfusion pressures from the vascular steal of the AVM. When these abnormal capillary beds are then exposed to normal pressures after extirpation of the lesion, hemorrhage, swelling, and congestion occurs. The dilated, tortuous draining veins are chronically exposed to high blood flows from the AVM; when the high flows are removed, these veins have a propensity to clot. Severe brain swelling and hemorrhage occurred postoperatively in both of our patients, and thrombosis of the draining veins contributed to the death of one of these patients. The complete extirpation of these lesions can be difficult as their margins are difficult to delineate. Residual AVM devoid of venous drainage can contribute to congestion and hemorrhage [15]. Residual AVM was seen at autopsy. The natural history of these lesions without operation is dismal. Sixty-six percent of 134 patients followed up without operation for an average of 12 years died or became disabled [16].

The use of extracorporeal circulation and hypothermic arrest has produced an improvement in the results of operation for giant intracranial aneurysm and facilitated operation on glomus jugulare tumors. The results of surgical treatment for giant intracranial aneurysms traditionally has been poor [17]. The largest reported experience shows rates of 37% to 22% for major operative morbidity and mortality for giant aneurysms of the vetebral, basilar, anterior, and middle circulation arteries [17]. Circulatory arrest collapses the aneurysm, exposing the neck and feeding vessels for confident control. Baumgartner and associates [11] had a good result using bypass and hypothermic arrest in nine of ten of these types of giant aneurysms. Although base of skull glomus jugulare tumors have been successfully resected without extracorporeal circulation, extracorporeal circulation with hypothermic arrest greatly facilitates confident, controlled resection of all gross disease in a tumor with a predilection for reccurrence without adding morbidity [18].

Cerebral protection remains the factor that limits the time of arrest or low flow. Hypothermia decreases the oxygen requirement and protects the brain from ischemia [19]. This cerebral protection cannot be totally explained by the temperature-induced decrease in basal metabolic rate. Studies in sheep show that hypothermia also increases the energy state and intracellular pH of the brain [19]. Flows as low as 10 mL $\cdot$ kg^{-1} $\cdot$ min^{-1} can maintain this high energy state and intracellular pH for 2 hours at 15°C, and this correlates with improved survival and preservation of neurological function (Swain JA and associates, unpublished results). We have used deep hypothermic arrest of up to 60 minutes at core temperatures of 8.4° to 13.7°C without clinically apparent neurological sequelae. The maximum time of deep hypothermic arrest has not been established definitively, but 30 years of clinical experience and animal studies suggest that at least 60 minutes is safe [6, 7, 10].

The temperature at which the brain is metabolically inactive varies, and the estimation of it depends on the site of temperature measurement. The isoelectric electroencephalogram or electrocerebral silence is thought to be consistent with metabolic inactivity of the brain [20]. Woodhall and co-workers [2] determined that this occurred consistently at a brain temperature of 20° to 22°C. Coselli and colleagues [20] reported a wide variation in temperature among body sites when electrocerebral silence occurred and believed that electroencephalographic monitoring should be used to determine the target temperature in each patient. We used a variety of temperature measuring techniques. Esophageal, tympanic membrane, and nasopharyngeal temperature correlated closely with each other. Rectal and bladder temperature tended to be unreliable. Flow during bypass is reduced, and there may be uneven distribution of flow and uneven cooling. Controlled vasodilation has been used to increase the rate of equilibration [11]. We did not find this to be necessary. Using more than one monitor of central core temperature seems prudent in the light of our experience of technical difficulties with the various methods.

Hypothermic hemodilution alters the oxygen carrying capabilities and rheologic characteristics of blood. Decreasing temperature shifts the hemoglobin saturation curve to the left, and hemoglobin more tightly binds oxygen. However, any ischemic anaerobic metabolism causes the opposite effect. The solubility of oxygen in plasma increases with decreasing temperature. Without any hemoglobin and with a normal cardiac output, this dissolved oxygen may be adequate if the temperature is lowered sufficiently [11]. This permits the hemodilution that protects the brain from ischemia during hypothermia, ameliorates the hypothermic bleeding diathesis, and prevents the sludging due to the increased viscosity of hypothermic blood [14].

Extracorporeal circulation through groin cannulation, profound hypothermia with the option of circulatory arrest, and exsanguination has been an indispensable adjunct to the management of giant intracranial aneurysms, glomus jugulare tumors, and cerebellar hemangioblastoma. Large AVMs continue to be a challenge even with these techniques.

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DISCUSSION

DR JULIE A. SWAIN (Las Vegas, NV): I would like to thank Dr Williams for this very nice presentation and for the opportunity to review the manuscript. This is a very nice adjunct to the Stanford group's 1970s report and updates us as to what can be accomplished with excellent results in the 1980s. I have a couple of questions.

One is the choice of core temperature to 10°C. The work by Baumgartner in the Stanford group in a similar series of patients showed that their target was 20°C with good results. Our biochemical and metabolic data from nuclear magnetic resonance spectroscopy studies in animals suggests that 15°C is protective. Most of your times were less than 30 minutes; in fact only one was up to 60 minutes. So why did you choose 10°C rather than something that could be obtained with less cardiopulmonary bypass time?

The second question is whether you would consider using percutaneous cardiopulmonary bypass with two venous lines as a less invasive technique. Finally, most of your cases were done with bubble oxygenators. Could you comment on that choice?

DR WILLIAMS: Thank you, Dr Swain, for those comments. It was a pleasure to have you review the manuscript, and I apologize for perhaps the not unprecedented step of inviting you to review it 1 month ago but not giving you the manuscript until today.

The choice of core temperature used was 10°C, and as Dr Swain said, the Stanford group has used 20°C and her studies indicate 15°C affords adequate cerebral protection. We use 10°C because 10°C is easily obtainable using this technique, and with our monitors of core temperature, we believed that this would give us the best assurance that the actual brain temperature was below those criteria.

Dr Swain mentioned percutaneous insertion of the lines. We have not done this and do not have any experience with that. We used two venous return lines so that venous return would be adequate. Among our 10 patients there was only 1 patient in whom venous return was limited by the cannulas, and we find it important that we use both veins.

Dr Swain also mentioned bubble versus membrane oxygenators. In our first 8 patients we used the bubble oxygenator; in the last 2 we used the membrane oxygenator. Our bubble oxygenator had excellent heat exchange capabilities and was excellent for cooling and rewarming the patient, but the bubble oxygenator is harder on the blood cells. In the last 2 patients the membrane oxygenator was used; but with our particular oxygenator we had to add heat exchangers in both the venous and arterial lines to quickly cool and rewarm the patient so as not to extend the procedure.

Surgical Options in Subaortic Stenosis Associated With Endocardial Cushion Defects

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Over a 15-year period, 12 patients with endocardial cushion defects undergoing correction had subaortic stenosis requiring operative intervention. Ages ranged from 4 months to 17 years (mean, 7 ± 6 years) and subaortic gradients from 15 to 100 mm Hg (mean, 60 ± 25 mm Hg). Subaortic stenosis was due to discrete fibromuscular tissues in 7 patients, mitral valve malattachment in 3, and tunnel outflow in 2. In 2, the subaortic stenosis was clinically significant at the time of endocardial cushion defects repair, whereas in 10 it was noted 2 to 14 years postoperatively (mean, 6.3 ± 5 years). Surgical relief of subaortic stenosis was accomplished by resection of muscle tissues in 7, apicoaortic conduit insertion in 2, modified Konno procedure (aortic valve preserved) in 2, and lifting of malattached mitral valve from the outflow in 1. There was no early death and one late death (infected conduit). Severe mitral insufficiency developed in the patient who had the mitral valve lifted and necessitated valve replacement. Postoperative echocardiographic gradient in 9 patients ranged from 0 to 36 mm Hg (mean, 10.5 ± 14 mm Hg). Clinically significant subaortic stenosis has not developed in any patient in 15 years of follow-up (mean, 5 ± 4 years). We conclude that in subaortic stenosis associated with endocardial cushion defects, resection is effective for discrete obstruction, whereas a modified Konno procedure is preferable for obstruction due to tunnel outflow or mitral valve malattachment.

(Ann Thorac Surg 1991;52:1076–83)

Subaortic stenosis is not uncommon in patients with endocardial cushion defects, with the incidence ranging from 3% to 7% [1–4]. The deficiency of the muscular septum and the abnormal displacement of mitral valve result in a long left ventricular outflow tract with predisposition for subaortic stenosis [5–7]. The obstruction can be due to the presence of discrete fibromuscular tissue, tunnel outflow, or malattachment of endocardial cushion tissues to the outflow tract.

Serious subaortic stenosis may already be present at the time of repair of the endocardial cushion defect or may develop later. The diagnosis may be missed preoperatively as the gradient across the outflow tract can be masked by the atrioventricular septal defect, or the diagnosis may be delayed due to inadequate follow-up [2, 8].

The presence of inherently narrow left ventricular outflow tract and of complex endocardial cushion abnormalities contributing to the obstruction poses a surgical dilemma. Simple resection is often inadequate and carries a high mortality rate [9, 10]. This report is a review of our experience in the surgical management of subaortic stenosis associated with endocardial cushion defect.

Material and Methods

Patients

Over a 15-year period ending in 1989, 12 patients with endocardial cushion defect had subaortic stenosis requiring operative intervention. The clinical profile is outlined in Table 1. Mean age was 7 ± 6 years and mean peak systolic subaortic gradient was 60 ± 25 mm Hg. Only 3 patients had Down's syndrome. Three patients had coarctation of the aorta. Two of these patients underwent coarctation repair with additional pulmonary artery banding followed by subclavian pulmonary artery shunt in 1 (patient 2) before repair of the endocardial cushion defect. One patient (patient 8) also had partial anomalous pulmonary venous drainage involving the right upper lobe that was repaired along with the coarctation at the time of endocardial cushion defect correction.

In 2 patients (patients 1 and 2), the subaortic stenosis from discrete fibromuscular tissues was recognized before repair of the endocardial cushion defect. In the remaining 10 patients, the subaortic stenosis was diagnosed 2 to 14 years (mean, 6.3 ± 5 years) after the repair of the endocardial cushion defect. The obstruction was due to discrete fibromuscular tissues in 5, mitral valve malattachment in 3, and tunnel outflow in 2 (Figs 1, 2). In 3 patients left ventricular angiography before repair of the endocardial cushion defect was suggestive of left ventricular outflow tract obstruction although no pressure gradient was demonstrated.

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Table 1. Clinical Profile

Patient No.	Age (y)	Down's Syndrome/ Coarctation	Type of ECD	Initial Repair to SAS[a] (y)	Type of SAS	Gradient (mm Hg)
1	0.3	D	Complete	. . .	Discrete	15
2	4	D/C	Complete	. . .	Discrete	38
3	16	. . .	Complete	14	Discrete	40
4	8	C	Partial	5	Discrete	100
5	5.5	. . .	Partial	4	Discrete	50
6	3	. . .	Partial	2	Discrete	65
7	7.5	D	Complete	6.5	Discrete	65
8	4	C	Partial	3.5	Tunnel	65
9	17	. . .	Partial	13	Mitral valve	65
10	2	. . .	Complete	2	Mitral valve	40
11	3.5	. . .	Partial	2	Tunnel	70
12	15	. . .	Partial	11	Mitral valve	100

[a] Time from repair of ECD to diagnosis of SAS.

C = coarctation; D = Down's syndrome; ECD = endocardial cushion defect; SAS = subaortic stenosis.

Operative Procedures

Two patients (patients 1 and 2) underwent resection of discrete fibromuscular tissues through a right atrial approach by retraction of the anterior bridging leaflet at the time of complete atrioventricular canal repair. Ample patch was used to close the ventricular septal defect underneath the anterior bridging leaflet and to enlarge the left ventricular outflow tract. The second patient also had pulmonary artery debanding and closure of a subclavian–pulmonary artery shunt.

In 5 patients (patients 3 through 7) with discrete fibromuscular narrowing diagnosed after the endocardial cushion repair, generous resection of muscle along with fibrous tissues was performed through an aortotomy.

In 2 patients (patients 8 and 9) in whom the obstruction was caused by malattached mitral valve and tunnel outflow, a left ventricular apicoaortic Dacron conduit with porcine valve was inserted (Fig 3) [11, 12]. The conduit exited through the pericardium, coursed upward medial to the left lung, reentered the pericardium, and was anastomosed to the proximal ascending aorta.

In 2 other patients (patients 10 and 11) in whom the obstruction was also due to malattached mitral valve and tunnel outflow, the modified Konno procedure was performed (Fig 4) [13, 14]. After bicaval and ascending aorta cannulation for cardiopulmonary bypass, the right ventricular outflow was opened. Through an aortotomy the subaortic area was assessed, the aortic valve protected, and a conal incision made just inferior to the pulmonary valve leaving a rim of tissue for subsequent placement of sutures. The conal septum was then enlarged using a polytetrafluoroethylene patch with pledgeted sutures. The infundibulotomy was closed with a polytetrafluoroethylene patch, and the aortotomy closed primarily.

In the last patient with severe subaortic stenosis diagnosed 11 years after an ostium primum repair, resection

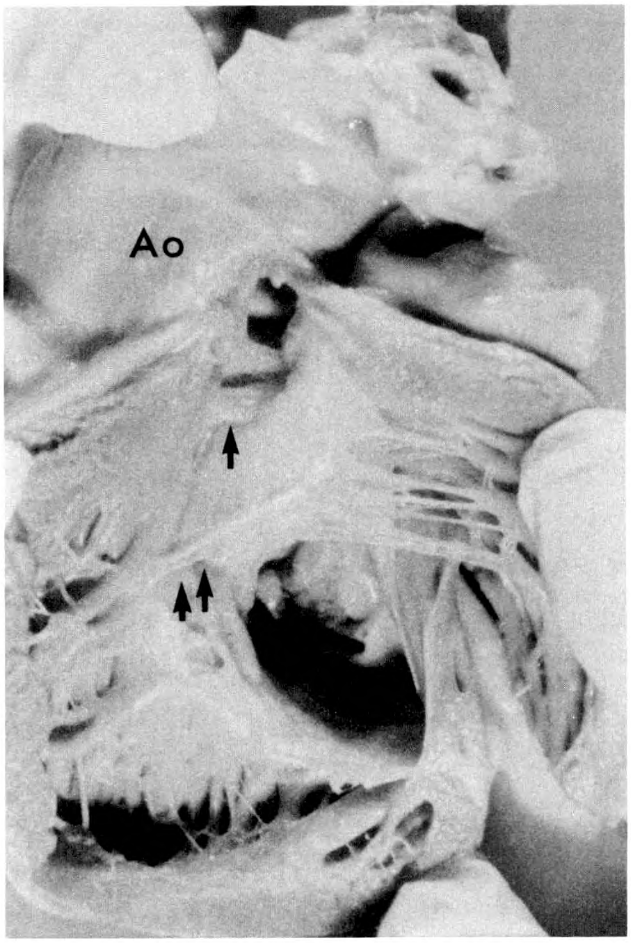
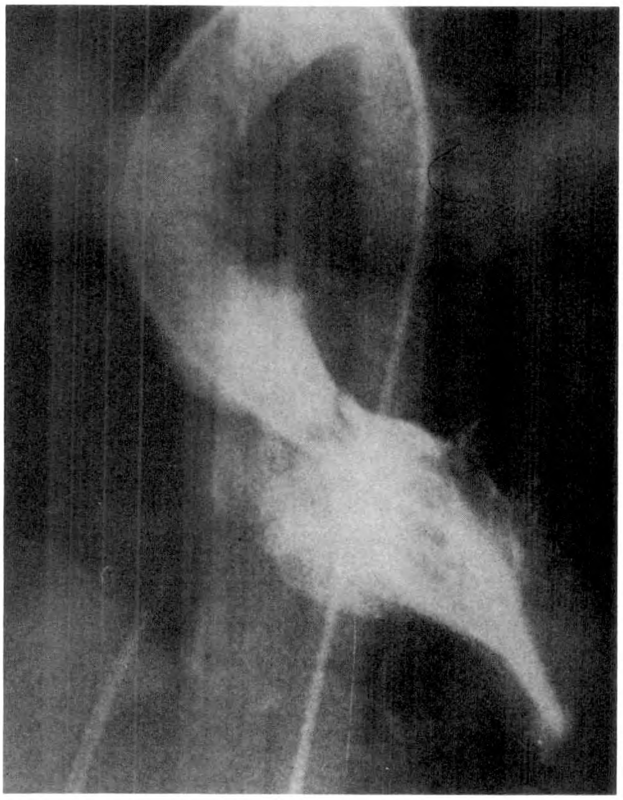


Fig 1. Left ventricular view of a heart specimen with atrioventricular septal defect showing anterior displacement of the anterior leaflet of the mitral valve causing outflow narrowing. Accessory mitral valve (arrow) *and chorda* (arrows) *contributing to obstruction are shown.* (Ao = *aorta.*)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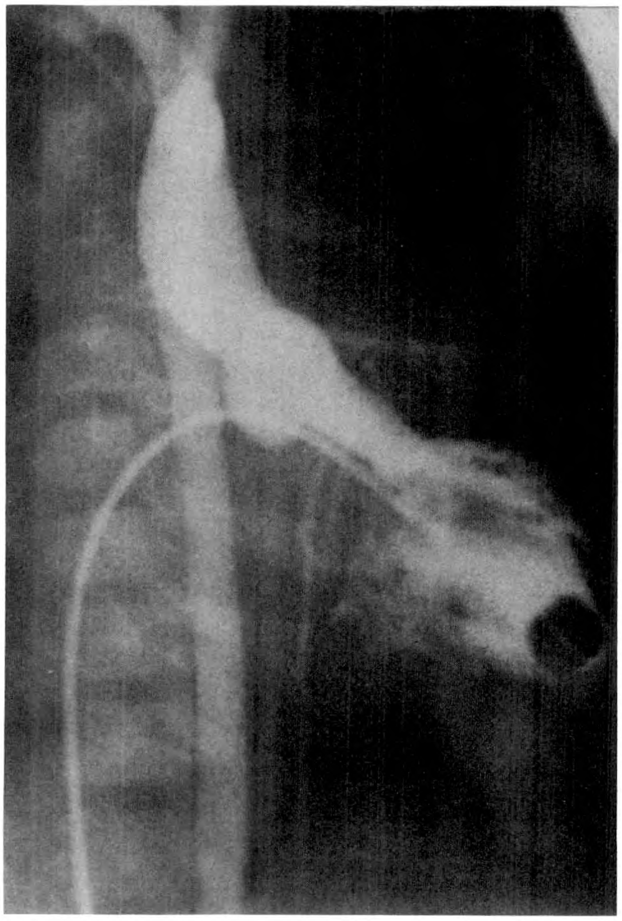
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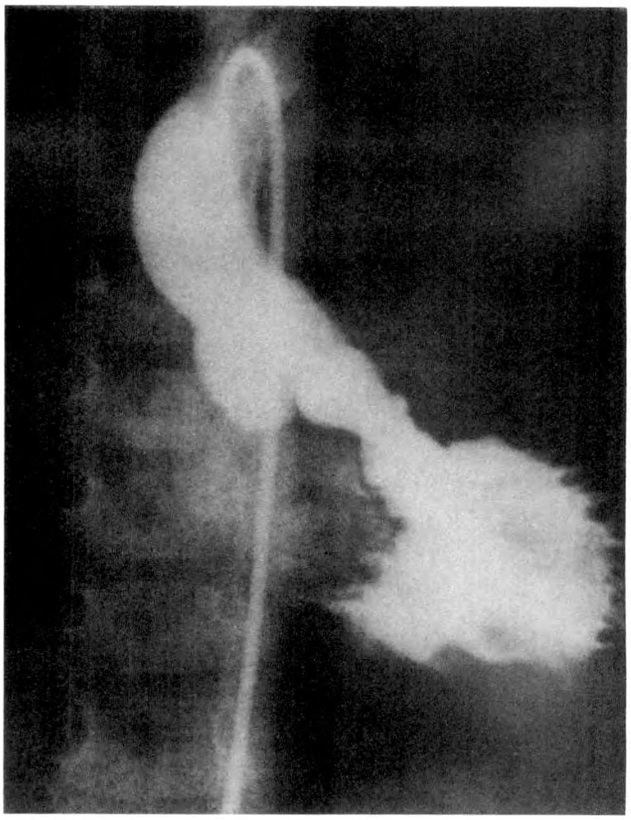
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Fig 2. (A) Left ventriculogram (patient 4) showing discrete subaortic stenosis. (B) Left ventriculogram (patient 11) showing tunnel outflow. The anterior displacement of the mitral valve probably is contributory. (C) Left ventriculogram (patient 10) showing malattached mitral valve causing outflow obstruction.

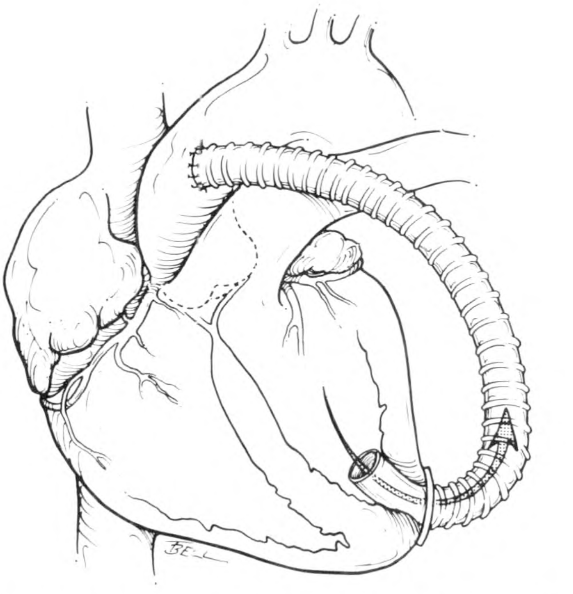

Fig 3. Apicoaortic valved conduit inserted in 2 patients. The conduit coursed between the pericardium and left lung.

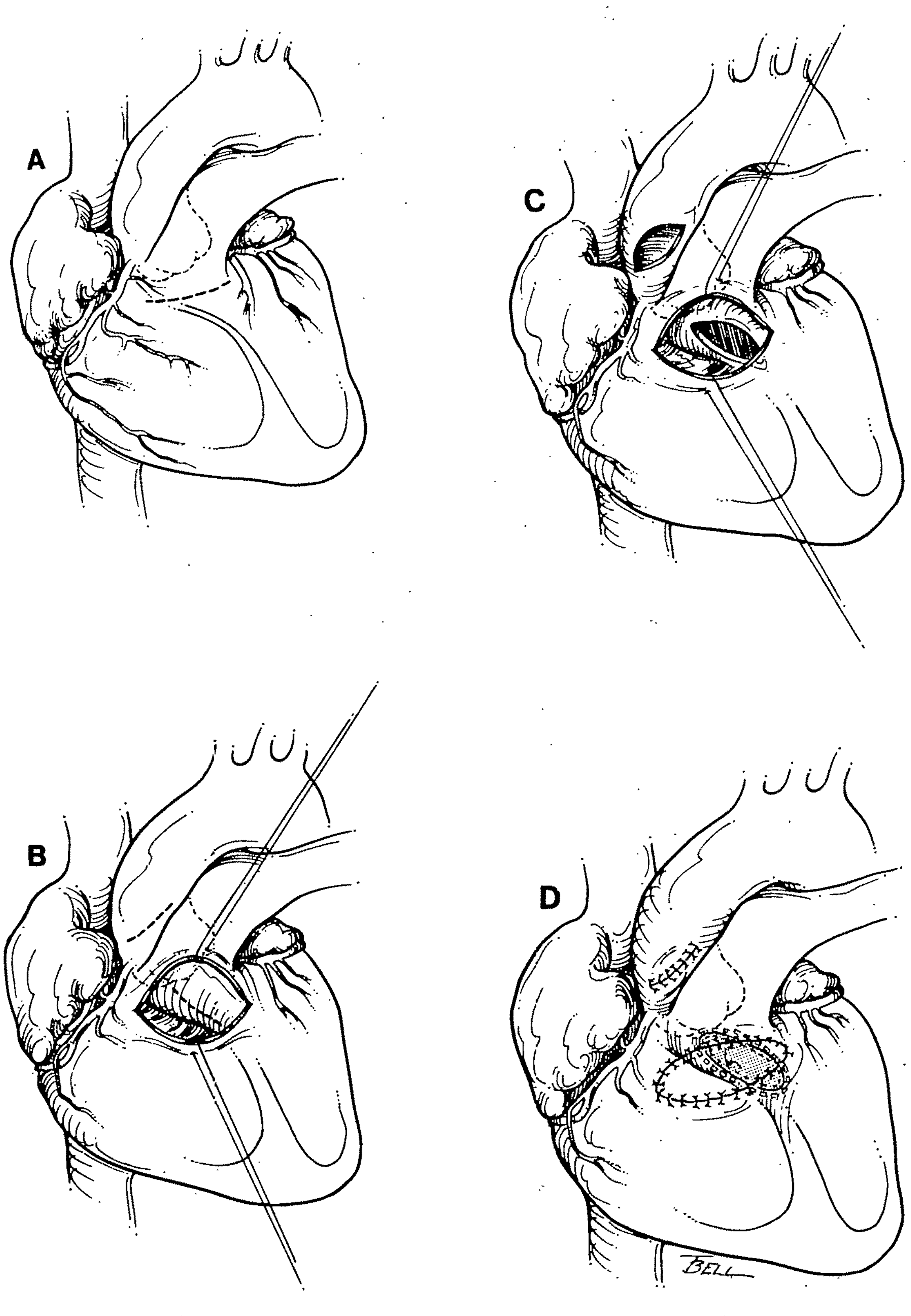

Fig 4. Modified Konno procedure (2 patients). The incision is marked in the infundibulum, ascending aorta, and conal septum. Aortotomy is done for assessment of the outflow and protection of the aortic valve. The outflow is enlarged with a polytetrafluoroethylene patch. The infundibulotomy is also closed with a patch.

and myotomy was performed. Two years later, cardiac catheterization revealed a subaortic gradient of 100 mm Hg secondary to the malattached mitral valve. Through a right atrial approach, the atrial septum was opened. The malattached mitral valve was detached from the top of the ventricular septum and a ventricular septal defect was created (Fig 5). A pericardial patch was then used to close the ventricular septal defect, enlarging the left ventricular outflow tract. The mitral valve was reattached to the upper edge of the pericardial patch [8, 15].

Results

Early

The postoperative course in all patients was uneventful.

Late

One patient died of *Staphylococcus aureus* sepsis 2 years after insertion of the apicoaortic conduit. Postmortem examination showed infection of the conduit and abscesses in the brain, kidney, and spleen. Unexplained fever and severe mitral insufficiency developed 2 months postoperatively in the patient who underwent lifting of the malattached mitral valve. Although no organism was isolated, infective endocarditis was suspected and treated. The mitral valve was replaced with a porcine valve and the patient did well with no evidence of subaortic stenosis. Replacement of the porcine valve with a metallic prosthetic valve was required 7 years later.

Postoperative echocardiography performed 1 month to 10 years (mean, 3 ± 3 years) in 9 patients showed a subaortic gradient ranging from 0 to 36 mm Hg (mean, 10.5 ± 14 mm Hg). In the 2 patients who had a modified Konno procedure, the subaortic gradient was 0 and 20 mm Hg, respectively, with the gradient localized across a mildly hypoplastic aortic valve annulus (Fig 6).

Ten patients who have been followed up for up to 15 years (mean, 5 ± 4 years) are clinically well, and none have shown serious subaortic stenosis. One patient (patient 5) was lost to follow-up.

Comment

The presence of an elongated left ventricular outflow tract in patients with endocardial cushion defects raises the possibility of potential or actual subaortic stenosis, which remains a major surgical problem [5–7]. Piccoli and colleagues [3] found unequivocal subaortic stenosis in 8 of 114 heart specimens (7%) with atrioventricular septal defects. DeBiase and colleagues [1] reported subaortic stenosis in 4 of 90 consecutive patients (5%) with atrioventricular canal. Taylor and Somerville [2] found fixed subaortic stenosis developing in 3 of 99 patients (3%) after repair of ostium primum defects.

Subaortic stenosis can be due to discrete fibromuscular tissues in the left ventricular outflow tract or to actual tunnel outflow narrowing. In addition, several endocardial cushion tissue abnormalities can occur that may be primary or contributory factors to the obstruction. These include anterior displacement of the mitral valve, malattachment of chordae and papillary muscle, presence of accessory mitral valve tissue, or presence of accessory tricuspid tissue protruding through a ventricular septal defect into the outflow tract [16–20].

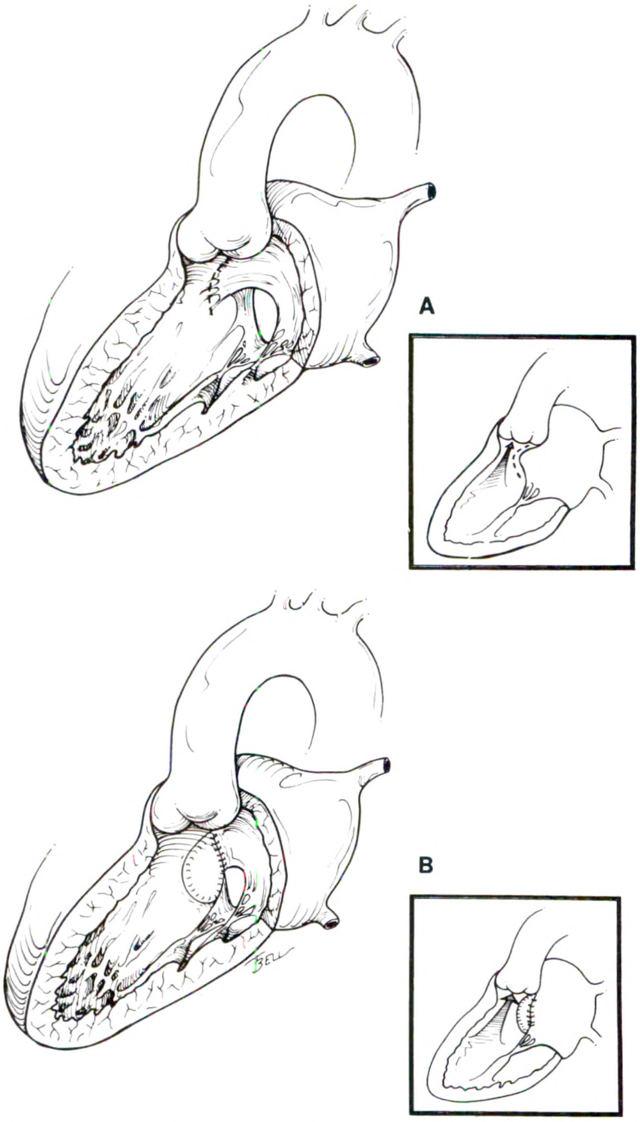

Fig 5. (A) Lifting of malattached mitral valve using a right atrial approach and incision through atrial septum (1 patient). The area is marked for detaching the anterior mitral leaflet from the left ventricular outflow tract, creating a ventricular septal defect. (B) The ventricular septal defect is closed with a patch enlarging the left ventricular outflow tract, and the anterior mitral leaflet is reattached to the top of the patch.

The diagnosis of subaortic stenosis based on conventional pressure data may be difficult during cardiac catheterization because of the potential masking effect of the left to right shunt on the gradient across the left ventricular outflow tract [8]. Our patients who had simultaneous repair of subaortic stenosis and canal had low preoperative outflow gradients. Lappen and colleagues [8] and Taylor and Somerville [2] claim that persistence or exaggeration during systole of the characteristic angiographic diastolic "gooseneck" deformity is diagnostic of potential or actual subaortic stenosis in endocardial cushion defect.

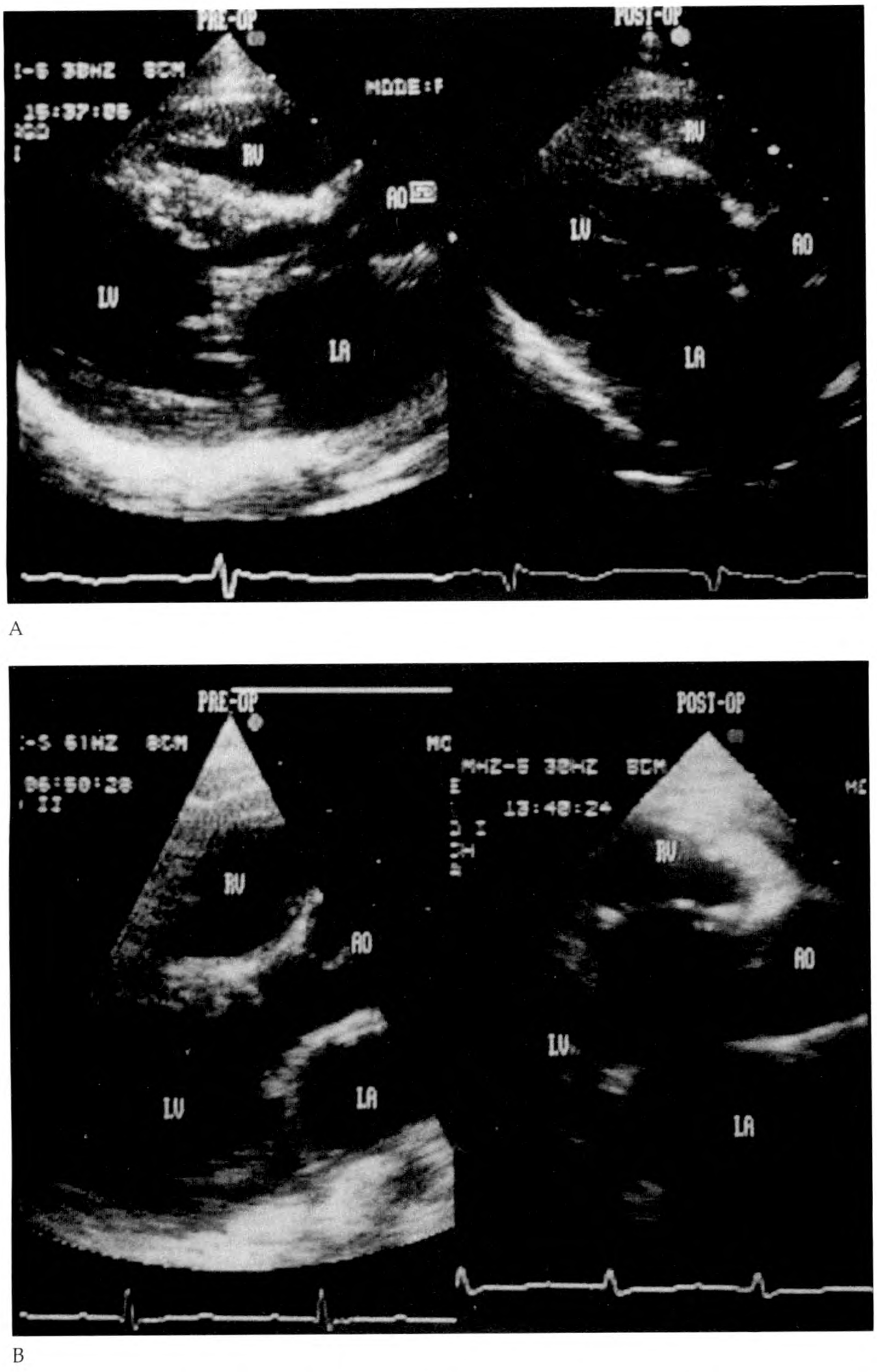

Fig 6. Echocardiograms of the 2 patients who had the modified Konno procedure. Preoperative pictures (left) show the narrowed left ventricular outflow tract, which became quite wide after the procedure (right). (Ao = *aorta;* LA = *left atrium;* LV = *left ventricle;* RV = *right ventricle.)*

In patients who had previous repair of atrioventricular septal defects, the possibility of subsequent subaortic stenosis cannot be ignored. Closure of the cleft of the anterior leaflet of the mitral valve may contribute to the narrowing of the left ventricular outflow tract [21]. Early detection of subaortic stenosis requires a high index of suspicion and follow-up endocardiographic examination looking for systolic fluttering and early closure of the aortic valve, narrowing of the left ventricular outflow tract, left ventricular hypertrophy, and increased systolic flow velocity across the outflow tract [2].

The appropriate surgical technique for relieving the subaortic stenosis chiefly depends on the anatomy of the outflow tract. Despite the important data provided by echocardiography and angiocardiography, the intraoperative findings ultimately determine the surgical technique that is needed. When the left ventricular outflow tract is not too narrow, relief of the obstruction by resection of discrete fibromuscular tissues may suffice, provided that generous myectomy is also performed. However, if the left ventricular outflow tract is inherently narrowed, resection of fibromuscular tissues alone may be ineffective and carries a high mortality risk as well as recurrence rate [9, 10, 21]. The increased turbulence across a narrowed subaortic area stimulates connective tissue proliferation, increased deposition of fibrous tissue, and further progression of obstruction in a vicious cycle [2].

When subaortic stenosis without a substantial outflow gradient is suspected before repair of the endocardial cushion defect, exploration of the outflow tract and resection of fibromuscular tissues along with the repair of the atrioventricular septal defect is a reasonable approach. In patients in whom a serious outflow obstruction as measured by serial angiography or echocardiography has developed after atrioventricular septal defect repair, intraoperative exploration should help in deciding between simple fibromuscular resection alone or a more aggressive surgical approach such as insertion of an apicoaortic conduit, modified Konno procedure, or lifting of malattached mitral valve. Where the subaortic stenosis is recurrent, a more aggressive surgical approach is indicated [22].

Although the modified Konno procedure has been reported in only a few cases [14, 23], it is preferable over insertion of an apicoaortic valved conduit as the native aortic valve is preserved. Long-term anticoagulation and possible replacement of valve prosthesis are avoided with the modified Konno procedure. Lifting of the malattached anterior mitral leaflet from the left ventricular outflow tract is an alternative. Experience with the technique, however, is quite limited and there is a risk of mitral insufficiency [8, 15].

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DISCUSSION

DR JOSEPH J. AMATO (New Hyde Park, NY): I congratulate Dr DeLeon and associates on the superb management of a difficult situation. I have, however, two questions for them. First, because only two of these 12 patients with endocardial cushion defects were diagnosed as having severe subaortic stenosis, is it possible that some technical problem with your use of the two-patch technique could have been the cause of the development of fibromuscular tissue or valve malattachment in the other 10 patients 2 to 14 years later?

Second, can you clarify your statement regarding accessory mitral valve tissue? Was the tissue found actual valvular tissue or excessive fibrous tissue developed from obstructive flow?

DR DeLEON: The majority of our patients had an ostium primum defect, although 5 also had a complete canal. What we have been warning all along is that when you repair a complete canal you should use an ample patch and not just suture the anterior bridging leaflet down to the ventricular septum, converting it to ostium primum and thereafter closing the atrial septal defect. We use a two-patch technique so that we do not interfere much with the mitral or tricuspid valve.

DR ADNAN COBANOGLU (Portland, OR): I was surprised at the number of discrete subaortic stenosis cases that you encountered. I would expect almost all of these cases to have a structural basis to them or to develop in the manner that you describe with fibrous tissue deposition in the subaortic region over a period of years. I think that even after a local myectomy, the underlying structural abnormality, which you very nicely indicated, is still there. I wonder if at 10 years of follow-up you will not see some of these patients come back for a third operation because of recurrence of a similar pathology in exactly the same area in the left ventricular outflow tract.

DR DeLEON: That is an excellent question. The incidence I quoted of 3% to 7% was mostly from pathology specimens. I think the lack of awareness of this defect resulted in very few cases operated on in the past, but now, with increased awareness, we should be able to recognize this problem more often. As I indicated in the presentation, the outflow tract is quite long in patients with endocardial cushion defect, which leads to turbulence in the area with increased tissue deposition. If you are not aware of this phenomenon, you may just resect what you might think are discrete tissues causing the obstruction, not knowing that the inherent problem is a long, narrowed outflow tract. In the past, this has led to increased recurrence and mortality rates.

I think I failed to answer one of Dr Amato's questions. Echocardiography and angiography of these patients would suggest the presence of tunnel outflow or malattachment of the mitral valve. The ultimate decision as to what to do, however, is in the intraoperative exploration. With recognition of an obstruction at the time of the initial presentation, resection of the obstructing tissues along with the repair of the canal should be adequate, unless there is no question that there is tunnel outflow or malattached mitral valve. In some patients in whom subaortic stenosis develops after canal repair, it will be obvious that there is tunnel outflow or malattached mitral valve. However, there will be certain patients who will fall into a gray zone. In these patients, do you do resection or do you go ahead with an invasive approach? In these patients, you may be justified to do resection with generous myectomy. In the event of recurrence, I think you are justified to go ahead with a more aggressive approach such as the modified Konno procedure.

DR COBANOGLU: Intraoperatively in these cases you have control as far as the diameter of the left ventricular outflow tract is concerned if you are correcting a complete atrioventricular canal defect, because you can move the ventricular septal defect patch toward the right side of the septum and have the outflow area larger than you would by suturing onto the crest. I think the problem is in the partial canal defects, where the leaflet is already tethered and attached to the crest of the ventricular septum; at least in our experience, those have been the more difficult cases to deal with when we are aware that there is some preoperative left ventricular outflow obstruction at the time of total correction.

Partial Fontan: Advantages of an Adjustable Interatrial Communication

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Systemic venous hypertension after the Fontan procedure is a major cause of mortality and morbidity, accounting for 11 of 16 deaths in our series of 228 Fontan procedures. A partial Fontan with a residual atrial septal defect (ASD) would allow controlled right-to-left shunting to reduce venous pressure and improve cardiac output while maintaining a reduced but acceptable arterial oxygen saturation. This allows complete or graded closure of the ASD after the discontinuation of cardiopulmonary bypass in the operating room or at any time in the postoperative period by exposing the snare under local anesthesia. From 1987 to 1990, 36 patients undergoing the modified Fontan procedure had placement of an adjustable interatrial communication. Indications for placement of an adjustable ASD included increased pulmonary artery pressures, increased pulmonary vascular resistance, reactive airway disease, previously increased or unknown pulmonary vascular resistance, small pulmonary arteries, and borderline ventricular function. Fourteen patients had the adjustable ASD closed at the time of operation, 8 patients underwent narrowing, and 12 underwent closure of the ASD in the postoperative period. Eight patients were discharged with the ASD partially open, and 2 patients underwent delayed closure. The partial Fontan with an adjustable ASD may increase the safety of the Fontan procedure for high-risk groups such as those with increased pulmonary vascular resistance, pulmonary hypertension, and impaired left ventricular function and for infants, who tolerate venous hypertension poorly. The ability to adjust the ASD in stages depending on the hemodynamic response increases flexibility and safety.

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The Fontan-Kreutzer procedure has been extensively used as a definitive procedure for complex lesions in which a normally functioning pulmonary ventricle cannot be achieved, such as in patients with tricuspid atresia and univentricular heart [1–16]. The pulmonary and systemic venous returns are separated, resulting in improved systemic oxygenation and a reduced ventricular volume load. However, systemic venous hypertension (right atrial pressure [RAP] > 14 mm Hg) is frequently encountered after a Fontan procedure. If mild (up to 17 mm Hg), it is usually well tolerated except for a transient period of fluid accumulation. If the pulmonary vascular resistance (PVR) is elevated, even transiently, in the immediate postoperative period, this can result in a severe low cardiac output state and marked accumulation of extravascular fluid. The use of pulmonary vasodilators, inotropic agents, and the venous assist device are not always successful in maintaining adequate cardiac output. Elevation in PVR and ventricular failure are the most frequent causes of early death after the Fontan procedure. Attempts to take down a Fontan procedure at this stage are also associated with a high mortality [17].

We hypothesized that in select patients undergoing the Fontan procedure, maintenance of an adequate cardiac output in the early postoperative period might be achieved by creating a limited right-to-left shunt to allow part of the systemic venous return to bypass the pulmonary circulation and directly enter the left side. This would result in some limited desaturation but improved blood flow to the left side with increased cardiac output and decreased systemic venous pressures. It can be calculated that a right-to-left shunt of 33% (pulmonary to systemic flow ratio = 0.67) would result in an arterial saturation of 85% and would, therefore, allow one third of the systemic venous return to bypass the pulmonary circulation.

Clinical observation of patients with residual right-to-left shunts of this magnitude after a Fontan operation has shown that such a shunt is well tolerated in association with a reduced RAP. An externally adjustable restrictive interatrial communication, or atrial septal defect (ASD), was therefore developed to provide such a controlled right-to-left atrial shunt that allowed graded narrowing or closure later without the need for cardiopulmonary bypass or interventional catheter closure. The adjustable ASD can be left open as the patient is weaned from bypass

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and, if hemodynamics are suitable, immediately closed in the operating room, or if pulmonary resistance is high, the adjustable ASD can be left open through the early postoperative period. With a reduction in PVR over the next few hours or days, the snare can be exposed under local anesthesia and the ASD adjusted or closed without the need for cardiopulmonary bypass.

Experimental Study

To evaluate the feasibility of the adjustable ASD, a snare-controlled adjustable ASD was developed in an experimental animal model. The ability to control the adjustable ASD was demonstrated in dogs in which the interatrial septum was replaced with a Dacron atrial patch that included an adjustable ASD and in which systemic venous hypertension was induced by creation of tricuspid regurgitation and a pulmonary artery band.

Methods

For this study, five mongrel dogs weighing 15 to 20 kg were anesthetized with ketamine hydrochloride and sodium pentobarbital, paralyzed, and intubated. The chests were opened through a midline sternotomy and the hearts suspended in a pericardial cradle. Bicaval cannulation was performed for venous drainage, and an aortic cannula was placed for perfusion. Cardiopulmonary bypass with moderate hypothermia (28°C) was instituted, the aorta was cross-clamped, and cold blood cardioplegia was given. The right atrium was opened, and the atrial septum was excised and replaced with a polytetrafluoroethylene patch (Fig 1). A flap approximately 7 by 5 mm was made in a trapdoor fashion on the right lateral aspect of the patch. Two 1-0 Prolene sutures were passed through the flap; one was brought out through the lateral aspect of the interatrial septum for closing the ASD, and the other was brought out through the right atrial wall near the base of the aorta for opening the ASD. The ends of the Prolene were placed through a short segment of 8F polyethylene tubing. The snares were tested to demonstrate opening and closing of the trapdoor (see Fig 1).

To raise RAP, tricuspid regurgitation was induced by excision of 50% of the anterior leaflet of the tricuspid valve; the atriotomy was then closed. Electromagnetic flow probes (Carolina Medical Electronics, Inc) were placed around the aorta and main pulmonary artery. Pressure catheters were placed in the left atrium, right atrium, and pulmonary artery, and the data were recorded on Camino 420 monitors. The animals were then weaned from cardiopulmonary bypass. A pulmonary artery band was placed using an umbilical tape passed through a snare and tightened to create an RAP of 14 mm Hg. Fluid administration was required to maintain cardiac output while the ASD was closed. Contrast echocardiography was used to confirm opening and closing of the shunt.

Right atrial, left atrial, and pulmonary artery pressures were measured. Pulmonary artery and aortic flow measurements were obtained with flow probes, and epicardial echocardiography was done with the ASD open and then repeated with the ASD closed. Two sets of each measurement were obtained along with serial arterial blood gases with the ASD open and closed.

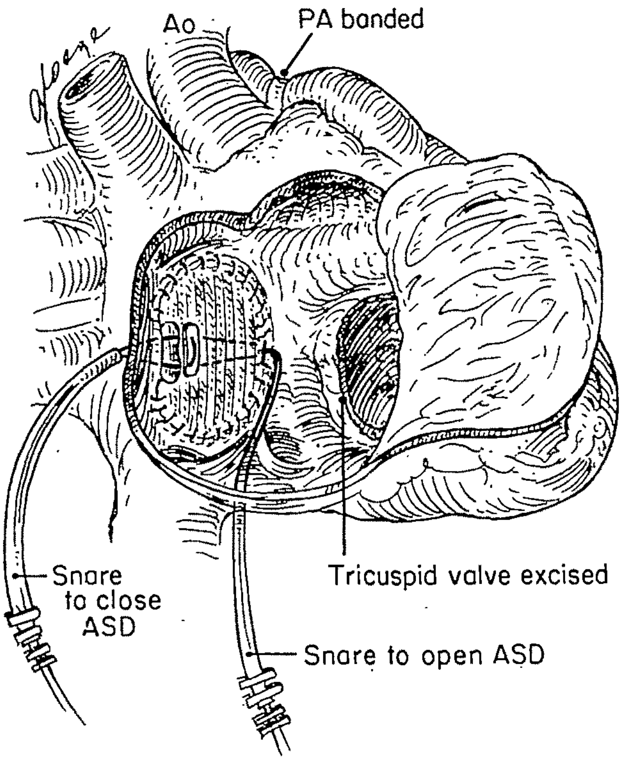

Fig 1. Experimental model of an adjustable atrial septal defect (ASD) *in a Dacron patch in a dog. The tricuspid valve has been excised and the pulmonary artery* (PA) *banded to create right atrial hypertension. A snare for closing and a separate snare for opening the adjustable ASD are present.* (Ao = *aorta.)*

All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

The dogs were in highly unstable condition with the ASD closed and required continuous volume infusion to support blood pressure and cardiac output. However, with the ASD open, the dogs remained hemodynamically stable despite a decreased oxygen saturation and required no volume infusion. Mean values for RAP, left atrial pressure, pulmonary flow, and oxygen saturation are shown in Table 1. The mean RAP was 15 ± 1 mm Hg with the ASD closed and decreased to 10 ± 1 mm Hg when the ASD was opened. Concomitantly, the left atrial pressure increased from 5 ± 1 mm Hg to 9 ± 1 mm Hg as the ASD was opened. The pulmonary artery flow decreased from a mean of 3.1 L/min to 2.2 L/min (29% decrease), whereas the aortic flow increased from 3 L/min to 4.3 L/min (43% increase) with the ASD open. These hemodynamic measurements demonstrated the effectiveness of the ASD in

Table 1. Experimental Model of Adjustable Atrial Septal Defect in Dogs[a,b]

ASD Status	LAP (mm Hg)	RAP (mm Hg)	Aortic Flow (L/min)	Pulmonary Flow (L/min)	Arterial Saturation (%)
Closed	5 ± 1	15 ± 1	3.0 ± 1	3.1 ± 1	97 ± 2
Open	9 ± 1	10 ± 1	4.3 ± 2	2.2 ± 2	78 ± 2

[a] The condition of the dogs was highly unstable with the ASD closed, and continuous volume infusion was necessary to support blood pressure and cardiac output. With the ASD open, their condition remained hemodynamically stable despite a decreased oxygen saturation, and no volume infusion was necessary. [b] Data are shown as the mean ± the standard deviation.

ASD = atrial septal defect; LAP = left atrial pressure; RAP = right atrial pressure.

reducing pulmonary blood flow and central venous pressure while increasing systemic cardiac output.

Clinical Experience

An adjustable ASD was placed in 36 patients undergoing a modified Fontan procedure. The diagnosis was tricuspid atresia in 15, univentricular heart in 19, and pulmonary atresia with intact ventricular septum in 2. The preoperative status of the patients is shown in Table 2. Six of the patients had had no prior palliative procedures, and 30 had undergone 52 prior procedures. The mean age was 5.7 years (range, 12 months to 15 years); there were 21 male and 15 female patients. The mean weight was 19 kg (range, 7.6 to 60 kg). The mean preoperative pulmonary artery pressure was 16 mm Hg (range, 6 to 26 mm Hg), the mean LVEDP was 9 mm Hg (range, 3 to 15 mm Hg), and the mean PVR was 2.4 Wood units (range, 0.4 to 3.6 Wood units) (Table 3).

The preoperative indications for use of the adjustable ASD are shown in Table 4. The adjustable ASD was used in 5 patients because of a borderline elevation of PVR and in 11 patients because of pulmonary hypertension secondary to high flow with normal calculated PVR. Mean pulmonary artery pressure greater than 20 mm Hg and PVR greater than 2 Wood units were considered borderline. In 11 patients, the ASD was used because preoperative pulmonary blood flow was less than systemic and there was uncertainty as to the true pulmonary resistance. These particular patients were considered to be at increased risk of having high postoperative RAPs and the potential for a low cardiac output state [1, 2, 6, 13, 17–21].

Surgical Technique

The systemic and pulmonary venous returns were separated in one of several ways. In 10 patients with tricuspid atresia, the ASD was partially closed and a snare placed around the remaining ASD (Fig 2A). In 9 patients with univentricular heart, a Dacron baffle was placed to allow communication between the left atrium and both atrioventricular valves. The adjustable ASD was placed in the Dacron patch (Fig 2B). In 17 patients (5 with tricuspid atresia, 10 with univentricular heart, and 2 with pulmonary atresia with intact ventricular septum), the adjustable ASD was left in a Gore-Tex patch used with the technique of total cavopulmonary diversion or lateral tunnel (Fig 3). In these patients, a bidirectional superior vena cava–right pulmonary artery anastomosis (Glenn) was also used [22].

The No. 1 Prolene sutures were passed through the lateral aspect of the interatrial septum, through a pericardial pledget, and then through an 8F polyethylene tube. The length of tubing was adjusted so that the end could be placed beneath the linea alba. The snare was tightened until the defect was the desired size, and then medium-sized hemaclips were placed on the Prolene suture to hold it in that position. Care was taken to avoid excessive force in applying the clips because they can cut through the Prolene.

The No. 1 Prolene was sutured to the edge of the trapdoor flap with 5-0 Prolene; pulling on the Prolene closed the trapdoor, and pushing on the heavy Prolene opened the trapdoor. The polyethylene tubing was sutured to the exit site in the interatrial septum so that inadvertent pulling on the snare would not close the ASD. The polyethylene snare was left beneath the linea alba for easy retrieval postoperatively without a thoracotomy. The distal ends of the Prolene suture were looped back and secured with a hemaclip to prevent erosion through the skin. A separate snare for opening the adjustable ASD was found to be cumbersome, and its use was abandoned after the first 2 patients. Because closing the ASD is more reliable than reopening it, which is not always possible, the ASD was closed in stages, adding one hemaclip at a time. More recently, we have preferred a simple defect in the Gore-Tex patch or atrial septum (see Fig 3) rather than the trapdoor just described.

The size of the ASD was estimated from the maximally acceptable shunt fraction and the Gorlin formula. It can be calculated that a pulmonary to systemic flow ratio of 0.67 would result in a 33% right-to-left atrial shunt and an arterial oxygen saturation of 85%, assuming a mixed venous oxygen saturation of 60%. This would reduce the volume of blood having to traverse the pulmonary vascular bed. Using the Gorlin formula, one can calculate that if the RAP is 15 mm Hg, if the left atrial pressure is 10 mm Hg, and if 1 $L \cdot min^{-1} \cdot m^{-2}$ (one third of the venous return) traverses the atrial communication, a defect 6 mm^2/m^2 is required. Right atrial and left atrial pressure lines and an arterial line were placed for hemodynamic monitoring. Systemic arterial oxygen saturation was determined by continuous-pulse oximetry and was confirmed with frequent measurements of arterial blood gases. Cardiac output assessment was made both by evaluation of systemic blood pressure and on clinical grounds with urine output, end-organ function, and systemic perfusion including skin temperature and color.

Adjustment of the ASD

The patient was weaned from cardiopulmonary bypass while the systemic blood pressure, the RAP, the left atrial pressure, and the systemic oxygen saturation were monitored with a pulse oximeter and by direct observation of

Table 2. Preoperative Profile of the 36 Patients

Patient No.	Age (mo)	Diagnosis	Systemic Saturation (%)	Hemoglobin g/dL	PVR (U/m^2)	Mean PA Pressure (mm Hg)	LVEDP (mm Hg)	QP/QS	Closed in OR	Tightened	Closed	Discharged Open	Closed Late (mo)
1	78	TA, central, B-TS	85	18	. . .	16	12	0.9	No	. . .	Yes	. . .	. . .
2	84	TA, VSD, s/p B-TS	73	23	. . .	. . .	10	0.45	No	Yes	Yes	. . .	. . .
3	60	TA, PAB, PA stenosis, VSD	78	19.8	. . .	17	7	1.3	No	Yes	. . .	Yes	. . .
4	28	TA, PS, B-TS	80	17.3	1.7	. . .	6	2	No	. . .	Yes	. . .	. . .
5	22	TA, TGA, PS	77	14	. . .	14	11	. . .	No	. . .	Yes	. . .	. . .
6	168	TA, VSD, B-TS	81	18	2.5	20	12	0.9	No	. . .	Yes	. . .	. . .
7	36	UVH, s/p Stansel, Glenn	86	17	. . .	. . .	8	. . .	No	. . .	Yes	. . .	. . .
8	47	PA-IVS, Waterson, Glenn	81	22	. . .	16	10	. . .	No	Yes	. . .	Yes	. . .
9	156	SV, s/p PAB	84	19	2.7	12	10	1.5	No	. . .	Yes	. . .	3.5
10	40	SV, MS, s/p PAB	93	14	. . .	26	11	2.4	No	Yes	. . .	Yes	. . .
11	23	DTX, UVH, PS	78	19	. . .	. . .	9	. . .	No	. . .	Yes	. . .	. . .
12	132	DISV, PAB, pulm hypertension	88	14	. . .	22	14	. . .	No	Yes	Yes	. . .	. . .
13	21	DTX, AV canal, AV regurgitation, SV	84	16	. . .	12	8	. . .	No	. . .	Yes	. . .	. . .
14	42	PA, SV, s/p Glenn, s/p left	78	18	2.7	. . .	10	1.2	No	Yes	. . .	. . .[a]	. . .
15	82	TA, Central, B-TS	79	18	. . .	. . .	15	. . .	No	. . .	Yes	. . .	. . .
16	180	AV canal, TGA, PS, SV	86	17.6	. . .	14	5	. . .	No	. . .	Yes	. . .	. . .
17	32	PA-IVS	76	16	. . .	18	6	0.49	No	Yes	Yes	Yes	3
18	53	SV, SA, DTX, TAPVR	81	16	. . .	17	10	. . .	No	Yes	. . .	Yes	. . .
19	72	Malposition, SV, MA, LSVC	63	21	3.4	20	9	0.22	No	. . .	. . .	Yes	. . .
20	29	UVH, DOLV, PA	77	18	. . .	. . .	12	. . .	No	. . .	. . .	Yes	. . .
21	48	TA, VSD, s/p PAB	76	20	. . .	16	11	0.77	No	. . .	Yes	. . .	. . .
22	32	TA	82	22	1.3	15	12	1.6	No	. . .	. . .	Yes	. . .
23	66	TA, PA, s/p GLENN, B-TS	86	16	3.5	20	10	0.75	Yes	Opened	. . .	. . .[a]	. . .
24	72	TA, VSD	88	17	. . .	17	11	1.1	Yes	. . .	. . .	. . .	. . .
25	62	SV, PS	89	16	. . .	20	7	3.1	Yes	. . .	. . .	. . .	. . .
26	48	SV, MA	84	20	. . .	10	14	0.85	Yes	. . .	. . .	. . .	. . .
27	159	PA, SV, AV regurgitation	87	16	. . .	. . .	12	2.1	Yes	. . .	. . .	. . .	. . .
28	93	SV, DI	81	18	. . .	13	8	1.4	Yes	. . .	. . .	. . .	. . .
29	58	SV	70	17	. . .	17	3	0.76	Yes	. . .	. . .	. . .[a]	. . .
30	72	SV, PA, TGA	82	20	. . .	18	10	2.1	Yes	. . .	. . .	. . .	. . .
31	62	SV, malposition, aorta	83	18	. . .	17	11	1.8	Yes	. . .	. . .	. . .	. . .
32	72	TA, VSD	86	18	. . .	33	10	2.6	Yes	. . .	. . .	. . .	. . .
33	12	TA	68	15	. . .	. . .	6	. . .	Yes	. . .	. . .	. . .	. . .
34	72	TA, VSD, ASD	85	23	. . .	16	16	1.6	Yes	. . .	. . .	. . .	. . .
35	30	TA, VSD	87	14	. . .	23	8	. . .	Yes	. . .	. . .	. . .	. . .
36	132	SV, s/p PAB	82	18	. . .	15	10	. . .	Yes	. . .	. . .	. . .	. . .

[a] Early death.

ASD = atrial septal defect; AV = atrioventricular; B-TS = Blalock-Taussig shunt; DI = DISV = double-inlet single ventricle; DOLV = double-outlet left ventricle; DTX = dextrocardia; LSVC = left superior vena cava; LVEDP = left ventricular end-diastolic pressure; MA = mitral atresia; MS = mitral stenosis; OR = operating room; PA = pulmonary artery, pulmonary atresia; PAB = pulmonary artery banding; PA-IVS = pulmonary atresia with intact ventricular septum; PS = pulmonary stenosis; PVR = pulmonary vascular resistance; QP/PS = pulmonary to systemic flow ratio; SA = single atrium; s/p = status post; SV = single ventricle; TA = tricuspid atresia; TAPVR = total anomalous pulmonary venous return; TGA = transposition of the great arteries; UVH = univentricular heart; VSD = ventricular septal defect.

Table 3. Preoperative Hemodynamic Profile[a]

Variable	
Pulmonary vascular resistance (Wood units)	2.5 (0.4–3.6)
Mean pulmonary artery pressure (mm Hg)	18 (6–26)
Systolic pulmonary artery pressure (mm Hg)	24 (10–38)
Pulmonary to systemic flow ratio	1.4 (0.45–3.1)
Systemic saturation (%)	81 (63–95)
Hemoglobin (g/dL)	18 (14–23)
Mean LV end-diastolic pressure (mm Hg)	10 (3–15)
Ventricular ejection fraction	0.56 (0.42–0.72)

[a] Data are shown as mean values with range in parentheses.

LV = left ventricular.

Table 4. Indications for Use of Adjustable Atrial Septal Defect

Indication	No. of Patients[a]
Increased PA pressure (>20 mm Hg) with normal calculated PVR	11
Increased PVR (>2.0 Wood units)	5
Reactive airway disease	6
Previously increased PVR	3
Small pulmonary arteries	10
Unknown PVR	11
Borderline ventricular function	9

[a] Many patients had more than one indication.

PA = pulmonary artery; PVR = pulmonary vascular resistance.

the aortic vent blood color. After stabilization, if the RAP was less than 17 mm Hg, the defect was slowly closed with the snare, with one hemaclip added at a time until the pressure reached 17 mm Hg. If the arterial oxygen saturation fell to less than 85%, the atrial communication was likewise made smaller. Intraoperative echocardiography was used in patients to document shunting across the ASD.

Results

Thirty-six patients underwent the modified Fontan procedure with placement of an adjustable ASD. The type of connection used and the additional surgical procedures performed are listed in Table 5. The average bypass time was 117 minutes (range, 73 to 200 minutes) and the average time of ischemia, 83 minutes (range, 55 to 157 minutes). Blood cardioplegia with systemic hypothermia to 24°C was used in all patients. Fourteen patients had the ASD closed with the snare in the operating room after coming off bypass and demonstrating acceptable hemodynamics with an RAP of less than 15 mm Hg. In the other 22 patients (9 with tricuspid atresia, 11 with univentricular heart, and 2 with pulmonary atresia with intact ventricular septum), the RAP was greater than 15 mm Hg after they were weaned from bypass, and the ASD was therefore left partially open. There was no significant difference in the preoperative hemodynamic profile be-

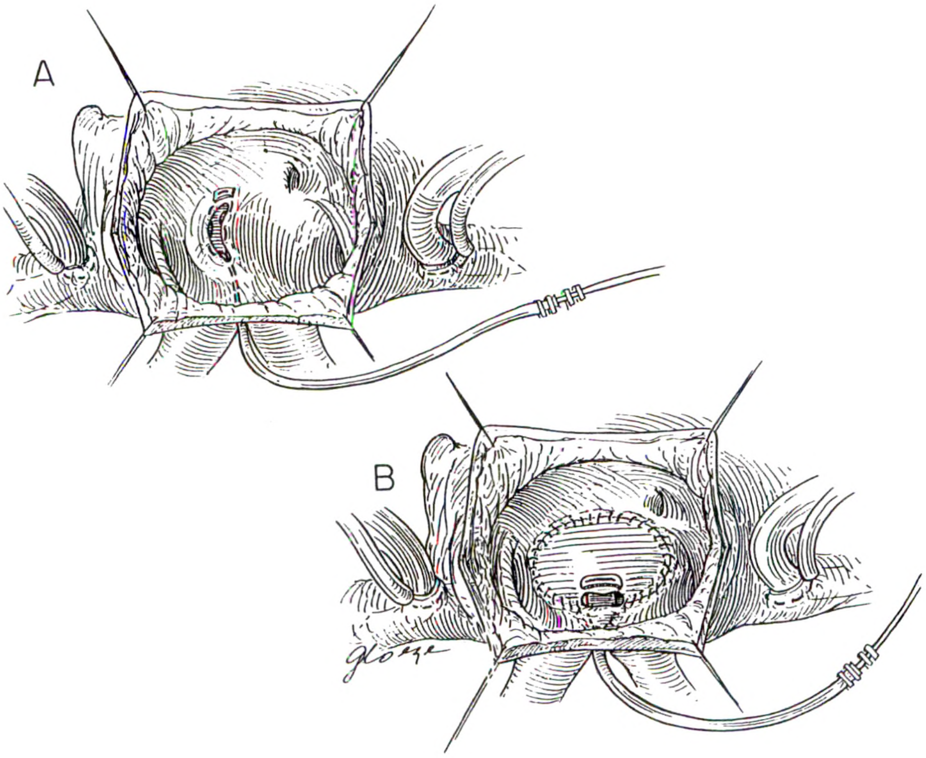

Fig 2. Adjustable atrial septal defect (ASD) in place with the snare. (A) If the native ASD is small, the adjustable ASD is put in the septum. A Prolene pursestring with a pericardial pledget is placed around the ASD. A single snare for closing is present. (B) If the ASD is large and a patch is required for its closure or for creation of an interatrial baffle, the ASD is placed at the edge of the Dacron patch. A pledget is used on the inner edge of the ASD to add strength to the closure by the Prolene suture.

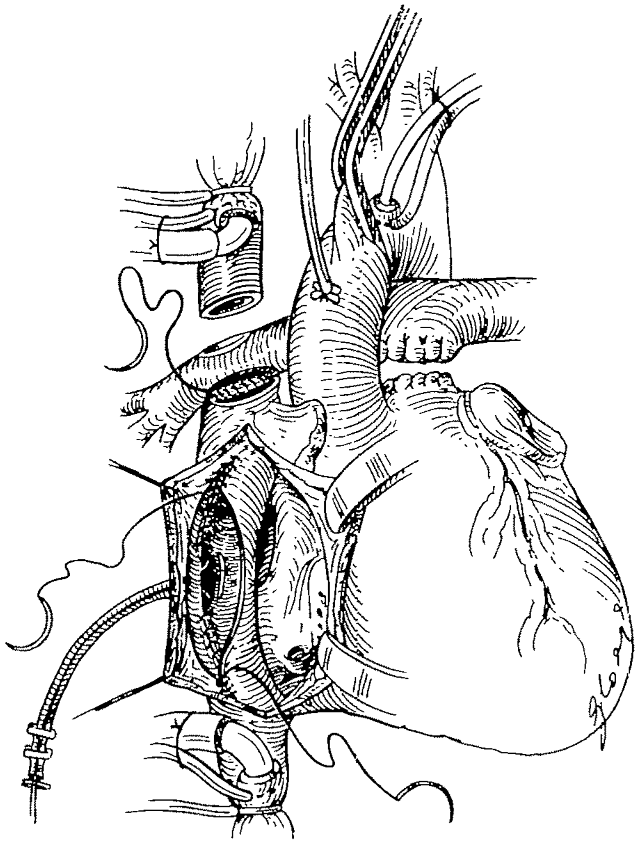

Fig 3. A completed cavopulmonary connection, or lateral tunnel Fontan procedure, with an adjustable interatrial communication between the tunnel and the left atrium. Currently, we prefer a more simple defect left in the edge of the patch, as shown, instead of the trapdoor technique, which we used initially. The Prolene suture is brought in through the lateral portion of the interatrial septum and secured to the edge of the atrial septal defect with 5-0 Prolene as shown. The Prolene is brought out through the interatrial septum and through a snare constructed of an 8F pediatric suction catheter. A pericardial pledget is placed where the sutures exit the heart. Care should be taken to prevent the ends of the Prolene from eroding through the skin.

tween patients who could and patients who could not tolerate closure of the ASD at the time of operation.

All patients received inotropic support when coming off bypass and in the postoperative period according to protocol. Low-dose dopamine hydrochloride (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was used in 32 patients and was supplemented by low-dose dobutamine hydrochloride (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) in 16 patients. Nine patients required moderate dosages (5 to 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) of dopamine, dobutamine, or both, and 4 patients required high doses of inotropic agents including isoproterenol and epinephrine. Thirteen patients received nitroglycerin or sodium nitroprusside, and 14 received prostaglandin E_1. In general, patients were weaned from dopamine after extubation.

Six patients required the use of a venous assist device postoperatively for hemodynamic support; 3 had an open adjustable ASD, and 3 had the ASD closed at operation. One of the latter patients had the ASD opened postoperatively. Eight patients were considered to be in unstable condition postoperatively because they required more than the usual amount of support and had signs of inadequate cardiac output. In general, patients with pulmonary hypertension and unstable hemodynamics were recognized immediately after they were weaned from bypass. However, in 4 patients who demonstrated acceptable hemodynamics in the operating room and had the ASD closed, elevated RAP developed in the postoperative period and necessitated inotropic and mechanical support.

The average time to extubation was 35 hours (range, 8 to 288 hours). Extubation resulted in a decrease in RAP of approximately 3 mm Hg. In general, this was accompanied by some improvement in clinical condition.

In 19 patients, the ASD was adjusted 1 day to 12 days postoperatively. It was made smaller in 6, closed in 10, and initially made smaller and then closed in 3. All 19 adjustments were done without cardiopulmonary bypass. Only one necessitated sternotomy; the other 18 ASDs were approached through the linea alba and successfully closed in the intensive care unit. Preadjustment and postadjustment data are presented in Table 6. One patient whose ASD was initially closed had the ASD opened during the postoperative period secondary to elevated systemic venous pressures. Three patients whose ASD had initially been left open had no adjustment of the ASD in the postoperative period. Two patients underwent ASD closure 3 months or more postoperatively.

Three patients had the ASD adjusted more than once in the postoperative period (see Table 6). Detailed data on 1 of these patients (patient 2) are found in Table 7. This patient had elevated preoperative pulmonary pressures as well as reactive airway disease. He underwent tightening of the adjustable ASD 14 hours postoperatively, as the RAP declined, and closure of the ASD on postoperative day 6, as the RAP fell again. Both adjustments were done at the bedside in the intensive care unit using local anesthesia. This patient demonstrated the progressive fall in PVR that can occur during the first few days.

Table 5. Surgical Procedures in 36 Patients Undergoing Placement of Adjustable Interatrial Communication as Part of Fontan Repair

Procedure	No. of Patients
Connection	
Direct RA–PA	15
RA–RV–PA	5
Lateral tunnel	16
Additional procedure	
Bidirectional Glenn shunt	23
PA reconstruction	15
Repair of TAPVR	6
AV valve repair	7
Damus-Kaye-Stansel	1

AV = atrioventricular; PA = pulmonary artery; RA = right atrium; RV = right ventricle; TAPVR = total anomalous pulmonary venous return.

Table 6. Summary of Data on 18 Patients Having Postoperative Adjustment of Atrial Septal Defect[a]

		ASD Open			ASD Tightened				ASD Closed					
Patient No.	Diagnosis	RAP (mm Hg)	LAP (mm Hg)	Oxygen Saturation (%)	RAP (mm Hg)	LAP (mm Hg)	Oxygen Saturation (%)	Time Postop (h)	RAP (mm Hg)	LAP (mm Hg)	Oxygen Saturation (%)	Time Postop (h)	Status at Discharge	Oxygen Saturation at Follow-up (%)
1	TA	12	. . .	87	. . .	. . .	. . .	. . .	17	. . .	92	136	Closed	94
2	TA	13	8	75	16	6	86	14	. . .	. . .	. . .	. . .	. . .	. . .
		9	. . .	92	. . .	. . .	. . .	. . .	12	. . .	98	140	Closed	97
3	TA	8	7	87	14	8	94	24	. . .	. . .	. . .	. . .	Open	94
4	TA	14	10	85	. . .	. . .	. . .	. . .	17	8	95	24	Closed	93
5	TA	8	. . .	86	. . .	. . .	. . .	. . .	10	. . .	95	24	Closed	91[b]
6	TA	14	. . .	86	. . .	. . .	. . .	. . .	16	. . .	92	168	Closed	93
7	UVH	12	7	92	. . .	. . .	. . .	. . .	15	6	98	48	Closed	96
8	UVH	13	6	83	16	6	91	72	. . .	. . .	. . .	. . .	Open	95
9	UVH	16	. . .	77	. . .	. . .	. . .	. . .	22	. . .	97	3.5 mo	Open	97[c]
10	UVH	16	9	78	18	10	82	120	. . .	. . .	. . .	. . .	Open	88
11	UVH	13	8	88	. . .	. . .	. . .	. . .	16	7	95	36	Closed	94
12	UVH	13	11	70	17	7	89	8	. . .	. . .	. . .	. . .	. . .	. . .
		12	. . .	91	15	. . .	98	96	. . .	. . .	. . .	. . .	Open	94[d]
13	UVH	12	. . .	88	. . .	. . .	. . .	. . .	15	. . .	94	120	Closed	Late death
14	UVH	15	8	78	17	7	86	36	. . .	. . .	. . .	. . .	Died	. . .
15	TA	10	7	85	. . .	. . .	. . .	. . .	14	5	91	6	Closed	93
16	UVH	14	9	77	. . .	. . .	. . .	. . .	16	7	94	24	Closed	95
17	PA-IVS	12	7	69	14	5	80	12	. . .	. . .	. . .	. . .	. . .	. . .
		13	. . .	80	. . .	. . .	. . .	. . .	16	. . .	90	288	Closed	92
18	PA-IVS	14	9	83	17	8	89	72	. . .	. . .	. . .	. . .	Open	98[e]
21	TA	11	8	88	. . .	. . .	. . .	. . .	14	6	94	48	Closed	94

[a] Seventeen patients underwent tightening or closure of the adjustable ASD under local anesthesia in the intensive care unit. One patient underwent delayed closure with the snare. [b] Echocardiography revealed a small patent ASD with a minimal right-to-left shunt. [c] This patient was readmitted 3½ months postoperatively for closure. [d] This patient had a patent ASD with minimal shunting; [e] The ASD was closed with placement of a transcatheter clamshell device.

ASD = atrial septal defect; LAP = left atrial pressure; PA-IVS = pulmonary atresia with intact ventricular septum; RAP = right atrial pressure; TA = tricuspid atresia; UVH = univentricular heart.

Table 7. Representative Data on Patient Requiring Several Adjustments of Atrial Septal Defect[a]

	Operating Room		Postoperative Day 1		Postoperative Day 6	
Variable	Open	Closed	Open	Tightened	Open	Closed
RAP (mm Hg)	15	19	13	16	9	12
LAP (mm Hg)	8	7	8	6	. . .	. . .
Arterial saturation (%)	92	100	75	86	92	98
Systemic blood pressure (mm Hg)	100	85	105	105	115	105

[a] Patient 2, a 7-year-old boy with tricuspid atresia and severe cyanosis preoperatively underwent a lateral tunnel procedure. He had critical pulmonary hypertension and could not be weaned from cardiopulmonary bypass without the adjustable ASD open. As the pulmonary hypertension decreased during the postoperative period, the adjustable ASD was able to first be tightened on postoperative day 1 and then closed on postoperative day 6 with maintenance of acceptable hemodynamics.

ASD = atrial septal defect; LAP = left atrial pressure; RAP = right atrial pressure.

There were three early deaths, which could not be directly related to the adjustable ASD. Two occurred in patients whose adjustable ASD was closed at the time of operation: one was related to an arrhythmia, and the other was due to ventricular failure. Attempts to reopen the ASD in this patient were successful, and there was temporary improvement in the patient's status, but multisystem failure ensued and the patient died. The third death occurred in a patient with a partially open ASD and low RAP in whom refractory supraventricular arrhythmias developed. The early mortality of 8.3% (3/36) in this high-risk group is not significantly different from the early mortality of 7% (16/228) in our entire Fontan series.

Pleural effusions necessitating chest tubes occurred in 12 of the 36 patients. The incidence is similar to that reported by us [22] in the past for a group of patients having a Fontan procedure who were not at increased risk.

Eight patients were discharged with the ASD still open. Two underwent delayed closure, 1 by use of the snare device (see next paragraph) and the other through transcatheter placement of a clamshell occlusion device at an outside institution. The other 6 patients are all doing well; they are in New York Heart Association class I or II and have an average systemic saturation of 88%. Two patients have minimal shunting by Doppler analysis with a patent ASD by two-dimensional imaging. Two patients have no shunting and no ASD visible on two-dimensional and Doppler echocardiography with normal systemic saturations.

One patient whose ASD was partially closed postoperatively was seen urgently 3½ months postoperatively with abrupt onset of cyanosis and increased right-to-left shunting as a result of reopening of the ASD, which was documented by catheterization. The patient was taken to the operating room where the sternum was reopened. It was found that the Prolene suture had been cut through by a hemaclip. The Prolene ends were retrieved, and the ASD was closed by ligating them without bypass. Saturation improved from 70% to 97% on room air, and the RAP increased from 16 to 22 mm Hg with closure of the ASD. This demonstrates the importance of using multiple clips to hold the Prolene in the polyethylene tube.

Another complication occurred in a patient in whom an infection related to the snare developed. This patient was seen 12 months postoperatively with a snare abscess, which necessitated open debridement and removal of the snare. He has subsequently done well. The snare abscess was related to a Teflon felt pledget used at the end of the snare. We have since abandoned the use of felt pledgets in the construction of an adjustable ASD and have had no other infectious complications related to the snare. To reduce the risk of infection, Betadine (povidone-iodine) solution is injected into the polyethylene tubing before the chest is closed.

In a mean follow-up of 24 months, there has been only one late death (3%). Twenty-seven patients are in New York Heart Association class I, and 5 are in class II. The average follow-up saturation for all patients is 94%.

Comment

The Fontan-Kreutzer procedure [7, 16] was proposed for the correction of complex lesions where a biventricular repair was impossible or could not be safely performed. It was soon recognized that this procedure provided only a partial physiological correction. Although the systemic arterial oxygen saturation could be raised to close to normal levels by separating the systemic and pulmonary venous returns, systemic venous hypertension of varying degree was an inevitable accompaniment of this operation. In the absence of an effective right ventricle, even slight increases in PVR could result in low cardiac output and poorly tolerated systemic venous hypertension.

An excessively elevated systemic venous pressure of greater than 20 mm Hg is associated with an increased early mortality. In addition, extravascular fluid accumulation and hepatic congestion cause long-term morbidity and mortality [13, 17, 19, 23, 24].

Early takedown of the Fontan procedure because of high venous pressure is also associated with a high mortality [17, 23, 24]. Various types of venous assist devices have been used to intermittently raise the systemic venous pressure and increase blood flow across the pulmonary vascular bed [8, 25–28].

The most common causes of an excessive elevation of

RAP are an increase in PVR and poor systemic ventricular or valvular function resulting in an elevation of left atrial pressure. Obstruction to the pulmonary arteries or pulmonary veins and increased systemic–pulmonary artery collateral flow can also contribute to an increased RAP. Careful selection of patients and a meticulous operation, which includes optimal myocardial protection, an unobstructed systemic venous pathway to the pulmonary vascular bed, and the correction of associated anomalies, are essential to avoid a high mortality and morbidity.

The original criteria of Fontan and associates [16] have been extended to include higher-risk patients. We have considered a PVR of greater than 4 Wood units as a contraindication to a Fontan procedure, and a PVR of 2 to 4 units is considered a significant risk factor. Patients with pulmonary hypertension are also at increased risk even if the calculated PVR is normal. This combination is seen in patients with excessive pulmonary blood flow because of a large shunt and in patients with mitral atresia and a restrictive atrial communication. If the pulmonary artery pressure is more than 25 mm Hg or if the transpulmonary gradient is greater than 15 mm Hg, these patients are considered at high risk for a Fontan procedure.

Patients with major associated lesions such as moderate or severe atrioventricular valve regurgitation or subaortic stenosis as well as those just described fall into the high-risk group. One option for this group is to plan a staged approach in which a bidirectional Glenn shunt is performed and associated lesions are corrected. The patients are then reevaluated after approximately 1 year and reconsidered for a Fontan procedure. A second option for patients with borderline criteria is to perform the Fontan procedure with a residual communication left between the systemic venous pathway and the left atrium for partial decompression of the right side.

This partial Fontan operation was described by us [29] in 1988. It allows a controlled right-to-left shunt of up to one third of the systemic venous return. This results in an arterial oxygen saturation of 85% with a relatively lower RAP and improved systemic cardiac output. This partial Fontan is particularly suited to the patient with an elevated PVR or pulmonary artery pressure. The PVR can be transiently elevated in some patients postoperatively because of pulmonary vasospasm. This may be related to the endogenous release of catecholamines, the administration of inotropic agents, noxious stimuli such as pain, and an increased flow from systemic to pulmonary artery collaterals. The ability to reduce the RAP with an adjustable ASD would avoid the effects of systemic venous hypertension and improve the systemic cardiac output until the PVR falls to normal levels. At this point, the ASD could be closed with the snare device. Our preliminary experience [30–32] with the snare-controlled device has been previously reported.

This concept of the partial Fontan procedure has been applied using a fenestration in the baffle with subsequent transcatheter closure depending on the findings at cardiac catheterization [33]. The required size of this fenestration can be difficult to predict, as widely varying amounts of flow can occur with small changes in the size of the defect and with variations in the gradient between the right and left atria. The use of the snare-controlled adjustable ASD allows staged closure of the ASD depending on the hemodynamics rather than complete closure, which occurs with the transcatheter technique. The snare can be exposed under the lower part of the incision under local anesthesia, giving rapid control of the right-to-left shunt without moving the patient to the cardiac catheterization laboratory. The snare-controlled adjustable ASD is simple to insert and can be used both with the right atrium–pulmonary artery connection (see Fig 2) and with the total cavopulmonary connection (see Fig 3).

A reduced systemic venous pressure during the early postoperative course can be associated with a reduced incidence of pleural and pericardial effusions, as reported by Bridges and colleagues [33]. In the present series, the incidence of pleural effusions was similar to that reported by us [22] previously. The group described in the current report, however, was at increased risk.

It could be argued that a communication should routinely be left in all Fontan procedures to lower the RAP in the early postoperative period. We now do this in all Fontan procedures and make the decision about closing the ASD after the patient has been weaned from bypass. If the patient was at increased risk preoperatively, the ASD is left open to be closed postoperatively in the intensive care unit provided the RAP is less than 15 mm Hg. In older children and in patients with excellent hemodynamics, the ASD can be closed in the operating room. We have used the ASD routinely in infants less than 2 years of age and particularly those in the first year of life, as venous hypertension is not well tolerated in this age group. The ASD is left open and can be closed 2 or 3 days postoperatively or is left open for the patient to outgrow. The ASD can close spontaneously late postoperatively in these patients as the result of a buildup of pseudointima-like tissue at the edges of the ASD.

Use of the ASD appears to be relatively safe. We have had two adverse events related to the snare in this series. In 1 patient, the ASD reopened late postoperatively. This occurred because only a single clip was used to fix the snare after adjustment of the ASD and is preventable. Currently, several hemaclips are applied without excessive force to the polyethylene tube to prevent the clip from cutting through the Prolene suture. In addition, we no longer use the trapdoor technique; instead we use the technique shown in Figure 3, which permits a more secure closure of the ASD.

The second adverse event was an infection, which is of particular concern with the use of a foreign body. In this patient, the snare was used to close the ASD in the catheterization laboratory. A Teflon felt pledget was the focus of infection. Removal of the felt pledget and polyethylene snare led to prompt resolution of the infection. We now avoid felt pledgets and fill the polyethylene tube with Betadine to avoid infection.

The results of the Fontan procedure are critically related to the degree of elevation of the systemic venous pressure in the postoperative course. This experience has shown that a controlled right-to-left shunt during the period of transient elevation of the RAP may provide improved cardiac output, prevent excessive extravascular fluid accumulation, and yet maintain an adequate arterial oxygen saturation. The snare control allows adjustment of the ASD size in the postoperative period. This type of partial Fontan results in a flexible operative procedure that can be adjusted depending on the postoperative hemodynamic state. It may allow patients at increased risk to undergo the Fontan procedure with a reduced mortality and morbidity.

Conclusion

An adjustable ASD can be incorporated safely into a Fontan operation. The ASD can be snared closed in the operating room after cardiopulmonary bypass depending on the RAP or in the postoperative period as the PVR decreases; closure is usually accomplished through the linea alba without sternotomy or bypass. The use of an adjustable ASD results in a decrease in RAP and systemic venous engorgement while improving cardiac output. In some patients undergoing a modified Fontan procedure, the PVR remains higher than normal, and long-term use of a restrictive ASD results in good cardiac output with an acceptable oxygen saturation. Unlike the fenestration technique, the snare-controlled adjustable ASD allows staged closure of the ASD depending on the hemodynamics rather than complete closure, which is necessary with the transcatheter method.

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DISCUSSION

DR WILLIAM G. WILLIAMS (Toronto, Ont, Canada): I congratulate Dr Laks on this excellent presentation and applaud his conception of the idea of the fenestrated Fontan. I am embarrassed to admit he first told us about it several years ago, and we have used it only in the last 6 months. I think he probably understates the efficacy of this concept of leaving a controlled shunt from the right to the left atrium.

My associates and I have elected not to use the snare technique for fear of infection and have accepted a fixed size with the intention of eventual closure with a catheter device. However, on occasion, we have regretted not being able to adjust the size of the fenestration. Based on your experience, how often do you have to adjust the defect to fine tune the hemodynamics in the postoperative course?

DR LAKS: Thank you, Dr Williams. The need for adjustment really has evolved over time, and initially we thought that every patient should leave the hospital with the atrial septal defect (ASD) closed. Now we believe that most of these defects, once they are quite restrictive, will close on their own and therefore do not all need to be closed completely. The patients in whom this is most critical are usually the sickest patients in whom the right atrial pressure is high in the operating room, and the ASD is left completely open. In these patients, we sometimes see quite a rapid fall in the pulmonary vascular resistance in the intensive care unit, resulting in a drop in saturations. If the saturations decrease to less than 82%, we have to snare the ASD. In the majority of patients, the ASD is made more restrictive in the operating room, and it is not necessary to adjust it in the intensive care unit.

In regard to the size of the hole and the issue of control of right-to-left shunting, we have always been concerned that if we make a fixed hole and it is not big enough, then we will not have the benefit from it. If it is too big, a catastrophic rise in right-to-left shunting may occur, which will cause serious enough desaturation that the patient will have to be rushed to the catheterization laboratory or the operating room for closure.

If you take one third of the systemic venous return and allow it to bypass the pulmonary circulation, and if you are going to have an arterial oxygen saturation of 85%, you need about a 33% right-to-left shunt. This would provide the maximal benefit from the partial Fontan with the maximal advantage in terms of oxygen saturation. As size increases, the amount of flow can double or triple. In a report from Boston, some children who were 1 year of age had a 4-mm hole, and some children 9 years of age had a 5-mm hole. From a physiological point of view, it is hard to understand how the hole could be big enough to be effective and yet controllable at the same time.

We studied this with an experimental device developed in the laboratory by Dr Pearl and our group to see what size hole and what relative pressure gradient, at different hematocrits, would determine the amount of shunting or flow across such a hole. In the case of the 4-mm hole, as the gradient changes from 5 to 10 mm Hg, the flow across the hole increases from 546 to 879 mL/min. Depending on the hemodynamic status and the pulmonary vascular resistance, huge changes in flow can occur across the ASD. We do not think it practical to take a chance on that and prefer to have some ability to control the size of the hole as the hemodynamics change.

DR ROSS M. UNGERLEIDER (Durham, NC): There have been a number of valuable contributions in the last several years that have really encouraged us to perform Fontan procedures earlier in life: the use of the bidirectional Glenn shunt, the fenestrated Fontan procedures, and this contribution of yours, Dr Laks. Have your results with this technique encouraged you to do essentially complete Fontan procedures in complex anatomy where previously you might have been tempted to do a bidirectional Glenn to give the patient an opportunity to have a partial Fontan with the idea of completing the Fontan later? With this technique, you can perform the entire surgical procedure as one stage, knowing that you can complete it gradually in the postoperative period.

Are there patients to whom you would recommend the bidirectional Glenn shunt and then a second operation, or are you using this snare so that you can do a complete operative correction in every patient and then adjust the remainder of the physiology postoperatively?

DR LAKS: That is a very good question, and the answer is evolving. We have always thought that the Fontan procedure, because it is not a very physiological procedure, should be applied only under the most optimal circumstances. If you use the Fontan procedure for a patient who has a high resistance or poor function, the condition of that patient can be made even worse than it was before the operation. We do stratify the patients. For those who have significant risk factors, we prefer the bidirectional Glenn shunt to correct those factors and then a reassessment as to whether we can do the Fontan; for those who have moderate or mild associated risk factors, we do the Fontan with the snare-controlled ASD.

We believe that there is a particular application for this in very young patients because, although the hemodynamics may be optimal, they tolerate venous hypertension less well than older patients. Because they have a large growth spurt and will outgrow the ASD, we think that an ASD should be incorporated as a standard part of every Fontan procedure in patients less than 2 years old, and particularly in those less than 1 year old. They tolerate the ASD well with very little hepatomegaly, very little accumulation of edema, and much lower right atrial pressures.

DR FRANK L. HANLEY (Boston, MA): My colleagues and I have used the fenestrated Fontan procedure in a similar group of high-risk patients. It would be fair to say that our knowledge of how large to make the fenestration is evolving. In the patients less than 2 years of age, we have routinely used a 4-mm punch in the baffle and have not gotten into any trouble with a nonrestrictive connection. Clearly, a nonrestrictive connection is catastrophic in that the two atrial pressures equalize, resulting in no pulmonary blood flow. In older patients, the fenestration is made slightly larger, up to 5 mm in diameter. One potential disadvantage of the fenestration is that it is a fixed communication. Our experience in more than 50 patients, however, is that we have not found it necessary to make acute adjustments in the size of the fenestration. The most attractive advantages of the fenestration are that it is technically simple to perform, is easily reproducible, and does not communicate with the outside environment.

Our usual protocol is to take the patient to the catheterization laboratory before discharge and perform a diagnostic catheterization by temporarily closing the hole with a balloon while measuring various hemodynamics including cardiac output as well as measuring mixed venous and arterial oxygen saturations. If the hemodynamics seem favorable, the hole is closed permanently at catheterization with a device. If the hemodynamics are not favorable, the patient is sent home with the fenestration in place and reevaluated 3 months later.

DR LAKS: Dr Hanley, what you are doing clearly works very well, and your results have been superb. I think that if you took the vast majority of patients who are good candidates for the Fontan procedure and added a restrictive opening, they would all benefit in one way or another because they would have a lower right atrial pressure. If you take only the highest-risk groups, particularly those with increased pulmonary resistance or a history of pulmonary hypertension, which makes the pulmonary vascular bed more reactive, you are faced with the critical decision of how large to make the ASD. It must be big enough to avoid a low cardiac output state but not so large that severe desaturation occurs. It is in that particular group that I am concerned about the inability to control the ASD. A single fixed ASD may not be big enough to make a difference or may be so big that desaturation occurs. I think that the experience with the high-risk groups gives us some indication as to ASD size. As you pointed out, however, when you make the 4-mm or 5-mm hole, it is a less physiologically controlled instrument than one that can be adjusted according to the hemodynamics on an ongoing basis.

We have had several patients in whom the changes have been profound. One patient had very hypoplastic pulmonary arteries and in the previous year would have had a bidirectional Glenn shunt, as Dr Ungerleider mentioned. We did very extensive reconstructions. The hematocrit in this patient was around 0.80, and pulmonary blood flow was very low. Postoperatively, with the ASD open, the patient had a saturation of around 80% with a right atrial pressure of 16 mm Hg, so we left the ASD open. Within 24 hours, the right atrial pressure had dropped to 13 mm Hg, and the saturation had decreased to 70%. Therefore we had to rapidly partially close the ASD. As we closed it, the saturation rose into the 80 mm Hg range and the right atrial pressure, to 15 mm Hg. Six days later, the right atrial pressure had again dropped to 13 mm Hg, and we were able to almost completely close the ASD with a saturation of 94% and a right atrial pressure of 15 mm Hg. Such dramatic changes can cause a serious problem with a single fixed ASD.

If the ASD is made big enough to allow adequate right-to-left shunting when the patient is in trouble, which is in the first 24 hours postoperatively, then as the resistance drops, you may find that pulmonary blood flow is inadequate. The patient will either have to be rushed to the catheterization laboratory, where your only option is to close the ASD completely, or have to be taken to the operating room, where, again on bypass, the risks and mortality go up significantly. For that type of patient, the adjustable ASD allows fine adjustment so that you can respond to the hemodynamics.

TNM Staging and Long-Term Follow-up After Sleeve Resection for Bronchogenic Tumors

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From 1960 to 1989, 145 patients (132 men and 13 women) with a mean age of 60.3 years underwent sleeve lobectomy or sleeve resection of a main bronchus for a bronchogenic tumor. Squamous cell carcinoma was predominantly found (116 patients, 80.0%), followed by carcinoid tumor in 13 patients (9.0%). Postoperative staging was: stage I, 61 patients (42.1%); stage II, 47 (32.4%); stage IIIA, 33 (22.8%); and stage IIIB, 4 (2.7%). Thirty-day mortality was 4.8% (7 patients). Follow-up was complete except for 1 patient who was lost to follow-up 4 years after operation. For the whole group, 5-, 10-, and 15-year survival rates were 49%, 37%, and 18%, respectively. Better survival was noted in patients with carcinoid tumor and squamous cell carcinoma. Considering 112 patients with T2 and T3 squamous cell carcinoma, 5- and 10-year survival rates for N0 disease (52 patients) were 59% and 47%, for N1 disease (51 patients) 21% and 0%, and for N2 disease (9 patients) 44% and 0%. Differences between N1 and N2 disease were not statistically significant. Survival after sleeve resection is best for carcinoid tumors and squamous cell carcinoma with negative nodes. Presence of N1 or N2 disease significantly worsens prognosis, with no 10-year survivors and no difference between N1 and N2 status.

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Bronchoplastic procedures including sleeve lobectomy were initially introduced for patients whose pulmonary function was insufficient to tolerate pneumonectomy [1]. In more recent years, sleeve lobectomy has evolved as an alternative to pneumonectomy in carefully selected cases of bronchial carcinoma, especially for centrally located lesions [2, 3].

In 1981 we [4] reported the results of 50 patients with bronchial carcinoma who underwent right upper sleeve lobectomy. In a subgroup of 22 patients with positive hilar lymph nodes no 5-year survival was found. In the present study long-term results were analyzed in 145 patients undergoing sleeve resection during the years 1960 to 1989. They represent our total experience with sleeve resection for bronchial tumors. Special attention was given to long-term survival in relation to lymph node involvement.

Material and Methods

Patient Material

During the years 1960 to 1989, 145 patients, 132 men (91.0%) and 13 women (9.0%), underwent complete sleeve lobectomy or sleeve resection of a main bronchus. Wedge excisions or other bronchoplastic procedures (eg, bronchotomies) and sleeve pneumonectomies were excluded. Mean age was 60.3 ± 11.8 years (range, 22 to 80 years).

Indications for sleeve resection were central localization of the tumor in 122 patients (84.1%), presence of metastatic lymph nodes near a main bronchus in 4 patients (2.8%), and compromised pulmonary function precluding pneumonectomy in 19 patients (13.1%).

Preoperatively, 128 patients (88.3%) underwent routine cervical mediastinoscopy, 4 of which were repeat mediastinoscopies, which showed no metastatic mediastinal lymph nodes except in 2 patients who had limited ipsilateral intranodal disease. In 17 patients (11.7%) no preoperative cervical mediastinoscopy was performed. Most of these had carcinoid tumors. During operation hilar and mediastinal lymph nodes were routinely sampled in all patients.

Procedures performed are listed in Table 1. Sleeve resection of the right upper lobe was the most common procedure, performed in 117 patients (80.8%). Six patients (4.1%) underwent sleeve resection of a main bronchus without resection of pulmonary parenchyma. Concomitant procedures on the pulmonary artery were wedge excision in 4 patients, sleeve resection in 4, and transection and reanastomosis to facilitate bronchial approach in

Table 1. Different Types of Sleeve Resections

	Lobectomy			
Side	Upper	Lower	No Parenchyma Resected	Total
Right	117	2	2	121
Left	12	8	4	24
Total	129	10	6	145

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Table 2. Histology

	Patients	
Histology	No.	%
Squamous cell carcinoma	116	80.0
Carcinoid	13	9.0
Adenosquamous carcinoma	5	3.4
Adenocarcinoma	4	2.7
Small cell carcinoma	2	1.4
Large cell anaplastic	1	0.7
Adenocystic carcinoma	1	0.7
Sarcoma	1	0.7
Mesenchymal (+ squamous cell carcinoma)	1	0.7
Combined tumor	1	0.7

Table 4. Early and Late Postoperative Complications

Complication	No. of Patients
Early postoperative complications (37.6%)[a]	
Air leakage (>3 days)	16 (17.4%)
Atelectasis	9 (9.8%)
Respiratory insufficiency	4 (4.3%)
ARDS	1 (1.1%)
Empyema	1 (1.1%)
Gastrointestinal bleeding	3 (3.2%)
Anastomotic dehiscence	1 (0.7%)
Late postoperative complications (8.9%)[b]	
Bronchostenosis	13 (8.9%)

[a] 92 patients from 1980. [b] All patients.

ARDS = adult respiratory distress syndrome.

1 patient undergoing a sleeve resection of the right main bronchus.

Histological diagnosis is given in Table 2; squamous cell carcinoma (80.0%) and carcinoid (9.0%) were most frequently found. All 145 patients were staged according to the most recent TNM classification [5]. Results of postoperative staging are shown in Table 3. Patients with N1 disease were subdivided into N1 distal and N1 proximal disease, this last group including patients with metastatic lymph nodes between the upper lobe and main bronchus.

Chemotherapy was given to the 2 patients with small cell carcinoma, and radiotherapy was given to 20 patients (13.8%). Indications for radiotherapy were N2 disease and extranodal N1 disease; a positive resection margin was the indication for radiotherapy in 4 patients, and in another 1 radiotherapy was indicated for a T4 tumor involving the trachea.

Methods

Follow-up data were gathered from the files of the patients followed up in our institution, and for the others information was obtained from the referring pulmonary physicians by a questionnaire.

Survival rates were calculated and analyzed using program 1L of BMDP Statistical Software (Los Angeles, CA, 1990). Survival curves were obtained according to the Kaplan-Meier actuarial method. Survival between subgroups was compared with the generalized Savage (Mantel-Cox) test or the trend from the Tarone-Ware test. Chi-square test for rectangular contingency tables (with continuity correction where necessary) was used for comparing qualitative survival data. Relative risks were calculated according to the Cox proportional hazards model.

Results

Operative Mortality and Complications

Operative mortality was 4.8% (7 patients). One patient died during operation because of ventricular fibrillation. Six patients died postoperatively. Causes were bronchovascular fistula in 1 patient, sepsis in 2, respiratory insufficiency in 2, and gastrointestinal bleeding in 1.

Table 3. Postoperative Staging According to TNM Classification [5]

Stage	TNM Status	No. of Patients
I	T2 N0 M0	61 (42.1%)
II	T2 N1 M0	47 (32.4%)
IIIA		33 (22.8%)
	T1 N2 M0	1
	T2 N2 M0	11
	T3 N0 M0	10
	T3 N1 M0	8
	T3 N2 M0	3
IIIB		4 (2.7%)
	T4 N1 M0	3
	T4 N2 M0	1

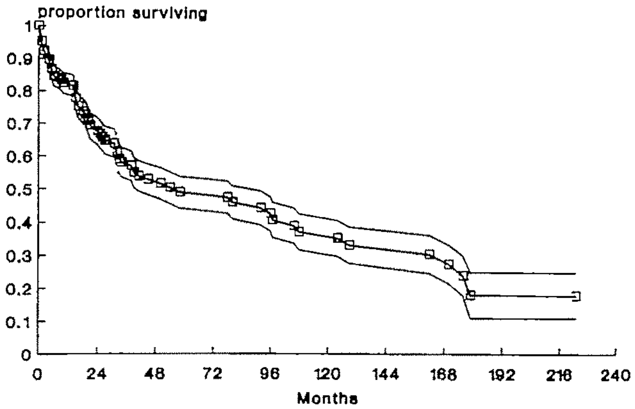

Fig 1. Actuarial survival curve with one standard deviation for the 145 patients undergoing sleeve resection.

Table 5. Causes of Death in 74 Patients (51%) Dying During Follow-up[a]

Cause	Patients No.	Patients %
Operative mortality	7	4.8
Local recurrence	9	6.2
Metastases	25	17.2
Local recurrence + metastases	10	6.9
Cardiac	11	7.6
Other	10	6.9
Unknown	2	1.4

[a] Percentages are calculated for the whole group.

Table 6. Survival Rate According to Stage for Patients With Squamous Cell Carcinoma[a]

Stage	No. of Patients	5-year	10-year	15-year
I	50	0.59 ± 0.07	0.47 ± 0.08	0.15 ± 0.09
II	45	0.22 ± 0.10	0.00 ± 0.00	0.00 ± 0.00
IIIA	17	0.42 ± 0.14	0.21 ± 0.16	0.21 ± 0.16
IIIB	4	0.38 ± 0.29	0	0

[a] Survival rates are given with standard deviation.

Positive resection margins were present in 10 patients (6.9%), of whom 4 were given radiotherapy, 3 underwent completion pneumonectomy within several weeks (before 1980), and 3 were given no specific treatment.

Early postoperative complications could only be examined with certainty in the patients operated on from 1980 on (n = 92); these are listed in Table 4, together with the late complications for all patients. There was no incidence of late bronchopleural or bronchovascular fistula. Ten of the 13 patients with bronchostenosis had completion pneumonectomy; the 3 others underwent bronchoscopic dilation.

Local recurrence was observed in 26 patients (17.9%). Five of these underwent completion pneumonectomy (mean interval from operation, 10.8 months; range, 2 to 17 months), 19 died with local recurrence, 10 of whom had also metastatic disease (mean interval from operation, 42.9 months; range, 2 to 124 months), and 2 patients are still alive 29 and 39 months after operation.

Survival Data

Follow-up was complete for the 138 operative survivors except for 1 patient who was lost to follow-up 4 years after operation. Mean follow-up was 47.9 months. Maximum follow-up was 223 months. All survival data include operative mortality.

Actuarial survival for all 145 patients is depicted in Figure 1. Median survival time was 58 months; 5-year survival rate, 0.49 ± 0.05; 10-year survival rate, 0.37 ± 0.05; and 15-year survival rate, 0.18 ± 0.07. During follow-up 74 patients (51%) died. Causes of death are listed in Table 5. Seventy-one patients (49%) are alive, 63 (43.4%) without evidence of disease. Two patients (1.4%) are alive with local recurrence, 3 (2.1%) with metastases, and 3 (2.1%) with another tumor. No difference in survival was observed between patients with compromised or noncompromised lung function ($p = 0.24$).

Survival was studied according to the histological type. For the 13 patients with carcinoid tumor the 15-year survival rate was 1.00, which shows the clinically benign behavior of these tumors. The 5-year survival rate for the patients with adenocarcinoma and adenosquamous carcinoma (n = 9) was 0.25 ± 0.22. The actuarial survival curve for the 116 patients with squamous cell carcinoma is given in Figure 2. Five-, 10-, and 15-year survival rates in this group were 0.46 ± 0.05, 0.32 ± 0.06, and 0.13 ± 0.06, respectively.

There was no difference in survival between left-sided or right-sided sleeve resections ($p = 0.89$) and between

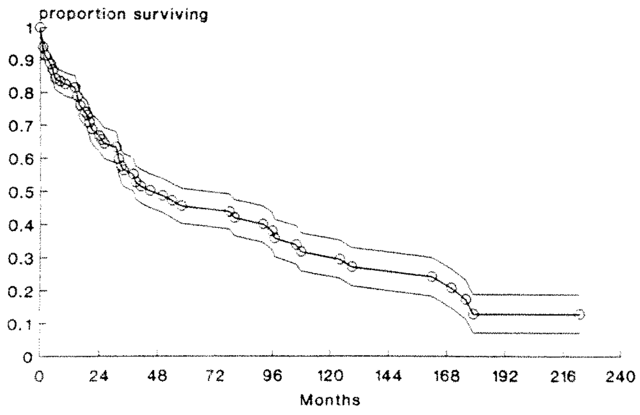

Fig 2. Actuarial survival curve with one standard deviation for the 116 patients with squamous cell carcinoma.

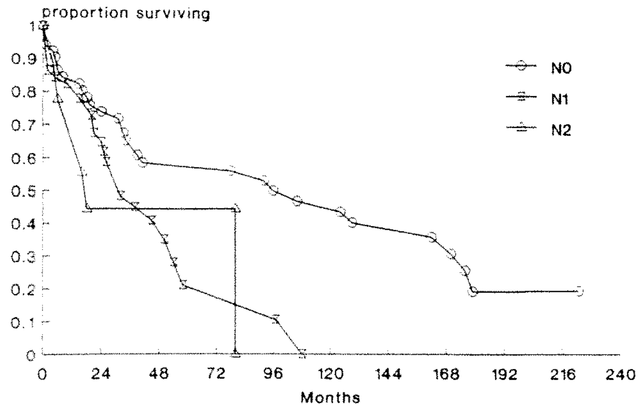

Fig 3. Actuarial survival curves according to lymph node involvement for 112 patients with T2 and T3 squamous cell carcinoma subdivided into N0, N1, and N2.

Table 7. Survival Rate According to Lymph Node Involvement for 112 Patients With T2 and T3 Squamous Cell Carcinoma[a]

N Status	No. of Patients	5-year	10-year	15-year
N0	52	0.59 ± 0.07	0.47 ± 0.08	0.19 ± 0.08
N1	51	0.21 ± 0.09	0.00	0.00
N2	9	0.44 ± 0.17	0.00	0.00

[a] Survival rates are given with standard deviation.

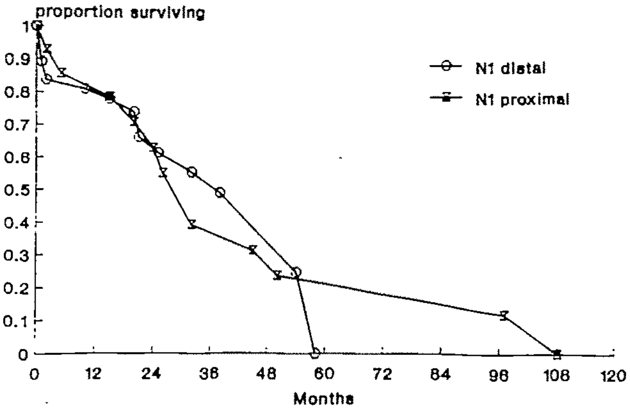

Fig 4. Actuarial survival curves for the 51 N1 patients with T2 and T3 squamous cell carcinoma subdivided into N1 proximal and N1 distal disease.

sleeve resections of upper or lower lobes ($p = 0.77$) in these patients.

Survival according to stage for the patients with squamous cell carcinoma is given in Table 6. Stage I patients did significantly better than the others ($p = 0.01$), but there was no difference between stages II, IIIA, and IIIB (the number of patients in the last two groups was small).

Survival according to lymph node involvement (Fig 3) was calculated for 112 patients with T2 and T3 squamous cell carcinoma. The results are given in Table 7. There were no 10-year survivors among the patients with N1 and N2 disease. There was a significant difference between patients with N0 disease and N1 or N2 disease ($p = 0.008$) but no difference between N1 and N2 disease ($p = 0.85$). Subdividing the 51 patients with N1 disease into N1 proximal and N1 distal disease (Fig 4) showed no significant difference in survival between these two groups ($p = 0.56$). The relative risk related to mortality of N1 versus N0 disease was 2.15 and of N2 versus N0 disease, 2.35.

Causes of death according to N category are given in Table 8. Looking at the different N groups, there was no significant difference in local recurrence rate ($p > 0.25$). However, comparing distant metastases between N0 disease and the two other groups, the difference reached significance ($0.01 < p < 0.025$).

Comment

In 1952 Allison performed the first sleeve resection for a bronchogenic carcinoma, although no details of the procedure were reported [6, 7]. After the report of Paulson and associates [8] in 1970, describing 54 patients with bronchoplastic procedures, sleeve lobectomy gained wider acceptance as therapy for centrally located benign tumors and tumors of low-grade malignancy, and also for patients with bronchogenic carcinoma and limited pulmonary reserve who could not tolerate pneumonectomy [9].

Many authors [2, 3, 7, 9–17] have reported their results with sleeve resection, and in more recent years it has been shown that sleeve lobectomy can be an alternative to pneumonectomy in carefully selected patients, even with good pulmonary function. Sleeve resection now offers both conservation of pulmonary function and enhanced resectability [18]. The reimplanted lobe contributes significantly to the overall pulmonary function [15]. The results of sleeve lobectomy compare favorably with those of pneumonectomy [13], and Faber [17] concluded in 1987 that sleeve lobectomy should be the operation of choice whenever technically feasible.

Naruke [2] analyzed the results of several reports dealing with bronchoplastic procedures. Operative mortality varied between 0% and 11.4%. The incidence of anastomotic strictures ranged between 0% and 17.0%, of bronchial fistula between 1% and 11.4%, and of local recurrence between 2% and 51%. Our operative mortality and early and late complications are in accordance with these data.

Table 8. Causes of Death According to N Category in Patients With T2 and T3 Squamous Cell Carcinoma

N Status	Operative Mortality	Local Recurrence	Metastases	Local Recurrence + Metastases	Cardiac	Other	Unknown	Total
N0	3	4	5	2	7	8	1	30
N1	4	3	11	4	2	2	1	27
N2	0	1	1	3	1	0	0	6
Total	7	8	17	9	10	10	2	63

Table 9. Survival After Bronchoplastic Resection According to Lymph Node Status

First Author	Year	No. of Patients	5-year survival			10-year survival		
			N0	N1	N2	N0	N1	N2
Van den Bosch [4]	1981	50	64	0	. . .	. . .	. . .	. . .
Ayabe [12]	1982	34	30		11[a]	. . .	. . .	. . .
Firmin [13]	1983	90	71	17	. . .	49	10	. . .
Belli [14]	1985	19	50		10[a]	. . .	. . .	. . .
Deslauriers [15]	1986	72	67	60	. . .	. . .	. . .	. . .
Vogt-Moykopf [16]	1986	248	37	30	0	. . .	. . .	. . .
Naruke [2]	1989	111	50	46	33	. . .	. . .	. . .
Present study[b]	1990	112	59	21	44	47	0	0

[a] Percentage for combined N1 and N2 patients. [b] Only the patients with T2 and T3 squamous cell carcinoma are considered here.

The relationship between long-term survival after sleeve resection and lymph node involvement remains controversial. In 1981 no 5-year survivors were found among patients with positive hilar lymph nodes [4]. Since then, numerous reports have been presented dealing with survival after bronchoplastic resection according to lymph node status. The most important are summarized in Table 9. Five-year survival rate in patients with N1 disease ranges from 0% to 60%. Different kinds of operations (wedge excision, sleeve resection, other bronchoplastic procedures) and different histologic types result in substantial heterogeneity in these papers.

In 1983 Firmin and co-workers [13] reported in detail the long-term survival after sleeve lobectomy for squamous cell carcinoma according to lymph node involvement. Five- and 10-year survival rates were 71% and 49% for patients with N0 disease (n = 53), and 17% and 10% for patients with N1 disease (n = 23), respectively. In the present report, in a group of 112 patients with T2 and T3 squamous cell carcinoma, the 5- and 10-year survival rates for N0 disease (n = 52) were 59% and 47%, and for N1 disease (n = 51) 21% and 0%, respectively. These results are very similar.

The N1 subgroup was divided into patients with proximal and distal N1 involvement, but no statistically significant difference was observed. There was also no difference between N1 and N2 disease. Although the number of patients with N2 disease was rather small, these patients did no worse than those with N1 disease. A possible explanation could be that the patients with N2 disease were given radiotherapy postoperatively.

When analyzing late death, patients with N1 and N2 disease mainly died of metastatic disease in contrast to patients with N0 disease. So, in this setting the question remains whether patients with N1 disease should be regarded as having systemic disease and be treated accordingly when experience with neoadjuvant therapy grows [18, 19].

In conclusion, survival after sleeve resection is best for carcinoid tumors and squamous cell carcinoma with negative nodes. Presence of N1 or N2 disease significantly worsens prognosis, with no 10-year survivors and no difference between N1 and N2 status.

We are greatly indebted to H. J. Gelissen, MD, and A. L. Schaepkens van Riempst, MD, who introduced the technique of sleeve resection in our institution; to M. J. Suttorp, MD, and I. Broeders, MD, for their help in data management; and to R. Ruelle and M. Van Duysen for typing the manuscript.

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DISCUSSION

DR ROBERT J. GINSBERG (New York, NY): I was asked to comment on this well-presented paper by Dr Van Schil and associates, and I have a few questions. The authors did not describe how accurately they staged the mediastinum in each case. Were these really N1 patients? In the first decade, they had no N1 or N2 patients survive beyond 5 years. Did they look at the second decade of patients where they did have 5-year survivors to see if there was some sort of difference between the two groups of patients? To have no 10-year survivors of resected N1 or N2 disease surprises me.

So the questions that I have are: how did you stage the mediastinum at operation; was it routine in every case? And in the latter group of patients where you finally do have 5-year survivors with N1 and N2 disease, can you predict that there will be 10-year survivors as well?

DR VAN SCHIL: Regarding the first question, 88% of patients had preoperative cervical mediastinoscopy. Mediastinoscopy was not performed in the patients with carcinoid disease. But nearly all the others had preoperative mediastinoscopy, so we think that we preoperatively staged the mediastinum correctly. During operation samples of hilar and mediastinal lymph nodes were routinely taken.

Regarding the second question, we looked at the causes of death according to the N category of the patients. We found no significant difference in local recurrence rate between N0, N1, or N2 disease; however, when we compared the rate of distant metastases between N0 disease and the two other groups, the difference reached statistical significance. So patients with N1 disease can be regarded as having systemic disease, and this probably determines survival. We did not find any characteristic difference between the last group and the first group of patients, or between the patients before 1980 and the other ones.

DR JEAN DESLAURIERS (Sainte-Foy, Qué, Canada): This well-presented paper looks at a large number of sleeve resections done for treatment of bronchogenic carcinoma. Dr Van Schil and associates report low operative morbidity/mortality and superb 5-year survival results. My question relates to the absence of 10-year survivors in patients with N1 disease and presumed complete resection. Because nearly all published series report 10-year survival results in the neighborhood of 20% to 30%, I would like to ask Dr Van Schil to comment on those differences of survival in this subset of patients. Do they relate to patient selection (sleeve lobectomy versus pneumonectomy), intraoperative staging technique (N1 versus N2), or perhaps cause of death (cancer death versus other causes)? Do you feel that sleeve resection should be avoided in N1 patients even if a complete and curative resection is possible?

DR VAN SCHIL: I did not say that. I think that survival with N1 disease is worse, much worse than with N0 disease. We have no definite data on this, but I presume that if these patients had had a pneumonectomy, survival would have been the same, because, as I told you, causes of death were mainly distant metastases and not local recurrence in patients with N1 and N2 disease.

Fulminant Pulmonary Embolism: Symptoms, Diagnostics, Operative Technique, and Results

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Fulminant pulmonary embolism associated with cardiac arrest has an extremely high mortality. The feasibility of pulmonary embolectomy initiated during resuscitation is still under discussion. Between January 1975 and January 1991, pulmonary embolectomy was performed in 27 patients, 21 to 79 years old. The diagnosis was established primarily by clinical findings in 18 patients, by angiography and ventilation-perfusion mismatch in 4 patients, and by transesophageal echocardiography in 1 patient seen recently. Eleven patients did not require resuscitation (group 1); 5 patients had to be resuscitated and underwent operation after circulation was reestablished without need of further cardiac massage (group 2); and 11 patients were connected to extracorporeal circulation devices during cardiopulmonary resuscitation (30 to 210 minutes) (group 3). Embolectomy was performed using extracorporeal circulation with the heart beating (n = 2) or fibrillating (n = 15) or using cardioplegia (n = 10). Fifteen patients received a caval clip or ligature at the end of the procedure. Twelve patients died early postoperatively; the mortality rates were 36%, 60%, and 45% for groups 1, 2, and 3, respectively. Eight patients died of right heart failure, and 2 patients each died of brain death and sepsis. Of the surviving patients, only 1 showed ischemic brain damage. Mean stay in the intensive care unit was 5.1, 7.0, and 9.75 days for groups 1, 2, and 3, respectively. There were no recurrent embolisms during the 15-year follow-up (mean follow-up, 4.6 years). This experience demonstrates that even with subtotal obstruction of the pulmonary arteries, effective cardiopulmonary resuscitation with maintenance of uncompromised brain function is possible. The decision to operate may be based on clinical features without invasive diagnostic procedures. In patients with fulminant pulmonary embolism requiring external cardiac massage, we favor immediate operation, which has an acceptable outcome.

(Ann Thorac Surg 1991;52:1102–7)

Fulminant pulmonary embolism resulting in hypoxia and right ventricular failure has an extremely high mortality. Many patients die without being referred to a cardiac surgical unit [1]. Survival not only reflects the efficacy of surgical treatment but also depends on the preembolic morbidity, the quality of perioperative cardiopulmonary management, and the postoperative complications. In this study we retrospectively analyze how the degree of cardiopulmonary deterioration determines diagnostic modalities and comment on the feasibility of pulmonary embolectomy initiated during ongoing cardiopulmonary resuscitation.

Material and Methods

Between January 1975 and January 1991, 27 patients underwent pulmonary embolectomy using cardiopulmonary bypass in our institution. There were 17 male and 10 female patients, 21 to 79 years old (mean age, 50 years). The medical reports, diagnostic modalities, the intraoperative course, and the postoperative course were reviewed. A complete follow-up of all surviving patients was obtained by telephone or letter.

Clinical Features

On admission to our unit, 10 patients were breathing spontaneously, and 17 required tracheal intubation and mechanical ventilation. The patients were divided into three groups based on the degree of cardiac deterioration. In 11 patients (group 1), adequate to borderline cardiac function could be maintained by moderate to high doses of catecholamines; no external cardiac massage was necessary. This group included all 10 patients who were breathing spontaneously as well as 1 patient requiring artificial respiration. In 16 patients, cardiac failure necessitating external cardiac massage developed, and they had to be intubated and mechanically ventilated. Five of these patients required only temporary or intermittent external cardiac massage (group 2). They underwent embolectomy after at least borderline cardiac pump function had been reestablished. All resuscitative measures failed in 11 patients (group 3). They were taken to the operating room and connected to extracorporeal circulation during continuous external and internal cardiac massage. The period of cardiopulmonary resuscitation lasted from 30 to 210 minutes.

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Table 1. Direct and Indirect Diagnostic Indications of Pulmonary Embolism

Indication	No. of Patients
Indirect	
ECG, chest roentgenogram, arterial blood gases	27
Shock and resuscitation/Swan-Ganz catheterization	16
History of previous operation/confinement to bed	13
Symptoms of acute phlebothrombosis	6
Transthoracic echocardiography	3
Previous pulmonary embolism/thrombolytic therapy	2
Direct	
Pulmonary angiography	4
Ventilation/perfusion scan	4
Transesophageal echocardiography	1

ECG = electrocardiogram.

Diagnostics

All patients had symptoms of severe pulmonary embolism including chest pain, cyanosis, tachycardia, decreased blood pressure, or shock. They had dyspnea or were already on artificial respiration. A history of previous operation or prolonged confinement to bed in 13 patients and acute phlebothrombosis in 6 patients suggested the diagnosis. Additional indirect evidence of acute pulmonary embolism was obtained by electrocardiography, chest roentgenography, and Swan-Ganz catheterization. In 18 (67%) of the 27 patients, the diagnosis of fulminant pulmonary embolism and the ultimate decision for urgent surgical intervention were based only on these indirect findings without further confirmation of the embolism by imaging techniques. Table 1 shows the predominant factors influencing the decision to operate on these patients.

In only 8 (30%) of the 27 patients was the diagnosis verified by pulmonary angiography or ventilation-perfusion mismatch, and in only 1 recent patient did we use transesophageal echocardiography to confirm the diagnosis of fulminant pulmonary embolism (Table 2). The percentage of patients who had ventilation/perfusion scan, angiography, or transesophageal echocardiography in the groups with cardiac massage (group 2, 40%; group 3, 27%) was comparable with that in the group without cardiac deterioration (group 1, 36%). In groups 2 and 3, these studies were done at a time of adequate cardiac function before acute deterioration necessitating cardiopulmonary resuscitation and emergency operation. In group 1, the clinical findings were mild to moderate, and operation was performed because of progressive deterioration of cardiopulmonary function based on the results of the original imaging, which was not repeated.

Sixteen of the 27 patients were referred to our institution from other hospitals. Five of these patients were in deep shock and were taken to the operating room without any further diagnostic procedure. Two were in group 2 and 3, group 3.

Operative Technique

In all patients, pulmonary embolectomy was performed using extracorporeal circulation. Access was always through a median sternotomy. In patients requiring continuous cardiopulmonary resuscitation, external chest compression was followed by manual cardiac massage after rapid sternotomy and pericardiotomy. Aortic and right atrial cannulas were inserted through stab incisions and were initially secured by hand. In 10 patients, the myocardium was protected by cardioplegia (St. Thomas' Hospital solution); in 15, pulmonary embolectomy was performed with the heart spontaneously fibrillating; and in 2, the heart remained beating throughout the procedure.

The pulmonary trunk was incised longitudinally, and the thrombus was removed using suction and forceps. In all patients, both pleural cavities were opened, and the peripheral pulmonary vascular bed of each lung was cleared by gentle manual compression. If necessary, the incision was extended into the left pulmonary artery, or the right pulmonary artery was opened through a second incision between the superior vena cava and the ascending aorta for visualization of lobar branches.

At the end of the procedure, a caval clip or ligature (2 patients seen early in the series) was placed in 15 patients. The inferior vena cava was approached by extending the median sternotomy caudally to the umbilicus and was exposed by the Kocher maneuver.

Results

The early mortality rate for patients undergoing pulmonary embolectomy without a previous cardiovascular collapse (group 1) was 36% (4/11). In patients with temporary external cardiac massage and subsequent operation (group 2), the mortality rate was substantially higher (60%, 3/5), whereas patients with continuous cardiac massage (group 3) had a mortality rate of 45% (5/11), which was nearly identical with the overall mortality rate of 44%. Table 3 summarizes the causes of death in the three patient groups. Of the 12 patients who died, 8 died

Table 2. Diagnosis and Patient Groups[a]

Group	Indirect Evidence Exclusively	Pulmonary Angiography	Ventilation/ Perfusion Scan	Transesophageal Echocardiography
1	7 (64)	1 (9)	3 (27)	0
2	3 (60)	2 (40)	0	0
3	8 (73)	1 (9)	1 (9)	1 (9)

[a] Numbers in parentheses are percentages.

of right heart failure, 3 of whom could not be weaned from extracorporeal circulation (embolectomy during cardioplegic arrest, n = 5; embolectomy during ventricular fibrillation, n = 3). Two patients died as a result of brain death and 2, of sepsis.

Postoperative complications among the surviving patients occurred predominantly in group 2. In this group, both survivors sustained reversible cardiac and renal failure, whereas in groups 1 and 3, only cardiac failure occurred (43% and 17%, respectively). Ischemic brain damage was found only in 1 patient in group 3 (17%), and septic complications were not significantly increased in either group. The mean stay in the intensive care unit for groups 1, 2, and 3 was 5.1, 7.0, and 9.75 days, respectively.

During the follow-up of 15 years (mean follow-up, 4.6 years), the surviving 15 patients were completely rehabilitated and did not sustain another pulmonary embolism regardless of caval clip implantation. All patients except 1 have been on a regimen of Coumadin (crystalline warfarin sodium) (n = 11) or aspirin (n = 2) since embolectomy. In 10 of these patients, a caval clip or ligature was used, and 6 of them have experienced recurrent symptoms of venous thrombosis (Table 4).

Comment

Fulminant pulmonary embolism is an infrequent but often fatal disorder despite improved understanding of its pathogenesis, diagnosis, and treatment. The need of surgical embolectomy rarely arises because most fatal emboli cause rapid circulatory collapse and death [2]. The first pulmonary embolectomy was performed by Trendelenburg [3] in 1908, but long-term survival was not achieved until 1924 [4]. It was soon recognized that far more patients died as a result of the procedure than survived. The introduction of extracorporeal circulation for pulmonary embolectomy by Cooley and colleagues [5] and Sharp [6] in 1961 constituted a major advance, and this technique is still the preferred approach. However, concomitant irreversible cardiac arrest still causes an excessively high mortality.

The diagnosis is usually suggested by a history of sudden circulatory collapse with progressive hypoxia and an increasing need of catecholamines. It is supported by chest roentgenography, electrocardiography, and Swan-Ganz catheterization. Direct evidence of pulmonary embolism is provided by ventilation/perfusion scan or pulmonary angiography, the latter being the most accurate method and the method strongly recommended by several authors [7–9]. However, transesophageal echocardiography, the speediest diagnostic approach, may ultimately replace pulmonary angiography.

Table 3. Causes of Death[a]

Group	Cardiac Failure	Sepsis	Brain Death
1	3/4	1/4	0
2	1/3	1/3	1/3
3	4/5	0	1/5
Total	8 (67)	2 (17)	2 (17)

[a] Numbers in parentheses are percentages.

Table 4. Long-Term Follow-up of 15 Survivors[a]

Variable	Caval Clip (n = 10)	No Caval Clip (n = 5)
Coumadin or aspirin (n = 2)	9 (90)	5 (100)
No anticoagulant treatment	1 (10)	0
Recurrent venous symptoms	5 (50)	2 (40)
Pulmonary embolism relapse	0	0

[a] Numbers in parentheses are percentages.

In most of our patients (67%), the diagnosis was established by indirect means. There were no false-negative diagnoses among these patients. We therefore agree with Clarke and colleagues [10] that angiography and pulmonary ventilation/perfusion scan are not mandatory diagnostic procedures. They may cause an unacceptable delay, as death can occur at any moment. We believe that these methods should be reserved for patients whose cardiac function is still stable. In patients who require cardiac massage, diagnostic procedures should be reduced to a minimum, and the patient taken to the operating room as soon as possible. However, in certain situations, the clinical differentiation between acute myocardial infarction and fulminant pulmonary embolism may be difficult, especially when medical history, electrocardiogram, respiratory compromise, and chest roentgenogram do not reveal typical findings. If an urgent surgical intervention seems indicated, clinical decision making is then based mainly on the results of Swan-Ganz catheterization.

There are only a few reports [11] suggesting that patients with pulmonary embolism who require mechanical ventilation or cardiac massage may be candidates for thrombolytic therapy. As no randomized trial exists, larger patient series will be necessary to compare the results of embolectomy and thrombolysis under these circumstances. We agree with Gray and co-workers [12] that decisions regarding surgical intervention have to be based on the needs of the individual patient. In patients with acute mild to moderate pulmonary embolism, thrombolysis is the therapy of choice unless contraindicated for other reasons. However, in patients requiring an increasing dose of catecholamines and with progressive hypoxia necessitating intubation and mechanical ventilation, thrombolytic therapy currently appears inappropriate. We believe these patients should undergo urgent surgical embolectomy. In our experience, the mortality rate for these patients was 36%. Our group 2 patients, who had operation after reestablishment of borderline cardiac function, fared less well than group 3 patients, those requiring embolectomy during continuous resuscitation, both in terms of cardiac and renal complications. This finding indicates that prolonged nonsurgical management of such patients seems to be inferior to immediate surgical intervention. In the case of acute cardiac deterioration because of subtotal obstruction of the pulmonary arteries mandating resuscitation, continuous external cardiac massage and artificial respiration have been sufficient to avoid lethal hypoxic brain damage.

The most widespread technique for pulmonary embolectomy is that described by Cooley and associates [5] and Sharp [6]. In a multicenter study, Del Campo [13] reviewed the cases of 651 patients operated on by this technique. The overall early mortality rate was 40% compared with 51% for patients having operation without cardiopulmonary bypass. Gray and co-workers [14] reported a hospital mortality of 11% in patients who had not sustained a cardiac arrest before embolectomy and 64% in those who had. There is a difference of opinion about whether patients requiring permanent external cardiac massage should be referred for surgical intervention. We advocate rapid median sternotomy with placement of arterial and venous cannulas during intermittent external and internal cardiac massage. Other groups [9, 12] prefer femoral vein–femoral artery partial cardiopulmonary bypass. We believe this approach should be reserved for patients who cannot immediately undergo operation and therefore benefit from preoperative cardiovascular stabilization. In our experience, femoral cannulation and bedside institution of extracorporeal circulation may gain no time and may even delay embolectomy.

In conclusion, our experience demonstrates that effective resuscitation with maintenance of uncompromised brain function is possible in patients with fulminant pulmonary embolism and resulting cardiopulmonary failure. In case of acute cardiac deterioration leading to external cardiac massage, no time-consuming efforts should be made to reestablish sufficient cardiac pump function. The diagnosis should and, in fact, can be established by clinical features only, thereby allowing pulmonary embolectomy to be initiated immediately, even if continuous external cardiac massage is necessary before the patient is connected to extracorporeal circulation.

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DISCUSSION

DR LAZAR J. GREENFIELD (Ann Arbor, MI): I congratulate Dr Schmid on his presentation and thank him for the opportunity to review the manuscript. An aggressive approach to massive pulmonary embolism is certainly appropriate, but there continue to be controversies regarding the indications and patient stratification, methods of diagnosis, and type of operative approach. The overall operative mortality rate of 44% achieved in this study is comparable with the mortality rates in other reported experiences, but some would disagree with the need for the procedure in patients whose condition had stabilized after the embolism without ventilatory support. In fact, it is surprising that the mortality rate was 36% in this group, which Dr Schmid and associates should explain.

What is remarkable, however, is the fact that the diagnosis was made on clinical grounds alone in two thirds of these patients. Because the clinical diagnosis of massive pulmonary embolism in every large series in the United States has been wrong in 70% to 80% of patients as confirmed by angiography, it would be very helpful for Dr Schmid and associates to share their clinical expertise. Is it true that you found emboli in every patient who underwent exploration over the 16 years of the study? Which clinical features or pulmonary arterial hemodynamic findings did you find to be specific? These details are not in the manuscript. Having performed embolectomy, you chose to clip the vena cava in only 15 patients. How were they selected? There were late venous symptoms in 60% of these patients. In how many were the venae cavae obstructed? As you know, this is the complication to be expected when a clip is used instead of a filter.

It seems clear that an overall mortality rate of 44% still leaves something to be desired. Therefore I will mention the alternative technique of catheter pulmonary embolectomy. The procedure is usually performed in the radiology suite once the diagnosis has been confirmed by angiography. After exposure of the jugular vein under local anesthesia, a steerable cup-catheter is inserted and positioned under fluoroscopy adjacent to the embolus. Syringe suction captures the embolus in the cup and holds it there while the catheter and embolus are withdrawn.

We have had experience with this technique in 32 patients. The overall mortality rate was 22%. This favorable experience is not unique to us, but has been demonstrated by others in the United States and also in a series from France. In that series, the overall mortality rate was 28%. However, the mortality rate was only 18% in those patients in whom emboli could be removed by the catheter technique. Therefore, there is an alternative approach for the patient with massive embolism who still can sustain a blood pressure with inotropic support. There obviously remains a role for open embolectomy in the patient in whom the catheter procedure fails or in the patient who requires constant closed-chest massage.

DR ERNESTO J. MOLINA (Minneapolis, MN): If heparin sodium therapy is the only alternative offered to the patient with pulmonary embolism, the most critically ill patients would have no choice but to undergo pulmonary embolectomy, with substantial morbidity and mortality. My colleagues and I at the University of Minnesota have developed a different approach for all patients with massive pulmonary embolism.

During the past 7 years, we have treated 14 patients by direct infusion of urokinase at the time the diagnostic arteriogram is obtained. Our standard protocol calls for a bolus injection of urokinase directly into the clot in the pulmonary artery at a dose of 2,200 U/kg. This is followed by continuous infusion of the same dose per hour through the same catheter, which is left in position until the clot is cleared.

The most important safety factor to monitor is the fibrinogen level during urokinase therapy. Keeping the level at 2 g/L, we have not experienced bleeding in any of our patients. Two of the patients needed resuscitation and were in shock. The other 12 had sizable emboli but did not require resuscitation. Clearing of clots was relatively rapid, ranging from 90 minutes to 24 hours. There were no deaths, and the patients were discharged from the intensive care unit in 48 hours.

We believe that implementing this protocol in patients with or without recent operation offers a better alternative than embolectomy on cardiopulmonary bypass. Realistically, it takes a minimum of 1 hour for a patient suspected of having a pulmonary embolism to be actually on cardiopulmonary bypass in the operating room. Our approach is prompt, effective, and safe. As a corollary, we have not operated on any patient for massive pulmonary embolism during the last 7 years.

DR AGUSTIN ARBULU (Detroit, MI): I congratulate Dr Schmid and his associates for resurrecting a valuable operation. Our experience is almost identical to theirs. It comprises 15 years and 20 patients, 7 of whom died.

I have a question that is illustrated by the case of a 65-year-old, 112.5-kg diabetic patient who had a fulminant pulmonary embolism in October 1976. The removed embolus grew *Escherichia coli*, which originated in a right hydronephrotic ureter, which resulted in a right ileo-femoral vein thrombosis. The patient survived pulmonary embolectomy and lived an additional 12 years, dying of cancer of the pancreas.

Dr Schmid, did you observe any septic pulmonary emboli in your series?

DR ARTHUR C. BEALL, JR (Houston, TX): I have two brief points to make. First, it is of interest that this presentation comes from Germany and that the February issue of *The Annals of Thoracic Surgery* has a large series from Paris. Current interest in pulmonary embolectomy using cardiopulmonary bypass seems to be centered in Europe, whereas it has become less and less used in the United States in the 30 years since it was first reported here. One of the reasons pulmonary embolectomy got a bad name in this country is that several large series were reported in which more than 50% of the patients did not have a pulmonary embolus. You would not expect very good results from pulmonary embolectomy in a patient who does not have a pulmonary embolus.

Second, I would like to raise the same warning that Dr Greenfield did, namely, that operating on clinical grounds alone will produce a number of patients without a pulmonary embolus. With the use of temporary cardiopulmonary bypass from femoral vein to femoral artery and with the portable equipment that is currently available, one can resuscitate the patient, take him or her to the radiology suite for a pulmonary angiogram, and then do pulmonary embolectomy only on those who actually have a pulmonary embolism.

DR KARL J. KARLSON (Boston, MA): I commend Dr Schmid and the group from Hannover on their series of pulmonary embolectomies. I will comment on our own experience in Boston with this operation, an experience primarily reflecting the work of Dr Robert Berger. Based on information in the literature and our own autopsy studies, we have established fairly firm criteria for acute pulmonary embolectomy. We have found that the mortality rate for an obstruction of more than 50% of the pulmonary vasculature approaches 50%, and this figure rises to 70% if the patient requires vasopressor therapy. If clinical deterioration continues, the mortality rate approaches 100%.

We have performed acute pulmonary embolectomies on 22 patients over the past two decades. All patients had femoral cannulation for cardiopulmonary bypass before the induction of general anesthesia, but none were placed on bypass until they manifested hemodynamic instability. Seventeen of these patients actually did require the institution of bypass before anesthesia was induced because their condition was unstable. Six of the patients died. Of special note are 2 patients who were categorized as dying of unrelated causes. These patients represent errors in diagnosis. They did not have pulmonary angiography and had operation on on the basis of clinical findings alone. They could not be weaned from bypass and died in the operating room.

Dr Schmid and his colleagues operated on most of their patients on the basis of clinical findings, and it is surprising to me that they report no negative pulmonary artery explorations. I, too, would like to ask them how confident they are in their ability to make the diagnosis of acute pulmonary embolism without a pulmonary angiogram.

I commend The Society for putting this paper about pulmonary embolectomy on the program. It was exactly 60 years ago this month that Dr John Gibbon wrote the following words on a patient's chart at the Massachusetts General Hospital while working as a research fellow for Dr Churchill:

> During that long night's vigil, watching the patient struggle for life, the thought naturally occurred to me that the patient's life might be saved if some of the blue blood in her veins could be continuously withdrawn into an extracorporeal blood circuit, exposed to an atmosphere of oxygen, and then returned to the patient by way of a systemic artery in a central direction. Thus, some of the

> patient's cardiorespiratory functions might be temporarily performed by the extracorporeal blood circuit while the massive embolism was surgically removed.

These words have obviously affected us all profoundly.

DR SCHMID: The number of remarks and questions shows that there is a great interest in this field. With respect to Dr Greenfield, we certainly also believe that an overall mortality rate of 44% covering a 16-year experience is not satisfying. The patients who had operation after stabilization (group 1) were, some would say, ideal candidates for thrombolysis. However, most of them were operated on from 1983 to 1988, when thrombolysis was not available. After 1988, thrombolytic therapy was initiated by our pulmonologists and angiologists. It is now performed even during resuscitation, as indicated in a report by a group in Göttingen, Germany, who had a mortality rate of 45% under these circumstances. However, our survival rate of 55% in patients requiring resuscitation without the possibility of angiography (group 3) is, we believe, the best that can be achieved. We think these results are possible only if there is no delay because of diagnostic procedures, that is, patients are immediately referred for surgical intervention.

Dr Molina described a thrombolytic protocol for direct urokinase infusion into the pulmonary artery at the time of the diagnostic arteriogram. However, most of the patients in group 3 required ongoing external cardiac massage, thus making angiography absolutely impossible.

Dr Arbulu asked about septic emboli. Two patients died of a fulminant sepsis, but in neither could bacteria be cultured from the embolus.

Last, I will comment on the warnings of Dr Beall, Dr Karlson, and Dr Greenfield about operating on clinical grounds alone. In our experience, fulminant pulmonary embolism requiring resuscitation should be managed aggressively. Pulmonary angiography is important but is not always mandatory. If it is not available or is impossible to perform because of ongoing external cardiac massage, the indication for immediate operation can and should be based on clinical findings only.

Surgical Management of Resistant Mycobacterial Tuberculosis and Other Mycobacterial Pulmonary Infections

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Between August 1983 and October 1990, 42 patients with resistant *Mycobacterium tuberculosis* underwent 44 pulmonary resections. During the same time, 38 patients with mycobacterial infections other than tuberculosis had 41 pulmonary resections. All patients either were poor candidates for medical therapy alone or had existing complications requiring surgical intervention. There was one operative death in each group, both from adult respiratory distress syndrome (postpneumonectomy pulmonary edema). Complications were high, with bronchopleural fistula most commonly occurring after right pneumonectomy in patients infected with *Mycobacterium avium* with superimposed infection with nonmycobacterial pathogens. In patients undergoing pneumonectomy for resistant *Mycobacterium tuberculosis*, the left lung was most often resected. It is recommended that if localized disease is present and medical treatment is likely to fail, pulmonary resection should be performed for resistant *Mycobacterium tuberculosis* infection after 3 months of drug-specific therapy. Muscle flaps were used frequently to avoid residual space and bronchial stump problems. Earlier resection in patients with indolent nontuberculous mycobacterial pulmonary infections is advocated before extensive polymicrobial contamination and right lung destruction.

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Since the introduction of rifampin and other antimycobacterial drugs, there has been a declining number of reports published regarding operation for pulmonary mycobacterial infections. Specifically, there is little information regarding surgical outcome for pulmonary resections in patients with resistant *Mycobacterium tuberculosis* compared to patients with pulmonary disease due to mycobacteria other than tuberculosis. This paper reports 80 patients undergoing pulmonary resection for mycobacterial pulmonary disease and compares the results and complications in those with resistant *Mycobacterium tuberculosis* to those with other mycobacterial infections.

Patients and Methods

Beginning in August 1983 and ending October 1990, 42 patients with pulmonary disease due to isoniazid- and rifampin-resistant *Mycobacterium tuberculosis* underwent 44 pulmonary resections. Two patients had staged bilateral resections. During this same period, 38 patients with mycobacterial infections other than tuberculosis underwent 41 pulmonary resections. Two had staged bilateral resections, whereas a third had a completion pneumonectomy 5 years after undergoing lobectomy. There was an equal distribution of men (21) to women (21) in patients with resistant *Mycobacterium tuberculosis*, whereas in patients with mycobacterial infections other than tuberculosis, women predominated 26 to 12. Patients with resistant *Mycobacterium tuberculosis* averaged 37 years of age (range, 21 to 63 years), whereas those with mycobacterial infections other than tuberculosis were older, averaging 50 years of age (range, 33 to 69 years). Racial distribution was also different for the two groups. Although there was relatively even distribution between whites, blacks, and Orientals in those operated on for resistant *Mycobacterium tuberculosis*, 37 of the 38 patients with mycobacterial infections other than tuberculosis were white, the remaining patient being black. At the time of operation, 29 of 42 patients with *Mycobacterium tuberculosis* had positive sputum, whereas 26 patients out of 38 with mycobacterial infections other than tuberculosis had positive sputum.

In those patients undergoing resection for mycobacterial infections other than tuberculosis, *Mycobacterium avium* complex was the most frequent causative organism, occurring in 33 patients; 3 patients each were infected with *Mycobacterium kansasii*, *Mycobacterium chelonae*, and *Mycobacterium xenopi*. Four of the 38 patients with mycobacterial infections other than tuberculosis had more than one mycobacterial organism identified from cultures.

Patients operated on for resistant *Mycobacterium tuberculosis* were resistant to multiple other drugs as well as isoniazid and rifampin. A few were operated on for anticipated noncompliance with little or no option for retreatment if they failed again. All patients had to have reasonably localized disease to be considered surgical candidates.

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Table 1. Pneumonectomies Performed on Patients With Resistant Mycobacterium tuberculosis *or Mycobacterial Infections Other Than Tuberculosis*

Operation	RMTB (n = 23)	MOTT (n = 17)
Left pneumonectomy	4	1
Left pneumonectomy with serratus muscle flap	9	1
Left completion pneumonectomy with serratus muscle flap	1	0
Left completion pneumonectomy with latissimus muscle flap	1	0
Left pneumonectomy with latissimus muscle flap	5	3
Left pneumonectomy with intercostal muscle flap	1	0
Left completion pneumonectomy	0	3
Right completion pneumonectomy	0	2
Right completion pneumonectomy with latissimus muscle flap	0	1
Right pneumonectomy with latissimus muscle flap	1	6
Right pneumonectomy with serratus muscle flap	1	0

MOTT = mycobacterial infection other than tuberculosis; RMTB = resistant *Mycobacterium tuberculosis.*

Patients infected with mycobacterial infections other than tuberculosis also had to have predominantly localized disease and usually were superinfected with other nonmycobacterial organisms or had other preexisting complications.

All patients in both groups had received multiple-drug chemotherapy before operation. The best multiple-drug regimen, based on drug susceptibility studies, was used preoperatively as well as postoperatively.

Work-up included complete pulmonary function tests, blood gases, computed tomography, and quantitative ventilation-perfusion scans.

Patients were operated on using double-lumen endobronchial tubes. Single-dose intrathecal narcotic analgesia (morphine sulfate [Duramorph; Elkins-Sinn, Cherry Hill, NJ], 0.5 to 1.0 mg) given before the thoracotomy incision has supplemented general anesthesia for the last 5 years to help with postoperative pain. Rib blocks were routinely used, and patient-controlled analgesia has been used for the past 5 years. Operations performed are listed in Tables 1 and 2. After the initial eight operations, all patients who had positive sputum at the time of operation had muscle flaps used to add support and to improve healing of the bronchial closures. Muscle flaps were constructed as described by Pairolero and associates [1]. In addition, all patients with polymicrobial contamination, those with preoperative bronchopleural fistulas, and those in whom space problems were likely to occur after lobectomy had muscle flaps used. A total of 40 muscle flaps were performed in the 85 operations. The latissimus dorsi muscle was used in 25 patients, the serratus anterior in 13, and the intercostal muscle in 2.

Two patients with resistant *Mycobacterium tuberculosis* had previous pulmonary resections, and 1 had a preexisting bronchopleural fistula. Three patients in the mycobacterial infections other than tuberculosis group had previously been treated for *Mycobacterium tuberculosis* infections. Seven patients with mycobacterial infections other than tuberculosis had previous lobectomies, 3 had preoperative bronchopleural fistulas, and 3 had radiation either to the chest wall or lung on the operative side. In addition, 9 patients with mycobacterial infections other than tuberculosis had extensive polymicrobial contamination at the time of operation.

Results

There was one operative death secondary to adult respiratory distress syndrome (postpneumonectomy pulmonary edema) among the patients with resistant *Mycobacterium tuberculosis*. There were six late deaths, from 2 months to 4 years postoperatively, only two of which

Table 2. Lobectomies Performed on Patients With Resistant Mycobacterium tuberculosis *or Mycobacteria Infections Other Than Tuberculosis*

Operation	RMTB (n = 21)	MOTT (n = 24)
Left upper lobectomy	3	2
Left upper lobectomy with latissimus muscle	1	0
Left lower lobectomy and lingulectomy	1	0
Left upper lobectomy, superior segment, intercostal muscle	0	1
Lingulectomy	0	2
Right upper lobectomy	4	6
Right upper lobectomy plus superior segment	0	2
Right upper lobectomy with latissimus muscle	4	0
Right upper lobectomy with serratus muscle	1	0
Right upper lobectomy plus superior segment (right lower lobe) with latissimus muscle	1	0
Right upper lobe plus right middle lobe plus superior segment	2	1
Right upper lobe plus right middle lobe with latissimus muscle	1	1
Right upper lobe plus right middle lobe	1	2
Right middle lobe	0	5
Right lower lobe	2	2

MOTT = mycobacterial infection other than tuberculosis; RMTB = resistant *Mycobacterium tuberculosis.*

Table 3. Complications in Patients With Mycobacterial Infections Other Than Tuberculosis

Complication	No. of Patients
Bronchopleural fistula	8
Prolonged air leak or space	4
Respiratory failure	2
Recurrent nerve palsy	1
Wound breakdown	1
Pericardial effusion	1
Horner's syndrome	1
Psychiatric	1

were related to the resistant *Mycobacterium tuberculosis* infection. Only 3 patients had persistently positive sputum postoperatively. One patient had her medication prematurely stopped by her local physician and subsequently died of tuberculosis, whereas the other patient who died had contralateral disease after right pneumonectomy which failed to respond to medical therapy. The other patient with positive sputum postoperatively had bilateral disease and refused a second operation. There was one late death each due to renal failure, myocardial infarction, drug overdose (self-inflicted) and respiratory failure.

There was one operative death among the patients undergoing pulmonary resection for mycobacterial infections other than tuberculosis. This patient also died of adult respiratory distress syndrome (postpneumonectomy pulmonary edema). There have been seven late deaths in this group, six due to respiratory failure and one of unknown causes. Of the 6 patients who died late due to respiratory insufficiency, all had progression of their disease postoperatively and 3 had bronchopleural fistulas.

Complications after operation for resistant *Mycobacterium tuberculosis* and mycobacterial infections other than tuberculosis have been frequent. There were eight major complications in patients undergoing resection for resistant *Mycobacterium tuberculosis*. There were 3 patients with wound complications, two instances of respiratory failure, one recurrent nerve palsy, one Horner's syndrome, and one reexploration for bleeding. The only patient with resistant *Mycobacterium tuberculosis* in whom a bronchopleural fistula developed was sputum positive preoperatively, had undergone a previous lobectomy, and had a preoperative bronchopleural fistula. This fistula was successfully closed by a transmediastinal transpericardial approach [2].

Postoperative complications in the mycobacterial infections other than tuberculosis group were more frequent than in patients with resistant *Mycobacterium tuberculosis* (Table 3). Bronchopleural fistulas developed in 8 patients. All patients with bronchopleural fistulas had at least two of the following: radiation to the chest wall or lung for unrelated disease (2 patients), previous pulmonary resection (2), positive sputum preoperatively (7), or extensive polymicrobial contamination in a destroyed lung before operation (7). Of the 8 patients in whom bronchopleural fistulas developed, 4 subsequently died of respiratory complications. All 4 had attempts at fistula closure either transmediastinally, with muscle flaps, or with thoracoplasty, and none were successful. The remaining 4 patients are still alive. One has had his fistula closed by use of a muscle flap plus partial thoracoplasty. The remaining 3 patients have persistent fistulas in spite of initial success.

All bronchopleural fistulas in both groups occurred after pneumonectomy. Of the nine fistulas, seven followed right pneumonectomy and only two were after left pneumonectomy.

Comment

The incidence of tuberculosis, once thought to be on the decline in the United States, has plateaued and in some areas risen over the past several years. In New York City in 1987, there was a rate of nearly 30/100,000, compared with 9.25/100,000 nationally. Death rates in New York City increased from 186 in 1986 to 219 in 1987 [3]. The cause of this increase can be traced to human immunodeficiency virus–positive patients, intravenous drug usage, immigration patterns, and complacency in medical therapy. Similar causes hold true for both patients with resistant *Mycobacterium tuberculosis* and those infected with mycobacterial infections other than tuberculosis.

In this series, all patients operated on were either medical failures or patients whose resistance pattern and destructive disease were likely to be medical failures based on our previous experience [4]. No patient in either group was operated on primarily for hemoptysis or to rule out malignancy. Before operation, those patients who had positive sputum were placed on the best regimen possible, based on drug susceptibility studies, for a period of approximately 3 months.

Although it is desirable to have negative smears and cultures before operation, this was believed not to be possible in all cases. We based the timing of operation on a previous group of patients seen at National Jewish Hospital between 1972 and 1983. Of the 136 patients with isoniazid- and rifampin-resistant *Mycobacterium tuberculosis*, 88 patients (65%) responded to medical management (3 consecutive months of negative smears and cultures). The mean conversion time was 2½ months. Based on this experience, we elected to operate after 3 months of drug-specific therapy. A similar approach was used in the timing of resection in patients with mycobacterial infections other than tuberculosis.

In deciding whether to do a lobectomy or a pneumonectomy, quantitative ventilation and perfusion scans were of great benefit. In several instances, although chest films and computed tomographic scans appeared to show substantial residual lung tissue, the scans demonstrated only scant functional tissue. In these cases, pneumonectomy was the procedure of choice.

The patterns of resection were different for the two groups of patients. Patients with resistant *Mycobacterium tuberculosis* requiring pneumonectomy had left-sided pneumonectomies in 21 of 23 cases. This left-sided predominance is similar to that reported by Ashour and associates [5]. However, in patients with mycobacterial infections other than tuberculosis requiring pneumonectomy, there was a more even distribution, eight of 17 being left-sided. A pattern of right middle lobe disease seen in 5 patients with mycobacterial infections other than tuberculosis, associated with lingula involvement in 3, was not seen in patients with resistant *Mycobacterium tuberculosis*. This may imply secondary infection with mycobacterial infections other than tuberculosis in areas of bronchiectasis or a primary role of *M avium* in producing bronchiectasis in these areas.

Prolonged air leak and residual air spaces occurred before the more liberal use of muscle flaps. We now routinely use muscle flaps in lobectomy patients who have positive sputum at the time of operation or in patients who have a substantial chance of having a residual space problem. No prolonged air leak or residual space problems have occurred since this policy has been adopted. Tailoring thoracoplasty or a conventional type of thoracoplasty has not been used in either group when less than a pneumonectomy was performed.

The predilection for bronchopleural fistulas to occur on the right has not been emphasized. One of the left-sided fistulas was probably due to leaving a residual left main bronchus too long after completion pneumonectomy. The other left-sided fistula occurred 3 months postoperatively and was probably related to ongoing infection in a previously irradiated bronchus. Of the seven right-sided bronchopleural fistulas in patients infected with mycobacterial infections other than tuberculosis, two occurred before routine use of muscle flaps and five have occurred in spite of their use. All of these patients had chronic infection due to nonmycobacterial pathogens, including gram-negative organisms.

Conclusions

Operation for mycobacterial pulmonary disease can be performed with a low mortality (2.4%). As reported by others [6, 7], however, the complication rates are high, particularly in those patients with mycobacterial infections other than tuberculosis undergoing right pneumonectomies. In these patients, there is a predilection for bronchopleural fistula formation. We also conclude that in patients with resistant *Mycobacterium tuberculosis*, when total lung destruction occurs, it is most often the left lung that is destroyed. Whether this is due to the smaller size of the left main bronchus, the more limited peribronchial space, the more horizontal course of the left main bronchus, or other causes cannot be answered from this series. Only 1 of 21 patients with resistant *Mycobacterium tuberculosis* undergoing left pneumonectomy had a totally obstructed bronchus at the time of operation, indicating that vascular rather than airway factors are probably responsible for the gross lung destruction.

Factors influencing morbidity and mortality include positive sputum at the time of operation, previous chest irradiation, prior pulmonary resection, and polymicrobial parenchymal contamination.

Only 3 of 42 patients (7.1%) with resistant *Mycobacterium tuberculosis* infections who underwent operation had positive sputum postoperatively, and only 6 (14.2%) have died of all causes. This compares with 22% dying of tuberculosis in a similar group of patients treated medically before this series [4].

More liberal use of muscle flaps may have diminished the development of bronchopleural fistulas and has certainly decreased the incidence of prolonged air leak and postoperative space problems.

We also conclude that poorer results are obtained after pulmonary resection in patients with mycobacterial infections other than tuberculosis than in those with resistant *Mycobacterium tuberculosis*. This in part may be due to the older age group of the patients (average, 50 versus 37 years), the more indolent nature of their disease leading to extensive parenchymal destruction, and the need to perform right pneumonectomy with coexisting polymicrobial contamination.

Based on this experience, we recommend the following: (1) Surgical resection in patients with resistant *Mycobacterium tuberculosis* for whom the prognosis with medical treatment is poor, performed after at least 3 months of drug-specific therapy, if disease is predominantly localized and there is adequate pulmonary reserve. (2) Earlier pulmonary resection in patients with localized indolent pulmonary infections with mycobacterial infections other than tuberculosis before entire right lung destruction and extensive polymicrobial contamination occurs. (3) Liberal use of muscle flaps in patients with positive sputum, when residual postlobectomy space is anticipated, when bronchopleural fistulas exist preoperatively, or when extensive polymicrobial contamination is present.

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DISCUSSION

DR DONALD F. ROWLES (Palo Alto, CA): The Centers for Disease Control has noted that there is a 5% and 6% increase of pulmonary tuberculosis each year. This report, therefore, is a timely one, and we need to be particularly interested in the variety of the atypical tuberculosis, which is commonly drug resistant. We have heard details of Dr Pomerantz and associates' resections and complications. Therefore, I would like to ask about some technical issues.

What about bronchoscopy? Such evaluations are classic in tuberculosis of mucosal and bronchial wall disease, and this should identify the improvement of chemotherapy or it should tell you the patients who would have trouble healing the bronchus if the operation was done too soon.

Also, what about staplers? At this meeting we have heard about several problems with bronchial walls whose thickness or inflammation make healing difficult, or lead to fistula formation. Therefore, again, preoperative bronchoscopy might give you serial studies to show the healing caused by chemotherapy or identify those patients in whom, as Dr Pomerantz pointed out, mucosal flap should be more liberally used. Drs Paul Sampson and David Dugan, my teachers, were always careful to tack living tissue on the bronchial stump, and I think that this should always be done. You will recall that in Dr Pomerantz's paper, approximately half of the patients had muscle flaps because of either positive sputum or preoperative x-ray therapy. This is a slightly different problem in tuberculosis as the radiation causes scarring or loss of blood supply in the surrounding tissue rather than be a problem of tuberculosis in the bronchus itself. Nonetheless I think it is even more important, therefore, that careful covering of the bronchial stump may have prevented some of these fistulas.

Finally, I have not used it, but I wonder if Dr Pomerantz has used fibrin glue to block any of the small openings of these fistulas.

I want to thank Dr Pomerantz for forwarding his manuscript. I encourage you to continue your efforts in this difficult group of patients.

DR POMERANTZ: I would like to thank Dr Rowles for his comments and try to answer each of the questions in order. All patients underwent bronchoscopy. Only 1 patient had endobronchial disease, and this was a patient who had a totally occluded left main bronchus with resistant tuberculosis. A bronchopleural fistula did not develop in this patient. The other bronchoscopy results were normal.

All patients had their bronchi closed using a stapler.

As far as covering the bronchial stump, all bronchial stumps were covered either with pleura or, in the case of the muscle flap, the muscle flap being sewn directly to the bronchus.

We have tried fibrin glue in a few patients. We used glue transbronchially in 1 patient with a persistent bronchopleural fistula on the right side last Thursday, and at least initially it has remained closed.

A Strategy to Increase the Donor Pool: Use of Cadaver Lungs for Transplantation

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A shortage of suitable donors is a serious obstacle to the widespread application of isolated lung transplantation for end-stage lung disease. We hypothesized that lung tissue likely remains viable for a sufficient period of time to allow for safe postmortem retrieval of lungs for transplantation. Studies were conducted in a nonsurvival model of canine lung allotransplantation. Donor animals were sacrificed, and subsequent lung harvest was delayed for 1 hour, 2 hours, or 4 hours. Pulmonary retrieval was then performed in a standard fashion, flushing the lung block with modified Euro-Collins solution. Lungs were then stored for 4 hours before single allotransplantation. Recipient animals were maintained anesthetized, and followed up for 8 hours. By occlusion of the pulmonary artery and bronchus to the native lung, recipient animals were forced to survive solely on the transplanted lung, with a constant inspired oxygen fraction of 0.40. All 5 recipient animals of 1-hour cadaver lungs survived the 8-hour observation period with excellent hemodynamics and gas exchange. Two of 5 recipients of 2-hour cadaver lungs survived the observation period, whereas a third animal survived for 5 hours with excellent gas exchange. One of 4 animals transplanted with a 4-hour cadaver lung survived the observation period. Retrieval of lungs from cadavers whose hearts are not beating may prove to be a safe and effective method to increase the pulmonary donor pool.

(Ann Thorac Surg 1991;52:1113–21)

Lung transplantation has become an effective method of palliating a variety of patients with end-stage lung disease [1]. Since the first successful lung transplant in 1983 [2], there has been a geometric increase in the number of lung transplants reported to the International Lung Transplant Registry at Washington University, St. Louis (Cooper J; personal communication). In the United States there were 89 isolated lung transplants compared with 70 heart-lung transplants reported to the United Network for Organ Sharing in 1989, the first year that isolated lungs outnumbered heart-lungs in this country [3].

Currently, the most serious impediment to the widespread application of lung transplantation relates to the relative scarcity of satisfactory pulmonary donors. In a recent survey of 150 consecutive cardiac donors reported to the United Network for Organ Sharing, lung function was determined to be adequate for consideration of transplantation in only 25% of donors [4]. Because the number of cardiac transplants performed annually in the United States appears to have plateaued at just less than 1,700, there is an urgent need to increase the pulmonary donor pool if lung transplantation is to be widely applied. Four hundred patients with cystic fibrosis alone die annually in the United States because of end-stage lung disease [5]. Many of these, and other patients with end-stage lung disease, could potentially be candidates for transplantation if the donor pool were large enough.

Recognizing that pulmonary epithelial tissue can be obtained from morgue specimens for successful cell culture [6], we hypothesized that retrieval of intact lungs after cessation of circulation might result in adequate gas exchange sufficient to allow for successful transplantation. This hypothesis was based on the notion that the lung is unique among solid organs in that it does not rely on vascular perfusion for cellular respiration. Rather, it would appear that in lung tissue, cellular respiration occurs directly across a gas interface. An additional factor unique to the lung is that the predominant gas exchange processes are entirely passive. Accordingly, one would expect the metabolic requirements of lung tissue to be low and, hence, that the lung might tolerate considerable periods of ischemia. We designed experiments to test the hypothesis that canine lungs could be extracted at intervals after death and interruption of the circulation and be successfully transplanted, producing adequate gas exchange for survival. Our laboratory has extensive experience with a canine nonsurvival lung transplant model for evaluation of early gas exchange function after transplantation [7, 8]. This article reports the results of early gas exchange and short-term survival using this approach.

Material and Methods

Reagents and Equipment

Sodium pentobarbital was purchased from Barber Veterinary Supply, Richmond, VA. Alpha-chloralose was obtained from Sigma Chemicals, St. Louis, MO. Cefazolin

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0003-4975/91/$3.50

was purchased from Eli Lilly Company, Indianapolis, IN, and sodium heparin was obtained from Elkins Sinn Company, Cherry Hill, NJ.

American Edwards Laboratories of Irvine, CA, manufactured the Swan-Ganz catheters, the femoral artery lung water catheters, and the Lung Water Computer. Transducer-derived pressures and cardiac outputs were recorded using a BNC to 34-pin ribbon cable interface on an Apple Macintosh II microcomputer.

Donor Preparation

Heartworm-negative, adult mongrel dogs were sacrificed with 60 mg/kg of intravenous sodium pentobarbital. Donors were then placed in a supine position, secured to a table, and left at room temperature for 1 hour (n = 5), 2 hours (n = 5), or 4 hours (n = 4). At the end of these intervals, the cadaver animals were intubated and ventilated with a tidal volume of 15 mL/kg with an inspired oxygen fraction of 1.0. Respiratory rate was 8 per minute.

Lung Harvest

We used the same method employed in human lung harvest for bilateral lung extraction in these animals [9]. A median sternotomy was performed, and the main pulmonary artery (PA) was identified and opened. Postmortem thrombus was aspirated, and a 24F catheter was inserted into the main PA. The left atrial appendage was incised, and postmortem thrombus was evacuated from the left atrium. The lung block was perfused over a 3- to 4-minute period with 1 L of modified Euro-Collins solution (K_2HPO_4, 7.40 g/L; $NaHCO_3$, 0.84 g/L; KHPO, 2.04 g/L; KCl, 1.12 g/L; $MgSO_4$, 0.48 g/L; cefazolin, 1.00 g/L; D50W, 50 mL/L; heparin, 5,000 U/L) stored at 4°C. Ventilation was continued during the flush. Pulmonary artery pressure was maintained at 25 cm H_2O, because this was the height of the drip chamber. At the completion of the flush, the heart was excised and the double-lung block was excised, leaving a clamp on the trachea. The block was then stored in Euro-Collins solution and kept at 4°C until the recipient animal was ready for the transplant.

Recipient Preparation and Transplantation

Heartworm-negative, adult mongrel dogs were anesthetized with 30 mg/kg of intravenous sodium pentobarbital. Anesthesia was maintained in recipient dogs by infusing alpha-chloralose (12.5 mg $\cdot$ kg^{-1} $\cdot$ h^{-1}) 1 hour after the initial pentobarbital dose. Recipient animals were ventilated with a tidal volume of 15 mL/kg, adjusting the ventilatory rate to maintain normocarbia.

One gram of cefazolin was administered to recipient animals after induction of anesthesia. A right femoral arterial line was placed before a Swan-Ganz catheter was advanced from the right femoral vein. Left thoracotomy was performed and all hilar structures were isolated. The contralateral PA and bronchus were encircled with umbilical tape tourniquets. A Millar pressure transducer was placed in the left atrium. The contralateral PA and bronchus were occluded, using the tourniquets after heparinization. An electromagnetic flow probe was placed around the left PA. After 5 minutes, baseline measurements of the native left lung were recorded.

The donor lung block was removed from storage and separated into constituent lungs, and the left lung was wrapped in cold saline sponges. Allotransplantation was performed in a manner similar to that previously described [7, 8]. Left atrial horizontal mattress anastomosis was performed using 5-0 Prolene (Ethicon, Somerville, NJ). The bronchus was reapproximated with running 4-0 Prolene, and 6-0 Prolene was used for the PA anastomosis. Before completion of the PA suture line, the PA was flushed to remove air. *Transplantation time* was defined as the time from removal of the lung from storage until blood flow was restored to the organ. *Storage time* was defined as the time from the initiation of the Euro-Collins flush to the donor lung until the lung was removed from cold Euro-Collins solution for transplantation.

A portion of the mediastinal aspect of the right lower lobe (approximately 3 cm^3) was excised and placed in paraformaldehyde for subsequent histologic examination.

Measurements

One hour after transplantation, the contralateral PA and bronchus were ligated, forcing the recipient animal to be completely dependent on the transplanted lung for gas exchange. The tidal volume was decreased by half and the respiratory rate doubled to maintain adequate ventilation. The inspired oxygen concentration remained at 0.40 during the entire follow-up period. The ventilatory rate was adjusted as needed to keep the carbon dioxide tension between 35 and 45 mm Hg. If metabolic acidosis occurred, intravenous sodium bicarbonate was infused to maintain a pH between 7.35 and 7.40. Ringer's lactate was used for maintenance intravenous fluid. Left atrial pressure was maintained between 5 and 10 mm Hg. Interventions were not made within 15 minutes of measurements.

A Doppler flow probe was placed on the left PA and the chest was closed loosely. Hemodynamic recordings of the PA, aorta, distal trachea, left atrium, and right atrium were taken every hour for the 8-hour follow-up period. In addition, an Edwards Lung Water Computer was used to estimate the extravascular lung water by a thermodilution method. All animals were followed up for 8 hours or until death. Animals that survived for 8 hours were sacrificed. At the time of death or euthanasia, a portion of the transplanted lung was taken, using sharp dissection, for histologic analysis. The portion of the lung sampled was the mediastinal aspect of the lower lobe inferiorly. Approximately 3 cm^3 of tissue was retrieved and immediately placed in paraformaldehyde.

Specimen Preparation

Light microscopic sections were prepared according to standard techniques. Dehydration was performed serially in ethanol, and specimens were washed in xylene and embedded in paraffin. Five-micrometer sections were stained with hematoxylin and eosin and reviewed in a blinded fashion by one of us (R.R.). Specimens were graded as histologically normal, mild to moderately abnormal, or severely abnormal. Mild to moderate abnormalities included clear evidence of perivascular and peribronchial edema, vascular congestion, or evidence of inflammation. Severe abnormalities consisted of intrapul-

Table 1. Survival After Cadaver Lung Transplantation

Group	Survival	Comments
1-Hour	5/5	Excellent hemodynamics and blood gases
2-Hour	2/5	One death after 2 hours (elevated PA pressure), 1 death after 2 hours (hypoxemia), 1 death after 5 hours (unexplained), 1 animal survived with deteriorating gas exchange, normal PA
4-Hour	1/4	One survived with poor oxygenation, 1 died immediately after tying off native lung, 2 died after 1 hour (hypoxemia)

PA = pulmonary artery

monary hemorrhage, severe vascular congestion, or evidence of intravascular thrombus.

Statistics

Hemodynamic and pulmonary function data were collected hourly. Results are presented as mean ± standard error of the mean.

Animal Care

The Institutional Animal Care and Use Committee of the University of North Carolina reviewed and approved the protocol for this study. All animals received humane care in accordance with the "Guide for the Care and Use of Laboratory Animals" (NIH publication No. 85-23, revised 1985).

Results

The cold ischemic time (storage time) for the left lungs averaged 3.8 hours and was similar in all groups. This is a reflection of the time required to prepare and instrument the recipient animal for transplantation. Transplantation time was also similar for all groups, averaging 1.5 hours.

Survival

Survival for the 8-hour observation period is depicted in Table 1. All recipient animals that were transplanted with a lung retrieved from a 1-hour cadaver donor survived the observation period with excellent hemodynamics and gas exchange function. Two animals transplanted with a 2-hour cadaver lung also survived with excellent gas exchange and hemodynamics. A third animal in this group survived 5 hours with excellent blood gases and suddenly died. An autopsy failed to disclose cause of death. The 2 remaining animals in this group died because of poor lung function. All 4 animals transplanted with 4-hour cadaver lungs had evidence of gross pulmonary edema in the transplanted lung, and all animals showed poor gas exchange function. Three of the 4 animals died within 1 hour of ligation of the contralateral PA and bronchus, whereas 1 of the animals survived the 8-hour observation period with an arterial oxygen tension that ranged between 38 and 65 mm Hg.

Hemodynamics and Gas Exchange

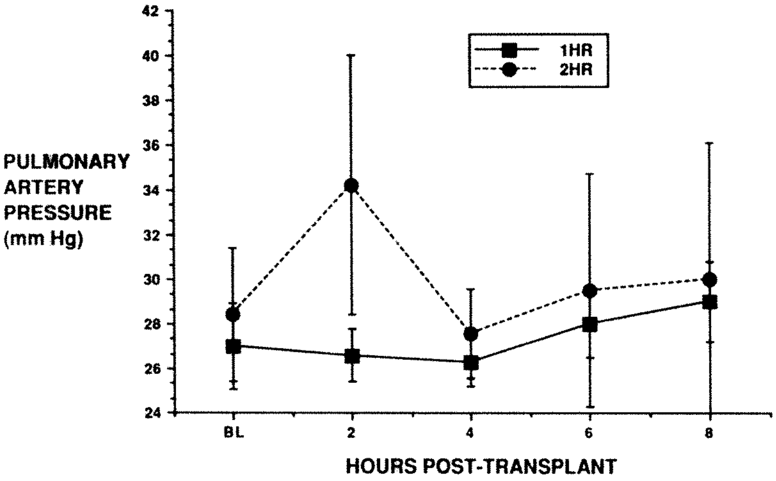
Hourly PA pressure, cardiac output, and arterial oxygen tension are listed in Table 2. For purposes of clarity in graphic presentation, results for 2-hour intervals after transplantation were averaged and are reported as data points for two-hour intervals after transplantation. Accordingly, figures have a baseline time point, which is a measurement performed with the native right lung excluded from the circulation before left pneumonectomy and left allotransplantation. Subsequent data points in all figures represent mean values from two time points for animals in each group.

Table 2. Pulmonary Hemodynamics and Oxygenation After Cadaver Lung Transplantation

Group	Baseline	1 Hour	2 Hours	3 Hours	4 Hours	5 Hours	6 Hours	7 Hours	8 Hours
Survival (n)									
1 Hour	5	5	5	5	5	5	5	5	5
2 Hours	5	5	5	5	3	3	2	2	2
4 Hours	4	3	1	1	1	1	1	1	1
Mean PAP (mm Hg)									
1 Hour	27 ± 2	28 ± 2	26 ± 1	26 ± 1	27 ± 2	27 ± 2	29 ± 2	29 ± 3	28 ± 2
2 Hours	32 ± 3	37 ± 10	35 ± 7	31 ± 3	28 ± 4	31 ± 6	30 ± 9	30 ± 11	31 ± 11
4 Hours	23 ± 2	23 ± 2	23	24	23	23	24	24	24
Cardiac output (L/min)									
1 Hour	2.2 ± 0.2	1.9 ± 0.1	2.2 ± 0.3	1.5 ± 0.1	1.4 ± 0.1	1.3 ± 0.1	1.3 ± 0.1	1.7 ± 0.3	1.8 ± 0.2
2 Hours	2.2 ± 0.2	1.8 ± 0.1	1.5 ± 0.2	1.8 ± 0.2	1.4 ± 0.3	1.1 ± 0.6	1.7 ± 0.4	1.8 ± 0.5	1.8 ± 0.5
4 Hours	1.9 ± 0.3	1.0 ± 0.1	1.1	0.9	1.2	1.0	1.3	1.3	1.4
Arterial pO_2 (mm Hg)									
1 Hour	161 ± 17	158 ± 26	157 ± 27	134 ± 25	135 ± 24	141 ± 24	137 ± 26	141 ± 24	142 ± 23
2 Hours	135 ± 16	137 ± 21	123 ± 27	156 ± 29	155 ± 30	155 ± 25	135 ± 43	115 ± 38	96 ± 43
4 Hours	144 ± 21	77 ± 37	65	58	51	53	42	45	38

PAP = pulmonary artery pressure; pO_2 = oxygen tension.

Fig 1. Mean pulmonary artery pressure for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean). (BL = *baseline.)*



Because of the amount of postmortem thrombus observed in the PA and left atrium of cadaver dogs at 2 hours, donors whose lungs were harvested 4 hours after death were heparinized before barbiturate overdose to exclude intravascular thrombosis as a cause of poor lung function. Accordingly, in the recipients transplanted with 4-hour cadaver lungs, intravascular thrombosis was not believed to be the reason for poor pulmonary function, and postmortem thrombus was not observed in these four donor lungs histologically.

Pulmonary artery pressure (Fig 1) and pulmonary vascular resistance (Fig 2) were remarkably consistent in the recipient animals transplanted with 1-hour cadaver lungs. Surviving animals with 2-hour cadaver lungs also demonstrated stability in pulmonary hemodynamics. Early elevated pulmonary vascular resistance and PA pressure reflected the behavior of all recipients of 2-hour lungs. After the death of 2 animals in this group, the PA pressure returned to more normal levels. Gas exchange was likewise consistently very good in recipients of 1-hour cadaver lungs and was also very good in the surviving animals with 2-hour cadaver lungs (Figs 3, 4). Recipients of 1-hour cadaver lungs were hemodynamically stable throughout the observation period (Figs 5, 6). A late improvement in the 2-hour cadaver lung recipients reflected the death of the animals that were unstable in the early posttransplantation period.

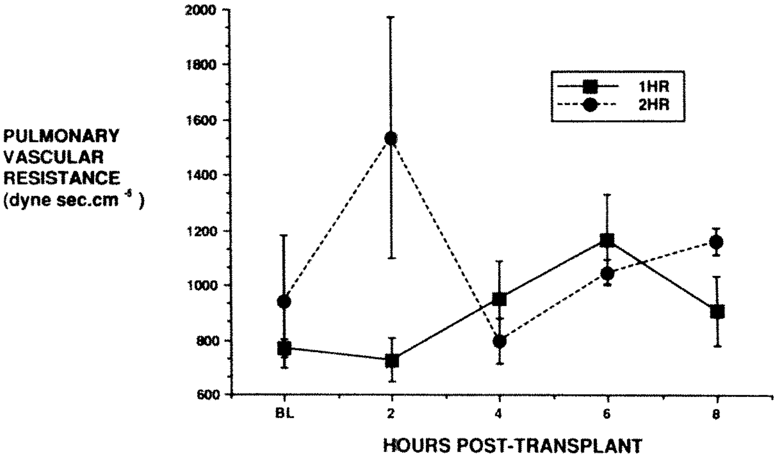
Extravascular lung water increased in all animals, but this increase was more marked in the 2-hour cadaver lung recipients. The subsequent decline in extravascular lung water (Fig 7) reflected the death of the 2 animals with poor gas exchange function in this group.

Histology

Specimens were reviewed by one of us (R.R.) in a blinded fashion and were classified as normal, moderately abnormal, or severely abnormal. Six histologic specimens were believed to be entirely normal, and all of these were from cadaver lungs before transplantation, ie, they were taken from the right lung of the block after harvest and preservation (3 one-hour lungs, 2 two-hour lungs, and 1 four-hour lung). The remaining samples of control lung specimens were moderately abnormal. All specimens from transplanted lungs manifested perivascular and peribronchial edema, hemorrhage, or evidence of vascular congestion.

Four of the histologic specimens retrieved after the observation period were considered to be severely abnor-

Fig 2. Pulmonary vascular resistance for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean). (BL = *baseline.)*



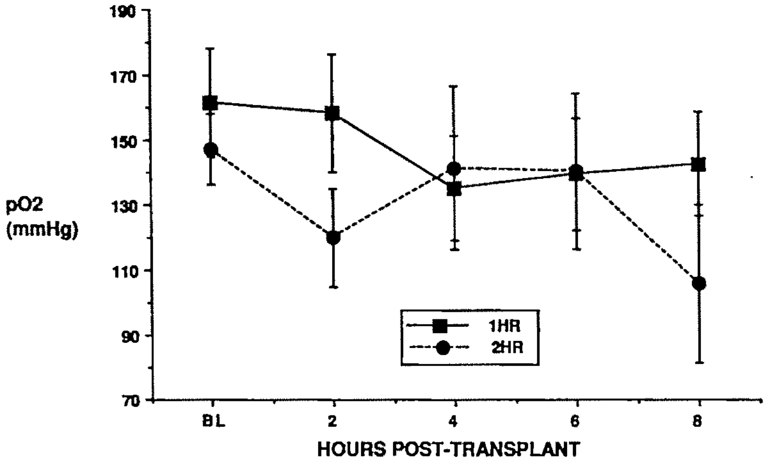


Fig 3. Arterial oxygen tension (pO_2) *for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean).* (BL = *baseline.)*

mal. Three of these specimens were from lungs that provided good gas exchange (2 one-hour recipients and 1 two-hour recipient). One of these specimens was consistent with pulmonary hypertension of long-standing duration in a lung from a 2-hour cadaver. The recipient of this lung died of right ventricular failure and pulmonary hypertension. Examination of the control biopsy specimen taken from the nontransplanted right lung also showed smooth muscle hypertrophy involving all arterioles in the specimen. In addition to these abnormalities, the transplanted lung showed histologic evidence of vascular congestion and intravascular thrombus. Preexisting pulmonary vascular disease in this animal is likely, therefore, based on the histologic findings, and probably contributed to the demise of the recipient. Otherwise, there was little correlation between severity of histologic abnormalities and physiologic outcome. In particular, aside from the 1 animal noted to have pulmonary vascular changes before transplantation, there were no light microscopic features of control lung specimens that predicted physiologic variables of the subsequently transplanted lung.

Comment

There are four strategies that might be effective in increasing the size of the pulmonary donor pool. First, programs of public education to increase awareness of transplantation and to address the reticence in the community to organ donation are necessary and may improve the retrieval of organs in general. The concept of presumed consent, currently in vogue in Europe, is unlikely to be introduced in this continent for some time because of the specter of human rights violation.

The second option is to relax the physiologic variables currently used to exclude donors. It is not known to what extent neurogenic pulmonary edema is reversible, and it would be reasonable to conduct experiments to answer this question. However, in the clinical realm, the distinction between neurogenic pulmonary edema and gas exchange dysfunction secondary to aspiration or infection is difficult to make. Accordingly, most lung transplant surgeons would use a less-than-optimal lung only in extreme circumstances.

A third strategy that merits further investigation is the use of xenografts, but our current understanding of immunology and immunosuppressive agents precludes the clinical use of xenografts at this time.

A fourth strategy is one that is the subject of this study. The concept of harvesting lungs from cadavers after cessation of circulation is based on the knowledge that lung tissue can be retrieved from cadavers and cell culture lines can be successfully established from such material [6]. We hypothesize that pulmonary cellular elements

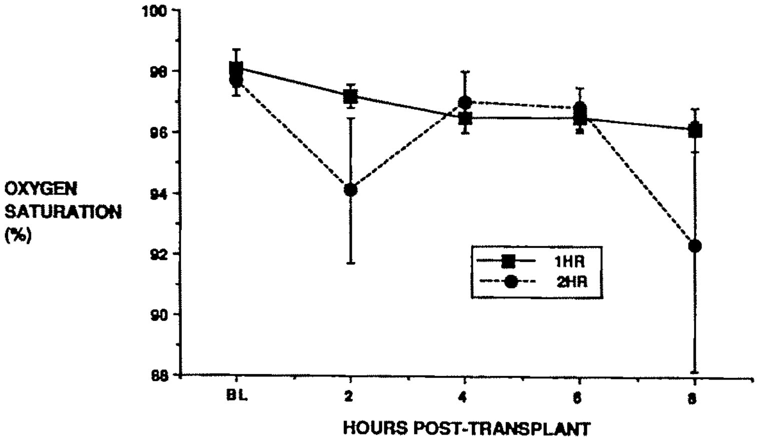
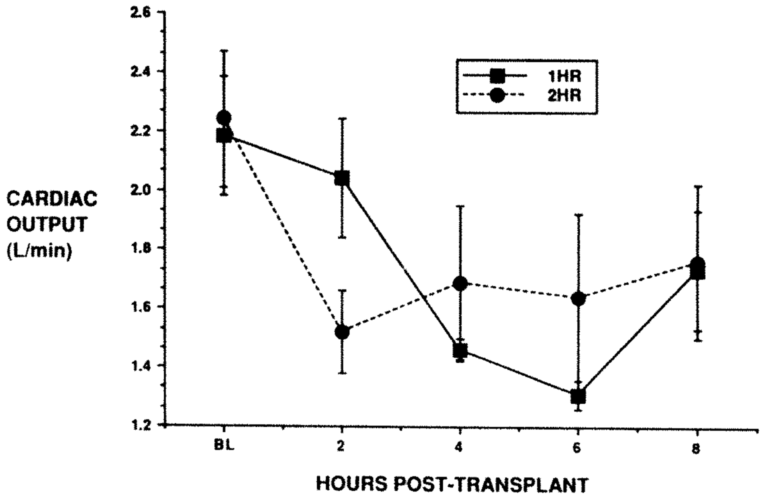


Fig 4. Arterial oxygen saturation for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean). (BL = *baseline.)*

Fig 5. Cardiac output, as measured by a left pulmonary artery magnetic flow probe, for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean). (BL = *baseline.)*



remain viable for a variable period after cessation of circulation. This viability is related to a unique aspect of lung physiology.

Whereas other organs rely on perfusion for the cellular function of respiration, in the lung, respiration at a cellular level can be accomplished in the absence of perfusion. Perfusion remains necessary for the continued availability of substrates for metabolism and for the removal of metabolic byproducts.

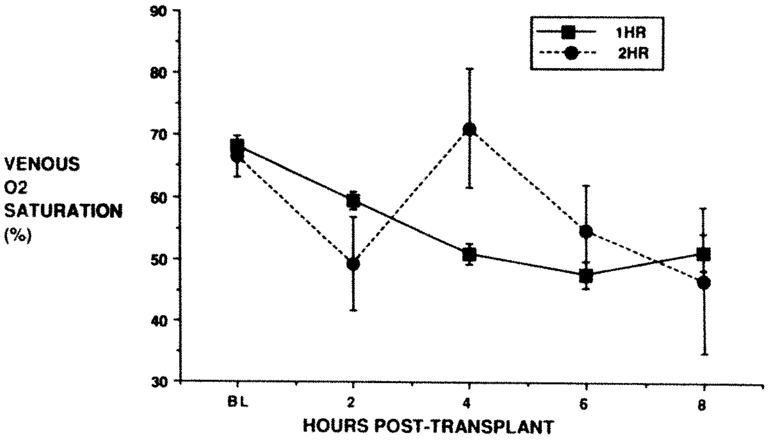
Little is known about the duration of the lung's viability after death. In 1952, Blades and associates [10] reported on their initial experiments with in situ occlusion of PA, veins, and bronchus in a canine survival model. The lungs of 3 of 4 animals tolerated 300 minutes of this type of "ischemia" at normothermia. A subsequent study [11] demonstrated considerable impairment of gas exchange in dogs dependent on lungs rendered ischemic for periods of 90 minutes at normothermia. This was warm ischemia, however, with the lungs rendered ischemic in situ.

The issue was addressed again in 1968 by Homatas and colleagues [12]. In this study, dogs were sacrificed and left at room temperature, mechanically ventilated with room air, for various periods up to 6 hours before excision of the lungs. Gas exchange function was determined using an isolated, perfused lung model. Gas exchange was evaluated for 3 hours or until pulmonary edema occurred. All 16 lungs harvested 2 hours or less after death demonstrated reasonable gas exchange. Beyond 2 hours, pulmonary edema was a more frequent occurrence. One difficulty with the interpretation of this study was the routine flushing of the pulmonary vasculature with heparinized Ringer's lactate solution before evaluation. Most studies of pulmonary preservation have demonstrated poorer lung function when the perfusate employed to cool the lungs has an ionic composition similar to extracellular fluid, such as saline or Ringer's lactate solution [13, 14].

A different approach was taken by Robicsek's group [15]. They harvested heart-lung blocks from cadavers and resuscitated the heart using extracorporeal oxygenation. Subsequently, the autoperfused heart-lung preparation was maintained for varying periods of time. Although it was cardiac function that was the primary subject of this study and that was believed to be the limiting factor in this model, the concept owed its success to satisfactory pulmonary function as well as cardiac function.

Early efforts at human lung transplantation involved the use of lungs from donors whose circulation had

Fig 6. Mixed venous oxygen saturation, drawn from a pulmonary artery catheter, for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean). (BL = *baseline.)*



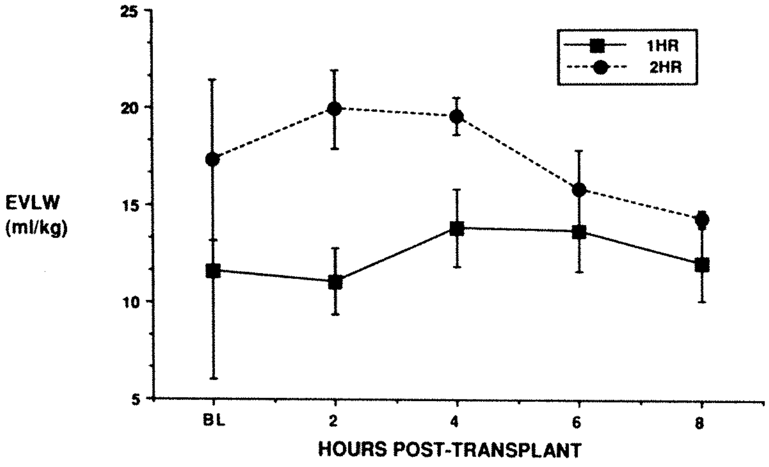


Fig 7. Extravascular lung water (EVLW), *determined by thermodilution, for recipients of 1-hour and 2-hour cadaver lungs (mean ± standard error of the mean).* (BL = *baseline.)*

recently stopped or was allowed to stop in the operating room [16, 17]. These early transplant efforts preceded the development of concepts relating to brain death. Currently, all clinical lung transplant programs use lungs recovered from brain-dead donors with intact circulation, except for the occasional "live related donor," only recently performed successfully (Starnes V; personal communication). Most clinical lung transplant programs are using some form of perfusion for cooling. Our practice is to flush the lungs with modified Euro-Collins solution at the time of circulatory arrest in the donor.

Organ retrieval from brain-dead donors maintained on mechanical ventilation poses some problems for lung transplant surgeons. For several reasons, the lung is generally the first organ to deteriorate after brain death. First, a considerable number of trauma victims aspirate gastric material into the airway at the time of intubation. Second, intubation and mechanical ventilation bypass the upper airway protective mechanisms and predispose to airway bacterial contamination, which can lead to purulent tracheobronchitis or pneumonia. Third, "neurogenic pulmonary edema" is a poorly understood but very serious accompaniment to some patients with major head injury. There is some evidence that this phenomenon is related to excess circulating catecholamines [18], but little effort has been directed at ascertaining the reversibility of this pulmonary lesion. Fourth, thoracic trauma is a frequent accompaniment to closed head injury resulting in brain death. This in turn can lead to pulmonary contusion, which is a relative contraindication to the use of lungs for transplantation. Finally, the massive crystalloid infusion that is given to donors with head injuries and diabetes insipidus can adversely affect pulmonary function, resulting in pulmonary grafts that are not useful for transplantation.

Clearly, a number of these problems could be circumvented if it were feasible to harvest lungs from cadavers after circulation has stopped. For patients who are pronounced dead shortly after arrival in the emergency room, there has been no antecedent period of mechanical ventilation and intubation and no time for the development of neurogenic pulmonary edema. Aspiration, which may have occurred before death outside the hospital, could be recognized bronchoscopically. In short, if it is feasible to transplant lungs from cadavers who are pronounced dead in emergency rooms, then there is a sizable potential donor pool for lung transplant recipients.

It has been estimated that more than 100,000 deaths occur annually in the United States because of trauma. According to a study by Baker and associates [19], more than half of these deaths occur at the scene or in the emergency room. Although many of these victims might have associated chest injuries, brain injury was believed to be responsible for more than half of the early deaths. Accordingly, many of these victims could be suitable organ donors if retrieval from cadavers were safe.

This study was designed specifically to address the question: How long after cessation of circulation and death is it feasible to use current methods to harvest lung and anticipate good gas exchange function? Clearly, for the concept of cadaver lung transplantation to be practical, there will be a time between death and retrieval of tissue for storage that must be long enough to allow for informing next of kin, acquiring consent, and assembling a team to harvest the organs. The limit to tolerable in situ ischemia for lungs must be long enough for these logistical issues to be resolved. Our study provides encouraging data in support of the concept, in that all recipients of 1-hour cadaver lungs demonstrated excellent gas exchange and hemodynamics after transplantation. Gas exchange was very good in some animals that had received a 2-hour cadaver lung. Two hours is likely a minimum practical period for the retrieval of lungs from patients who die shortly after admission to emergency rooms, but it would be ideal to have a longer time frame.

Conceivably, more recipient animals might have survived if we had increased inspired oxygen fraction or not excluded the other lung from circulation. However, we chose to maintain a constant inspired oxygen fraction of 0.4 to eliminate animals that developed a high arterial-alveolar gradient if transplanted with a cadaver lung. This model sets a high performance standard for the transplanted lung, because it subjects the lung to the entire cardiac output and requires good gas exchange for animal survival.

It is likely that modified Euro-Collins solution is not the ideal solution for pulmonary preservation. We have demonstrated that the addition of a free radical scavenger, dimethylthiourea, ameliorates lung injury observed with 24-hour hypothermic storage [7], and the addition of dimethylthiourea to modified Euro-Collins solution significantly improved gas exchange and pulmonary hemodynamics in animals transplanted with lungs preserved for 12 hours [8].

Indeed, if the cellular function of respiration can be achieved in the lung across the gas phase as we have surmised, then it is likely that ventilation of cadavers may increase the tolerable period of ischemia. None of our 1-hour, 2-hour, or 4-hour cadaver animals were ventilated until *after* these time periods, and then only long enough to allow perfusion of the lung block with modified Euro-Collins solution. If the tolerable period of ischemia can be increased so that reliable gas exchange function is observed for periods longer than an hour, then it may be realistic to consider eventually expanding the pulmonary donor pool using this resource.

There is currently considerable interest in the potential use of kidneys and livers from cadavers [20, 21]. Our current results support the concept that retrieval of lungs from human cadavers may become a practical method which may have a substantial impact on the size of the current pulmonary donor pool. Ideally, a histologic or physiologic variable will be identified in future studies to enable prediction of a favorable outcome with a lung from a given cadaver. Innovative approaches to the problem of donor shortage are necessary if lung transplantation is to be widely applied.

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DISCUSSION

DR G. ALEXANDER PATTERSON (Toronto, Ont, Canada): I enjoyed the presentation. This is an interesting idea. We have some information from our own laboratory that shows that ostensibly dead lungs continue to take up ^{3}H thymidine and make DNA for some hours after harvest without any flush at all. So I think the idea that these are viable lungs for some period of time is quite correct. However, I would be interested to know the state of ventilation during the period of time between sacrifice and harvest and also the state of ventilation of the lung after you intubated the animal and ventilated it for preharvest. We have some interesting information that suggests that it is the state of ventilation or the degree of inflation of the lung that is more important in terms of posttransplantation function than is the solution used to flush it with or the vasodilator you use or whatever.

DR EGAN: We put the animals to sleep and then let the lungs fall to FRC. We did not intubate the animals until the intervals alluded to, 1, 2, and 4 hours, and at that point we would ventilate the donors with a tidal volume of 15 mL/kg and a rate of 8 per minute, essentially to allow us to perfuse the lungs with Euro-Collins solution, because we believed we could not adequately perfuse a nonventilated lung.

DR PATTERSON: But did the lungs ever get hyperinflated?

DR EGAN: No, not until we placed them in the chest of the recipient and completed the transplantation, and then we would hyperinflate them to get rid of the atelectasis, which was a part of every procedure. The lungs were allowed to collapse down, just as we do in a typical human harvest. Once the pulmonary flush was through, we disconnected the ventilator and allowed the lungs to collapse, and they were essentially stapled at the trachea in a semiinflated state.

DR FRANCIS ROBICSEK (Charlotte, NC): I think this presentation was predictable. At first we accepted only "ideal" donors. Later we also accepted "nonideal" donors and then donors who were in shock or septic. And now we are considering accepting "dead" donors! To me, this report is most interesting because it closely matches our own observations published in the *Archives of Surgery* (1969;99:750–2). At that time we exsanguinated dogs and, after the occurrence of clinical death, we removed the heart and lungs en bloc, "resuscitated" them using the pump oxygenator, and then converted them into an autoperfusing heart preparation. We found results very similar to those of Dr Egan and associates, ie, up to 2 hours after clinical death we had excellent recovery with pump oxygenator resuscitation and then conversion into heart-lung preparation. After 3 hours of maintaining the heart in this artificial state, we had no difficulties, but after 4 hours of clinical death we could not get either the hearts or the lungs functioning properly.

We also did this method on 5 human cadavers from which the hearts and lungs were removed after clinical death. The organs were resuscitated with the pump oxygenator, and in 4 of 5 we got excellent cardiac and pulmonary function later on.

It gives me special pleasure that, in your very elegant and sophisticated experiments, you came to similar results.

DR EGAN: Thank you, Dr Robicsek. In fact, we were able to reference your very seminal contribution in the manuscript.

DR HANI SHENNIB (Montreal, Que, Canada): I enjoyed the report. Obviously lung cells are monolayered, so it makes a lot of sense that if you give adequate oxygen and CO_2 and have adequate substrate, the lung can endure a long time. The question becomes not what the parenchyma will do but actually what the vessels and the bronchus will do, and that is something that will have to be looked at later on.

I have a small clinical experience that may actually enforce your experience in the laboratory, which I did not think about until I saw your report today. About a year ago we were caught late trying to send our team to get the lung, and by the time we arrived, the heart was separated, the liver team was packing the liver, and the lung was left in the chest for about 20 minutes before we got in. By the time we flushed the lung and so on, it was another 10 or 15 minutes. So by definition the donor was a cadaver and the lung had been there for 30 minutes, and the lung functioned very well. This lung not only was 30 minutes without perfusion but in addition it was from a carbon monoxide poisoning victim. This reflects that we are really pushing the donor pool, I think, to the edge. The lung worked very well, and the patient is about a year after transplantation.

DR DENNIS MODRY (Alberta, Canada): I enjoyed the report as well, but I have one cautionary remark. Many of us realize, in harvesting hearts and lungs with our colleagues below the diaphragm, that those retrieving the kidneys usually do not like to tolerate more than a nanosecond of ischemia. And part of the reason for that is there is some evidence in the renal transplant literature that ischemic injury to the kidney is associated later on with a greater frequency of rejection episodes. Whether that is true in the lung or not remains to be seen.

DR EGAN: I am sure these findings will have to be confirmed in a survival model looking at long-term lung function as well as bronchial healing.

DR JOEL D. COOPER (St. Louis, MO): From a critical standpoint, really what you have shown is that the lung will tolerate an hour of warm ischemia and maybe two. Now, there are a number of models that have shown that. What concerns me is not what happens after the perfusion has stopped, but what has happened to that lung before the perfusion has stopped, that is, if the donor is potentially in any period of shock or hypotension. I know of at least one animal model where 7 minutes of anoxia with persistent circulation led to trashing of the lungs when they were subsequently preserved and stored. So although you have demonstrated that warm ischemia is tolerated, and I think other models have done so, how do you see this fitting in clinically? I think that what happens before cessation of circulation, ie, any period of hypotension, may in fact be more critical than what happens afterwards.

DR EGAN: Well, you are quite right, and we obviously wanted to address this in a very controlled situation in an effort to address one question at a time. The first question is, when does the lung cease to be transplantable? We still have not answered that question.

With respect to the issue of when will this be clinically relevant, we have recently been exposed to two potential donors in our intensive care unit who did not meet the criterion for brain death in that they still had brain stem reflexes, but the family requested that support be terminated. The dilemma here is that we were unable ethically to take that patient to the operating room as an organ donor because he was not "brain dead"; the patient was disconnected from life support and was not used as a donor. In both cases, those lungs were functioning very well, and it would appear to me that this would be one way to introduce this principle clinically. We see a fair number of patients with gunshot wounds to the head in whom there has not been a lot of time for hemorrhage to occur but, rather, death was fairly instantaneous, and that is another population of patients that may be very amenable to harvest based on these principles.

DR COOPER: Well, you have introduced, I think, a very, very stimulating topic, and we appreciate that very much.

Ambulatory Mediastinoscopy and Anterior Mediastinotomy

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Ambulatory facilities are being used more and more for various diagnostic and therapeutic procedures. We report 158 consecutive mediastinoscopies and anterior mediastinotomies performed in an ambulatory setting from July 1981 to February 1990. There were 120 patients with a malignancy: 114 bronchogenic carcinomas, 4 lymphomas, 1 teratocarcinoma, and 1 carcinoma of the stomach. Thirty-eight patients had a benign condition, including sarcoidosis in 27 and miscellaneous diagnosis in 11. Twenty-two patients (14%) were admitted the same day: 9 for elective operation in view of bed availability, 8 for medical observation, and 5 for overnight admission for nonmedical reasons. Six nonfatal complications were encountered: hemoptysis (2), atrial fibrillation (1), pneumonia (1), mediastinal self-contained bleed (1), and tear of a pulmonary artery (1). There was no operative mortality. Overall, ambulatory mediastinoscopy and anterior mediastinotomy permitted a diagnosis in 47 patients (20%) and confirmed unresectable malignant disease in 29 patients, thus sparing unnecessary admission to a surgical ward in 76 (48%) of the 158 patients. Mediastinoscopy and anterior mediastinotomy can be safely performed in an ambulatory setting and do alleviate the need for hospitalization in a substantial number of patients.

(Ann Thorac Surg 1991;52:1122–6)

Ever-diminishing resources in a universal medicare system, associated with better instrumentation and techniques, have broadened the field of ambulatory care in Canada [1–4]. In the United States as well, increasing costs have favored the development of both hospital-integrated and free-standing ambulatory surgical units without a decrement in the quality of patient care [5–7]. Facing a severe shortage of hospital beds, the Department of Chest Medecine of Hôpital du Sacré-Coeur de Montréal created in late 1980 an outpatient diagnostic section to allow investigation of many pulmonary conditions previously needing hospitalization. Mediastinal nodal sampling for diagnosis or staging was often required. As a consequence, outpatient mediastinoscopy was initiated using day surgery facilities. We report our experience with mediastinoscopies and anterior mediastinotomies performed in this setting.

Material and Methods

Facilities

L'Hôpital du Sacré-Coeur de Montréal is a university-affiliated tertiary care center with an inpatient capacity of 714 beds. Since 1981, a fully integrated 21-bed ambulatory surgical unit has been operational using hospital facilities including the operating and recovery rooms. In addition, to facilitate accessibility of this unit to patients residing some distance away, a hostelry service was established within the grounds of the medical center in February 1989.

Patient Selection

Only patients referred to us by the respirologist of the outpatient diagnostic section of the Department of Chest Medicine of our hospital underwent mediastinoscopy in the ambulatory mode. During the same period there was a number of hospitalized patients who also had a mediastinoscopy, but these are not included in this report. Hospitalized patients were either not fit to have an outpatient investigation, did not have the possibilities to travel back and forth to the hospital, or lived too far away from the hospital.

Patients were thus examined by a chest physician and a decision was made as to the suitability of candidates for the outpatient diagnostic section according to their health status, their acceptance of the outpatient program philosophy, and their capability and easiness in accessing the hospital. Referral for ambulatory mediastinoscopy by the investigating respirologist usually followed baseline investigation, which included routine blood work, sputum studies, urinalysis, chest imaging, and electrocardiography. Respiratory function tests, arterial blood gases, nuclear scans, bronchoscopy, transthoracic needle biopsies, and preoperative cardiology consultation, when needed, had usually been performed before referral as well. Charts of patients needing mediastinoscopy were screened by the thoracic surgeon and the anesthetist before the patients were accepted for ambulatory mediastinoscopy. A patient considered fit for outpatient mediastinoscopy by the investigating chest physician was

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rarely turned down by the thoracic surgeon or the anesthetist.

Day of Operation

Patients are instructed to fast for a minimum of 8 hours before operation. They are asked to arrive at the hospital early the morning of the operation. This permits a first encounter with the surgeon and the anesthetist and allows completion of the preoperative workup if needed. We have also obtained preoperative consultations in subspecialties before operation on a few occasions. Discharge is scheduled late in the afternoon of operation. Before discharge, every patient is seen by a member of the surgical team. Depending on the results of the frozen section analysis, patients are given an appointment for follow-up in 1 week or arrangements are made for future admission to the surgical service.

Patients

From July 1981 to March 1990, 783 patients have undergone complete investigation within the outpatient diagnostic section. Of these 158 (20%) have required assessment of mediastinal lymph nodes. There were 125 cervical mediastinoscopies, 20 anterior mediastinotomies (19 left, 1 right), and 13 combined cervical mediastinoscopies and left anterior mediastinotomies performed in the ambulatory setting. For the purpose of discussion, these will be grouped in one category referred to as "mediastinoscopy." Case records of all patients were reviewed.

The operative risk of each patient was prospectively evaluated by the anesthetist before operation using the American Society of Anesthesia (ASA) classification [8, 9]. Follow-up at 1 week or more was available for all patients. The techniques of mediastinoscopy and anterior mediastinotomy have been described elsewhere [10–12]. Statistical analysis used χ^2 analysis with the Yates modification when appropriate.

Results

One hundred fifty-eight patients underwent mediastinoscopy on an ambulatory basis over the 9-year period. This represents 21% of the total mediastinoscopy patient population seen in our hospital during the same period. There were 119 men and 39 women. The average age was 54.9 years (range, 19 to 76 years). Forty patients (25.3%) were older than 65 years.

The majority of patients had, other than their pulmonary disease, a good general condition. Eighty-eight patients (56%) were classified as in physical state ASA I, 40 (25%) ASA II, and 30 (19%) ASA III.

One hundred thirty-six patients (86%) lived within a 40-km radius from our hospital, 8 (5%) within 40 to 100 km, and 14 (9%) further than 100 km. The latter were usually accommodated by relatives living nearby or, more recently, have used our hostelry facilities.

On average, patients spent 7.9 hours in hospital during the day of operation, ranging from 4.5 to 11 hours.

Over the years, there has been a gradual increase in the number of patients investigated within the outpatient diagnostic section, resulting in a steady increase in the number of ambulatory mediastinoscopies. In the last year, of 71 mediastinoscopies performed within our service, 36 (51%) were ambulatory. Hospitalization was considered necessary for 35 patients (49%) because of traveling distance varying from 100 to 650 km for 13 patients and because of poor general condition or associated diseases needing further investigation or treatment for 20 patients. Two patients were already hospitalized in another section at the time of consultation.

Table 1. Final Pathological Diagnosis of Conditions Investigated by Ambulatory Mediastinoscopy at Hôpital du Sacré Cœur de Montréal, 1981–1990

Diagnosis	No. of Cases
Malignant	
Lung cancer	114 (72%)[a]
Lymphoma	4
Gastric cancer	1
Teratocarcinoma	1
Benign	
Sarcoidosis	27 (17%)
Benign adenopathy	6
Bronchogenic cyst	1
Benign nodules	3
Unknown	1 (1%)
Total	158

[a] Percentage of the total group studied.

Pathology

Final diagnosis demonstrated malignant disease in 120 patients and benign conditions in 37. One patient with a suspected lung cancer refused further investigation and was lost to follow-up after a nondiagnostic mediastinoscopy (Table 1).

Of the 120 patients with proven malignancy, mediastinoscopy showed metastatic or primary malignant nodal disease in 43, confirming a preoperative diagnosis of cancer in 26 and establishing a histological diagnosis in 17. In 3 of the latter, it modified the premediastinoscopy histological diagnosis from non–small cell to small cell lung carcinoma.

All 27 patients with suspected sarcoidosis had had nondiagnostic bronchoscopy and transbronchial biopsies before referral for mediastinoscopy. The latter was diagnostic in 26 patients, being falsely negative in 1 patient in whom biopsy specimens could not be obtained owing to the dense mediastinal fibrosis. Histological proof of sarcoidosis was obtained 2 months later by scalene node biopsy.

Sarcoidosis or lymphoma was suspected in 6 other patients in whom mediastinal biopsy specimens showed benign nodal changes. Follow-up in all 6 patients confirmed the benignity of these changes.

Four patients underwent mediastinoscopy in view of the radiological presence of a mass of unknown nature on

Table 2. Complications After Ambulatory Mediastinoscopy (n = 6)

Year	Sex	Age (y)	ASA	Procedure Type	Procedure Results	Complication	Outcome
1984	M	65	3	CM	Pos	Cervical hematoma	Observed; home day 7
1985	M	70	2	CM	Pos	Rapid atrial fibrillation	Pharmacological control; home day 2
1987	M	76	1	B/CM/AM	Neg	Hemoptysis	Self-contained; elective thoracotomy day 6
1987	F	65	2	AM	Neg	Pneumonia	Elective admission on day of mediastinoscopy for thoracotomy; pneumonia developed while waiting in hospital; elective thoracotomy day 10
1988	M	60	1	B/CM/AM	Neg	Hemoptysis	Self-contained; elective operation day 4
1989	M	69	1	CM	Neg	Right pulmonary arterial branch tear	Controlled by tamponade; elective operation day 2

AM = anterior mediastinotomy; ASA = American Society of Anesthesic classification; B = bronchoscopy; CM = cervical mediastinoscopy; Neg = negative; Pos = positive.

chest radiography. Fine-needle aspiration at mediastinoscopy confirmed the presence of one paratracheal bronchogenic cyst. The other 3 patients were shown to have benign pulmonary nodules at thoracotomy.

Overall, ambulatory mediastinoscopy permitted a histological diagnosis in 50 patients (malignant 17, benign 33) and confirmed unresectable malignant disease on 26 occasions, thus sparing unnecessary admission to a surgical ward in 76 (48%) of the 158 patients.

Hospitalizations

Twenty-two patients (14%) were admitted the same day: 9 for elective operation in view of bed availability and 8 for medical observation necessitated by either slow recovery from the anesthetic (n = 3) or the occurrence of a postoperative complication (n = 5). Five patients stayed overnight for humanitarian reasons: lack of transportation (n = 1), late start of operation (n = 1), or at the patients' request (n = 3).

Complications

Six nonfatal complications (3.8%) were encountered (Table 2). All of the complications were diagnosed before discharge. Follow-up at 1 week did not reveal any delayed complications. Five patients required admission for a period longer than 24 hours. Five of the 6 patients with complications were older than 65 years ($p < 0.005$). Half of the patients who sustained complications had had anterior mediastinotomies, but this did not reach statistical significance. Preoperative assessment of the physical state by the ASA classification did not help predict patients at risk for complications. There was no operative mortality.

Comment

Airway and upper gastrointestinal endoscopy with tissue sampling or dilation, as well as manometric or pH studies of the gastroesophageal area, represent most of the thoracic surgical work done in ambulatory facilities. We have already reported our experience with the ambulatory care of pneumothoraces [1, 2]. Herein, we report our experience with mediastinoscopy performed in such a setting.

In Canada, within a universal medicare system, health care resources are becoming less and less abundant. For physicians as well as surgeons one particular area that is being hit repeatedly is the number of beds available for the investigation as well as for the treatment of patients. As a matter of fact, shortage of hospital beds was the main reason for commencing some years ago an outpatient investigation program with ambulatory mediastinoscopy. It thus permits a certain optimization in the use of in-hospital facilities, preferentially to treat rather than to investigate patients. It has allowed patients to avoid prolonged periods of waiting.

The cost-benefit issue has been well established in previous reports comparing ambulatory and in-hospital operations for various procedures [4, 5, 7, 9, 13]. Because this review extends over a period of 9 years, during which costs have increased significantly, it is impossible to retrospectively analyze the real cost savings realized during the whole period with our use of the outpatient approach for mediastinoscopy. Furthermore, in our Canadian medicare system, the sole payor is the government; subsidies to hospitals are given in the form of a global yearly budget, and costs are evaluated on a "per diem" basis for hospitalized patients. Presently, at our hospital there is a major difference in cost for a 12-hour-day operation stay ($226.00 Canadian) versus a 24-hour inpatient stay ($426.00 Canadian). Physicians' fees for working with ambulatory patients are identical to those for working with a hospitalized population, and preoperative workup is the same for both groups. We estimate that because many patients who undergo mediastinoscopy, in fact, stay 48 hours in the hospital from admission the night before to discharge the following day, a 73% cost saving is accomplished at this level every time a mediastinoscopy is done in an ambulatory setting. Also, there are cost benefits deriving from the outpatient rather than the in-hospital investigation of various pulmonary conditions by the respirologists. In 1990 they undertook the workup of 220 patients in their outpatient section. But it is not within the scope of this report to analyze these benefits.

One other potential advantage of outpatient operation is a reduction in postoperative infection rates [5, 7, 9, 14]. Mediastinoscopy and mediastinotomy are usually clean operations and thus carry a very low risk of wound infection. The only infectious complication encountered in this series involved a patient admitted on the day of mediastinoscopy for elective thoracotomy. A lobar pneumonia developed within 24 hours of her admission. Avoidance of prolonged hospitalizations could be beneficial to many mediastinoscopy patients who suffer from chronic obstructive lung disorders, reducing their risk of postoperative nosocomial pulmonary infections.

We do not recommend that this type of ambulatory operation be performed within free-standing ambulatory surgical centers. Even though, in this series, we were not faced with complications requiring immediate thoracic exploration, these are inherent to the technique, can be life threatening, and probably contraindicate performance of mediastinoscopy outside of hospital-based ambulatory surgical units.

Other points and precautions should be stressed. Back-up facilities to permit hospitalization or overnight stay are mandatory, although with experience, the number of patients requiring admission either for observation or for nonmedical reasons (lack of transportation, escort, or family) may be reduced to a minimum. In particular, overnight stays for humanitarian reasons will be almost completely eliminated with good individualized preparation of each patient during a previous visit where each step of the operation and of the day of operation are discussed. Informative documentation is also helpful. During the same visit, it should be stressed that ambulatory operation is conditional to the presence of a responsible adult who will accompany the patient home after operation and similarly for the presence of someone to care for the patient at home the evening of operation.

Patient selection also contributes to the success of ambulatory surgical programs, but definite criteria for selecting eligible candidates have not yet been defined [8, 15]. Contrary to popular belief, the extremes of age are not thought to be a deterrent in the selection of ambulatory surgical patients [8, 15]. In our series, however, 5 of the 6 complications encountered were in patients 65 years old and older. This is statistically significant; however, small numbers were involved in the statistical analysis. We have, however, included complications such as hemoptysis and iatrogenic pulmonary arterial tear that relate more to the surgical technique itself than to the patient's overall general condition. We continue not to exclude patients of the elderly age groups based on chronological age alone, although awareness of these statistics has led us to be more cautious in operating on these patients.

The presence or absence of associated illnesses could be of some importance in selecting patients for ambulatory operation. Of the 36 patients who had in-hospital mediastinoscopies in the last year, 20 had been admitted because of associated illnesses needing investigation or treatment. Meridy [15] restricted outpatient surgical procedures to patients considered healthy (ASA I) and to patients with mild to moderate systemic disturbances (ASA II). Others have included patients in less healthy ASA classes without an increase in morbidity or mortality [8, 9, 14]. Most patients in our series were considered ASA I and ASA II (81%). Thirty patients were classified ASA III mostly because of underlying chronic obstructive pulmonary disease (n = 20) or stable coronary artery disease (n = 10), and they tolerated the operation without any related complications. Every patient with suspected or known coronary artery disease had been evaluated by a cardiologist before referral for outpatient mediastinoscopy.

The average stay in the hospital for our patients was 8 hours, recovery time being about 5 to 6 hours. Scheduling operation in the morning should permit ample recovery time for discharge in late afternoon or early evening.

We routinely use frozen section pathological analysis of the material sampled. Initial results can thus be transmitted to the patients before discharge. According to the results, arrangements for admission to the surgical service can also be initiated or, when needed, same-day consultation and referral to consultants in medical oncology and radiotherapy services can be made.

In conclusion, we have shown that, in experienced hands, mediastinoscopies and anterior mediastinotomies can be done safely in an ambulatory setting. Appropriate patient selection is important. Ambulatory mediastinoscopy will alleviate the need for unnecessary admission to thoracic surgical wards in a substantial number of patients.

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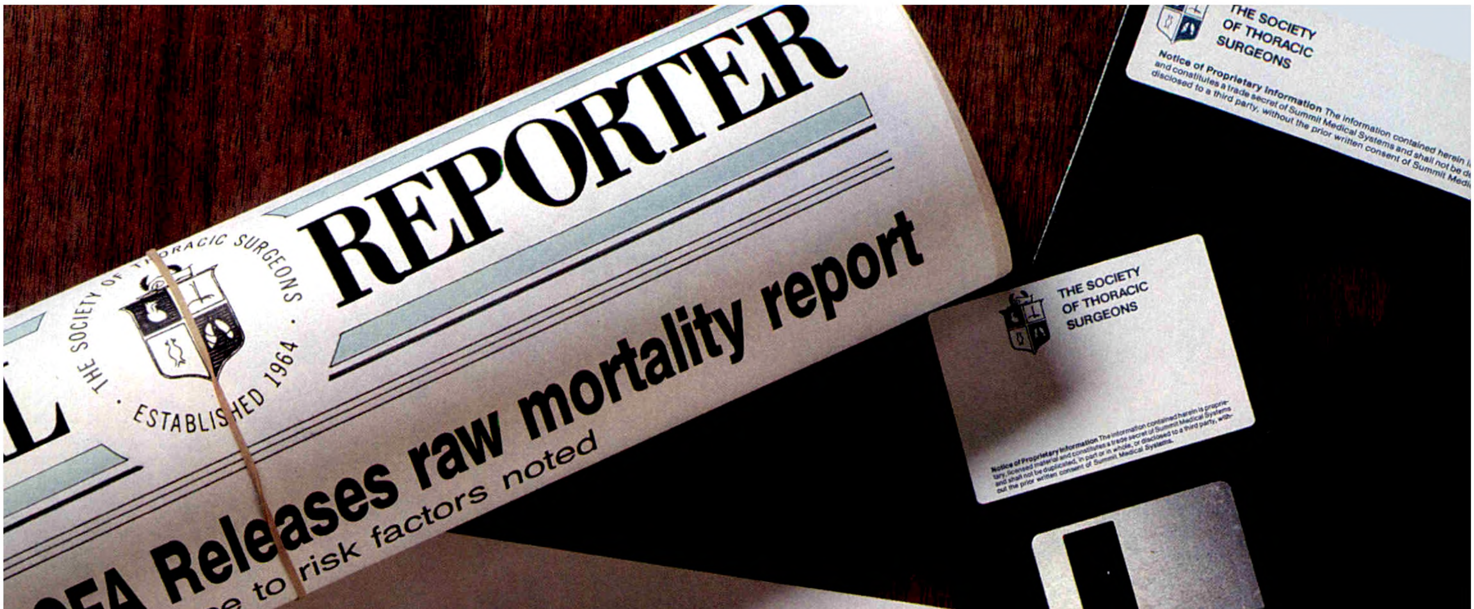
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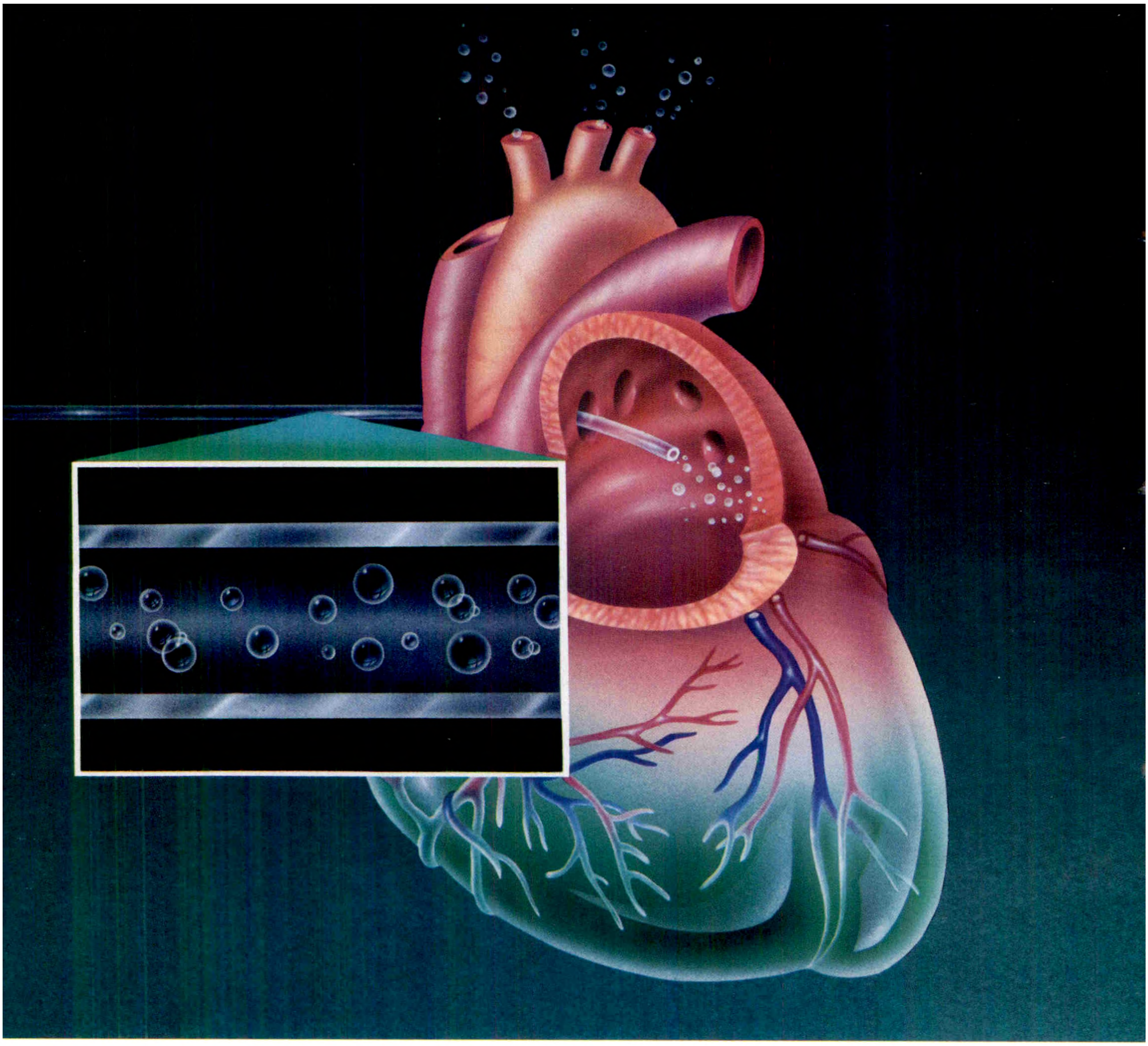

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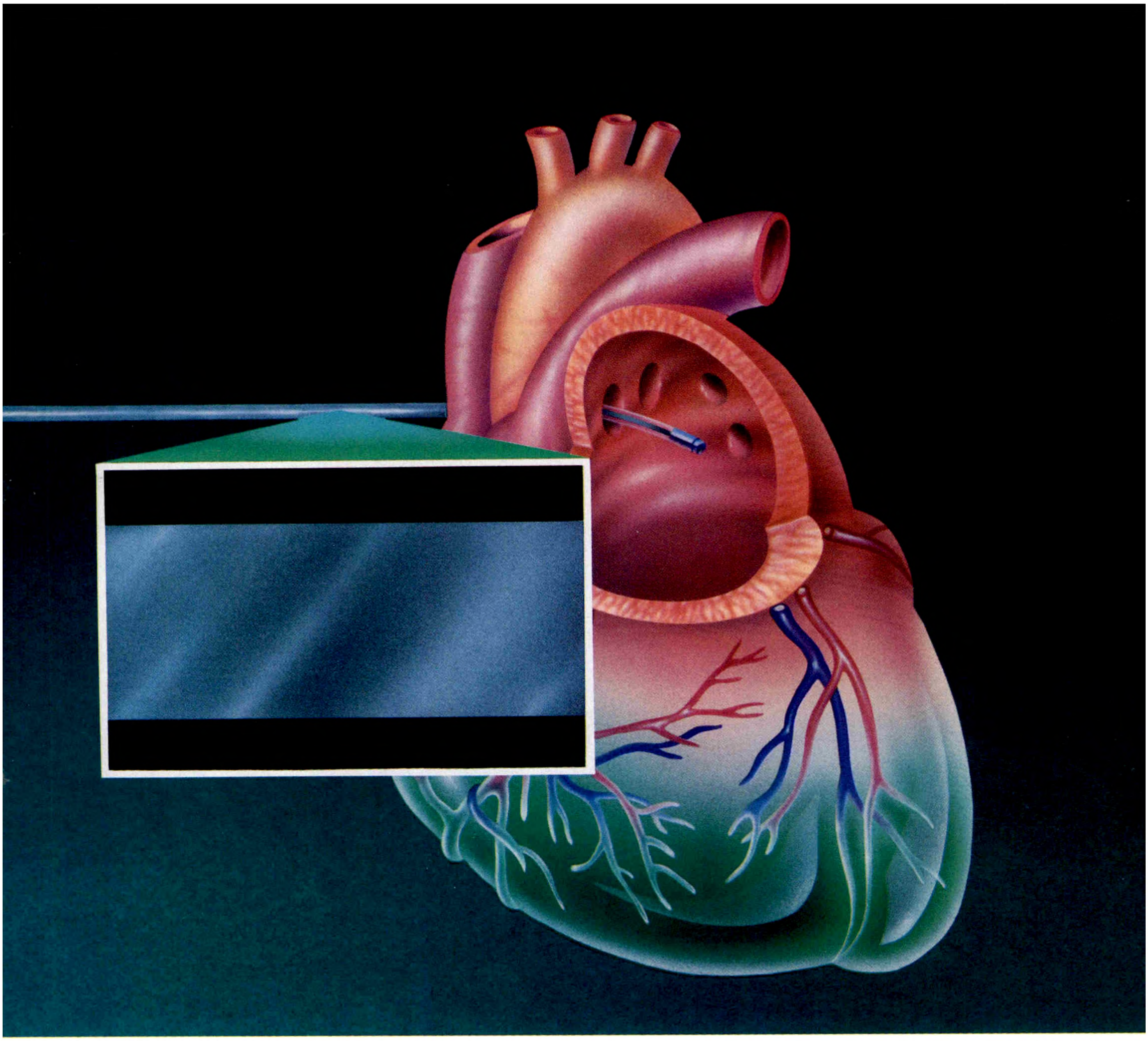

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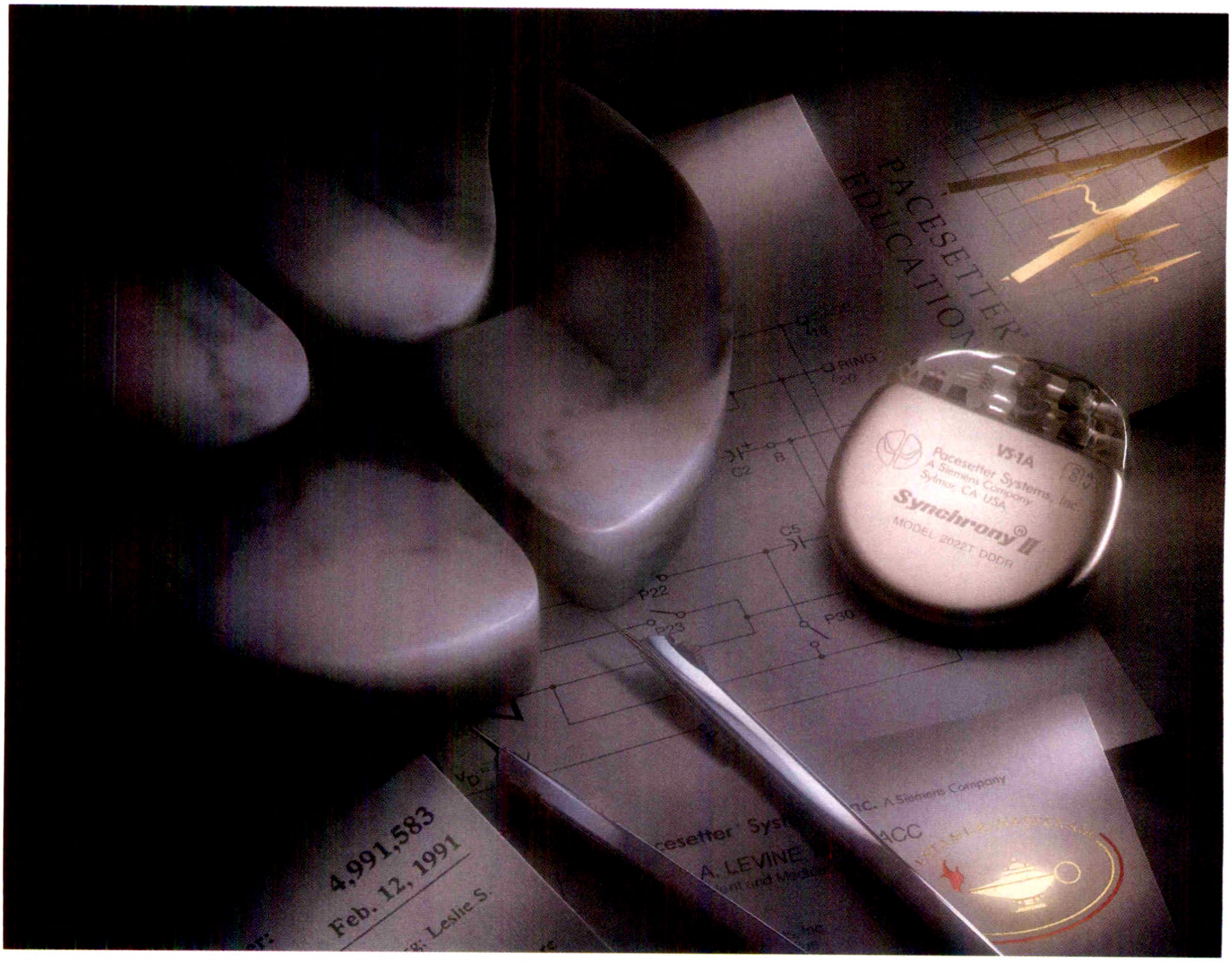

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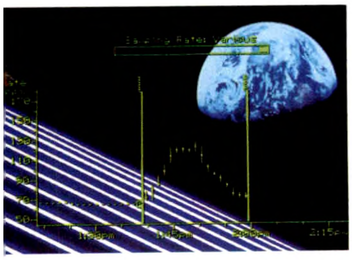

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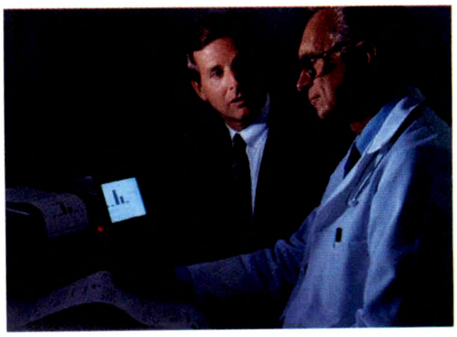

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Primary Cardiac Valve Tumors

Fred H. Edwards, MD, Douglas Hale, MD, Amram Cohen, MD,
Lenardo Thompson, MD, A. Thomas Pezzella, MD, and Renu Virmani, MD

Department of Surgery, Walter Reed Army Medical Center, Washington, DC; Uniformed Services University of the Health Sciences, Bethesda, Maryland; and Armed Forces Institute of Pathology, Washington, DC

To investigate the characteristics of primary cardiac valve tumors, we retrospectively analyzed our multiinstitutional experience from 1932 through 1990. We encountered 56 valvular tumors in 53 patients. The average age of these patients was 52 years (range, 2 to 88 years) and 79% (42/53) were male. Symptoms were present in 38% (20/53) and were neurological in 15% (8/53). Four patients experienced sudden death. Each of the four valves was affected with approximately equal frequency: 16 aortic, 15 mitral, 13 pulmonary, and 12 tricuspid. All but four tumors were benign. The most common histological type was papillary fibroelastoma (41), followed by myxomas (5), fibromas (4), sarcomas (2), hamartoma (1), hemangioma (1), histiocytoma (1), and undifferentiated (1). Average tumor size was 1.15 cm (range, 3 mm to 7 cm), and the average size of fibroelastomas was 8 mm (range, 3 to 15 mm). Mitral valve tumors were more likely than aortic valve tumors to produce serious neurological symptoms or sudden death (8/15 versus 3/16; $p < 0.05$). Six patients underwent echocardiography, and results were positive in each. All 6 underwent uncomplicated valve repair or replacement. Compared with a series of 407 nonvalvular tumors, cardiac valve tumors are more likely to occur in male patients ($p < 0.001$) and adults ($p < 0.001$). Valve tumors are also more commonly benign ($p < 0.001$) and asymptomatic ($p < 0.001$). These tumors demonstrate somewhat less aggressive behavior compared with nonvalvular tumors, but their distinct propensity to produce serious clinical sequelae argues in favor of surgical resection for all cardiac valve tumors.

(Ann Thorac Surg 1991;52:1127–31)

Primary cardiac valve tumors are quite rare but can have important clinical implications. In most series of cardiac tumors, primary valve tumors comprise less than 10% of cases [1–6]. The majority of information regarding these tumors has accumulated from isolated case reports and from general reports of cardiac tumors. Consequently, only scattered reports, each consisting of a small number of valve tumors, are presently available. To specifically investigate the clinical characteristics of this entity, we reviewed our experience with 56 primary cardiac valve tumors from 1932 through 1990.

Material and Methods

The records from Walter Reed Army Medical Center, Brooke Army Medical Center, and the Armed Forces Institute of Pathology were reviewed for tumors primarily involving the cardiac valves. We excluded those tumors that were metastatic or infectious. Tumors extending into extravalvular structures were included only if the major part of the tumor clearly involved a cardiac valve.

Information was obtained from patient records, autopsy reports, and direct histological examination of the excised tumors. Adequate records were available from 1932 through 1990.

Statistical calculations were made using a standard software program (BMDP Statistical Software, Los Angeles, CA). Univariate comparison of selected parameters was tested by calculating a χ^2 value with 2 × 2 contingency tables and one degree of freedom except where otherwise stated. A p value of less than 0.05 was considered significant.

Results

Fifty-three patients were found to have 56 primary cardiac valve tumors. Forty-two (79%) were male, and the age ranged from 2 years to 88 years, with an average of 52 years of age (Fig 1). Only 1 patient was less than 21 years of age.

Presentation

Most patients were asymptomatic. Twenty patients (37.7%) had cardiopulmonary or neurological symptoms and 4 had sudden death. Among these symptomatic patients, there were significantly more left-sided tumors than right-sided tumors ($p < 0.05$). The mitral valve was most commonly associated with symptoms (10), followed by the aortic valve (7), tricuspid valve (5), and pulmonary valve (4) (p = not significant). Excluding the 4 patients with sudden death, most symptomatic patients (14/20) had complaints consistent with moderate congestive heart failure, whereas 40% (8/20) had neurological symptoms (Table 1).

The majority of patients with heart failure had small tumors, although at least 3 had tumors of sufficient size to

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The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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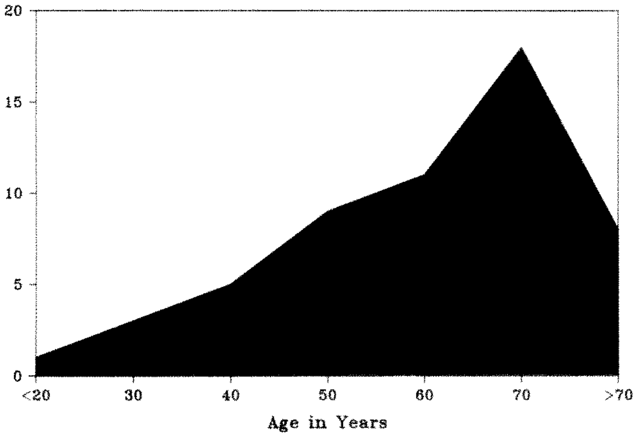

Fig 1. Age distribution of patients with cardiac valve tumors.

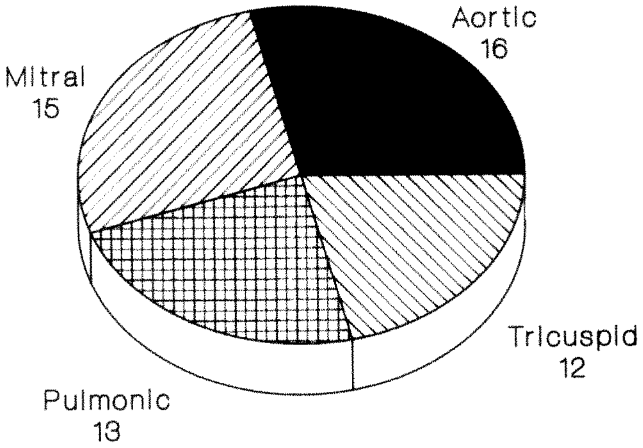

Fig 2. Location of valve tumors. Each of the four valves was involved with approximately equal frequency.

seriously obstruct flow across the valve (see Table 1). Each of these three tumors was malignant, and each was addressed with operative resection. The remaining tumors associated with heart failure were incidentally discovered at autopsy, thereby making it impossible to determine whether these tumors played a role in the development of symptoms. In each of these cases, no other cardiac abnormalities were noted.

Table 1. Clinical Presentation[a]

Presentation	Valve Involved	Tumor Type
Congestive heart failure	Pulmonic	PF
	Pulmonic	Sarcoma
	Pulmonic	Myxoma
	Tricuspid	PF
	Tricuspid	Undifferentiated
	Tricuspid	PF
	Tricuspid	PF
	Tricuspid	PF
	Mitral	MFH
	Mitral	Hamartoma
	Aortic	PF
	Aortic	PF
	Aortic	PF
	Aortic	PF
Neurological	Mitral	Myxoma
	Mitral	Myxoma
	Mitral	Myxoma
	Mitral	Undifferentiated
	Mitral	Sarcoma
	Mitral	MFH
	Aortic	PF
	Pulmonic	PF
Sudden death	Mitral	Hemangioma
	Mitral	PF
	Aortic	Fibroma
	Aortic	PF

[a] There were 20 patients having a total of 22 symptoms.

MFH = malignant fibrous histiocytoma; PF = papillary fibroelastoma.

The 8 patients with neurological symptoms (see Table 1) made up 15% of this series (8/53) and 40% (8/20) of those who were symptomatic. Six patients had mitral valve tumors, half of which were myxomas and half of which were malignancies. All neurological symptoms were unequivocal, but only 1 patient suffered a stroke and only 1 had a transient ischemic attack. None of the neurological sequelae were fatal.

Four patients had sudden death. A left-sided valvular tumor was present in each instance. Two had acute myocardial infarctions from papillary fibroelastomas of the aortic valve, which had embolized to the left anterior descending coronary artery. The other 2 had mitral valve tumors. One of these was a large hemangioma and the other was a fibroelastoma of unspecified size. In each of the mitral valve cases, no direct autopsy evidence was found to link the cardiac tumors with the cause of death.

Findings on physical examination included clinical signs consistent with congestive heart failure in 14 patients and neurological findings in 3 (dysarthria, right-sided extremity weakness, and confusion). Only 2 patients had firmly documented new systolic murmurs.

Tumor Characteristics

The size of tumors ranged from 3 mm to 7 cm with an average of 1.15 cm in largest dimension.

As shown in Figure 2, the aortic valve was most commonly involved. A χ^2 analysis using a 2×4 contingency table, however, revealed that there was no significant difference in the frequency of involvement for each of the valves.

Fifty-two of the 56 tumors (92.8%) were benign. There were 41 papillary fibroelastomas, 5 myxomas, 4 fibromas, 1 hamartoma, and 1 hemangioma (Fig 3). All of the aortic valve tumors were benign, as were 13 of 15 mitral valve tumors and all but one of the pulmonic and tricuspid valve tumors.

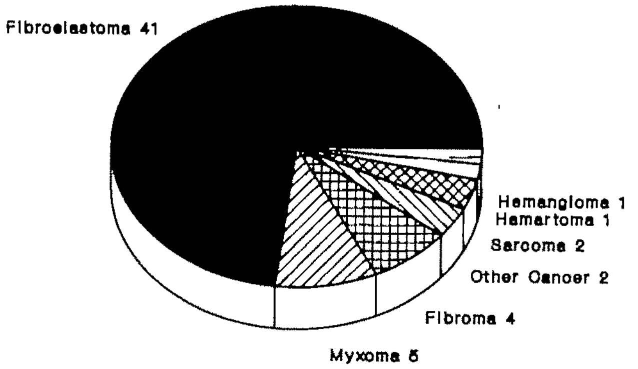

Fig 3. Histological distribution.

The remaining four tumors were malignant. There was 1 malignant fibrous histiocytoma of the anterior mitral valve leaflet, 1 undifferentiated carcinoma of the tricuspid valve, and 2 sarcomas, 1 involving the pulmonic valve and the other involving the mitral valve. All malignant valve tumors were associated with preoperative symptoms, three of which were neurogenic. None of the malignancies were seen with extracardiac metastatic disease and none produced sudden death.

Three of the 4 patients with malignant tumors were female, and there was a statistically significant difference in the frequency of cancerous valve tumors among female patients (27%) as compared with male patients (2%) ($p < 0.01$). Because of the small number of malignant tumors, it was not possible to draw conclusions regarding the possibility of age as a risk factor for malignancy. The oldest (88 years) as well as the youngest patient (2 years) in this series had malignant valve tumors. The other 2 patients with cancerous valve tumors were 31 and 63 years of age.

Five patients had multiple cardiac tumors. Three of these involved two different valves and all were papillary fibroelastomas (pulmonic and aortic, tricuspid and mitral, tricuspid and pulmonic). The other 2 patients had multiple tumors involving extravalvular structures (aortic valve malignant fibrous histiocytoma and left atrial appendage malignant fibrous histiocytoma, tricuspid valve undifferentiated carcinoma and right ventricular free wall fibroma).

Diagnostic Studies

Valve tumors were discovered at autopsy in 45 of the 53 patients, so few diagnostic studies were performed in this series. In the 22 symptomatic patients, all studies were normal except in 8 patients. In these patients, echocardiography was performed in 6 and revealed a mitral valve tumor in each instance. In another, chest radiography demonstrated a lung abscess. Documentation on 1 other patient indicates only that preoperative evaluation revealed a tricuspid valve tumor.

Treatment

One patient with a mitral valve sarcoma had multiple cerebral infarcts and did not undergo operative intervention. All others with a preoperative diagnosis of valvular tumors were surgically explored. Our general approach was to remove the tumor while preserving the native valve if at all possible. Four patients with mitral valve papillary fibroelastomas were treated with excision of the tumor and valve repair. One patient with a mitral malignant fibrous histiocytoma had a mitral valve replacement and another patient with an undifferentiated carcinoma had a tricuspid valve replacement. Both patients in whom valve replacement was necessary had large malignant tumors, which precluded valve repair. An additional patient had an open lung biopsy and open drainage of a lung abscess which resulted in his death. At autopsy the lung abscess was found to be due to a pulmonary infarct that had been created by tumor embolization from a pulmonic valve sarcoma. All other patients undergoing operation survived the procedure. Long-term follow-up of these patients is not available.

Comment

Because of the rarity of cardiac valve tumors, there is an incomplete understanding of their clinical characteristics. Today it is particularly important to have an appreciation for the behavior and surgical implications of valve tumors because these lesions are being recognized more frequently [5–7]. Most valve tumors will be incidental findings at autopsy, but recently there has been an unequivocal increase in the premortem diagnosis of cardiac tumors, including valve tumors [5–7]. This has been most directly attributable to the technologic advancements and more frequent use of echocardiography [5, 7, 8]. It appears that a preoperative noninvasive diagnosis of even small valve tumors can be made with good confidence [9], and this will almost certainly result in an increase in the number of patients being referred for evaluation of these echocardiographic findings. To gain a better understanding of the clinical and pathological nature of these tumors we have reviewed our multiinstitutional experience over the last 58 years.

Papillary Fibroelastoma

The great majority of valve tumors are papillary fibroelastomas, so it is appropriate to describe this tumor in some detail. These benign lesions are characteristically small avascular tumors derived from normal components of the endocardium, fibrous tissue, elastic fibers, and smooth muscle cells. The majority are valvular; in the Armed Forces Institute of Pathology series, only 13% (6/45) were nonvalvular [2]. They most frequently arise from the valvular endocardium and grossly resemble a sea anemone with multiple papillary fronds (Figs 4, 5). When the atrioventricular valve is involved, the tumor is most often on the atrial side of the valve, but when semilunar valve is involved, there is no predilection for one side or the other. Along the valve leaflet, the most common site of occurrence is in the midportion, well away from the free edge or the annulus.

Histologically, one finds a central core of dense connective tissue that appears to be a direct extension of the

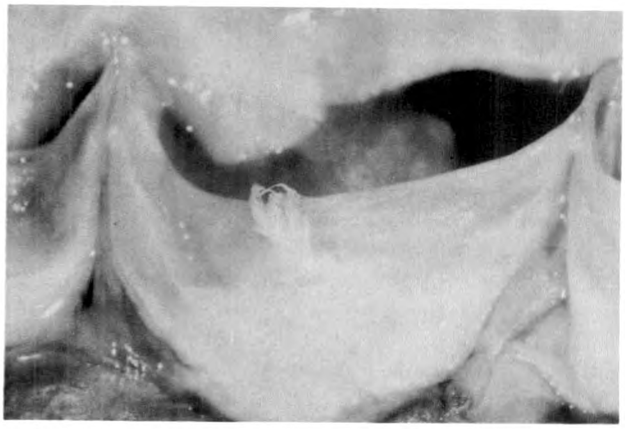

Fig 4. Gross appearance of papillary fibroelastoma. This tumor arose from the ventricular surface of the aortic valve.

endocardium. Surrounding this central core is a layer of hyperplastic endocardial cells, loose connective tissue, and a mesh of elastic fibers (Fig 6).

Early reports on the clinical behavior of these tumors indicated that they were not responsible for cardiac symptoms [2, 3, 10, 11], but our findings as well as those of others has shown this not to be invariably true. In fact, the association between aortic valve fibroelastomas and serious coronary or cerebral emboli has been clearly established [2, 3, 4, 8, 10, 12–16].

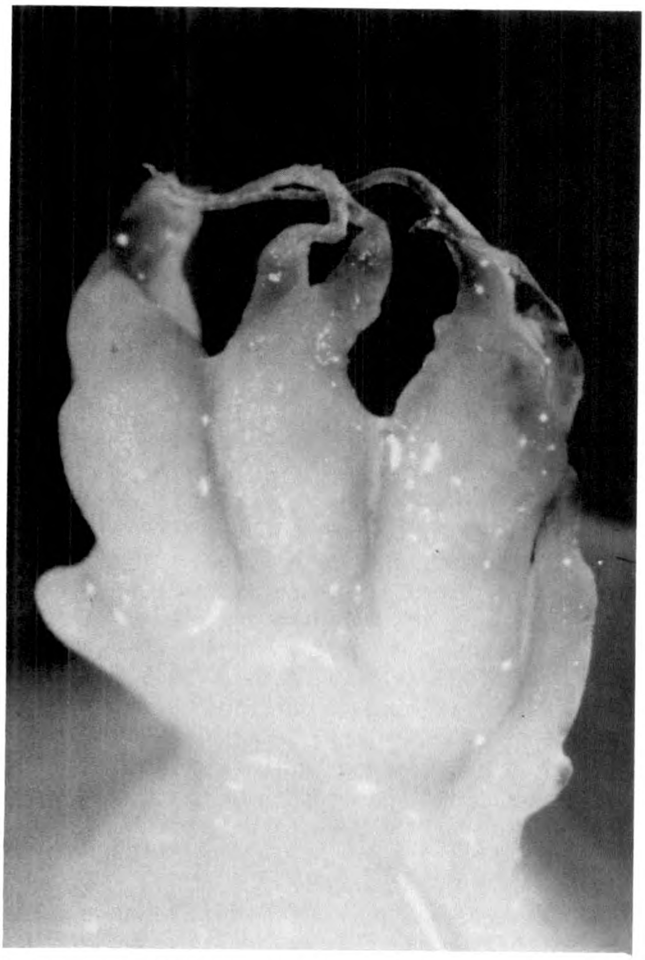

Fig 5. A more detailed view of the tumor shown in Figure 4. The fronds are particularly friable at the most distal aspect and may produce serious coronary or cerebral embolic events.

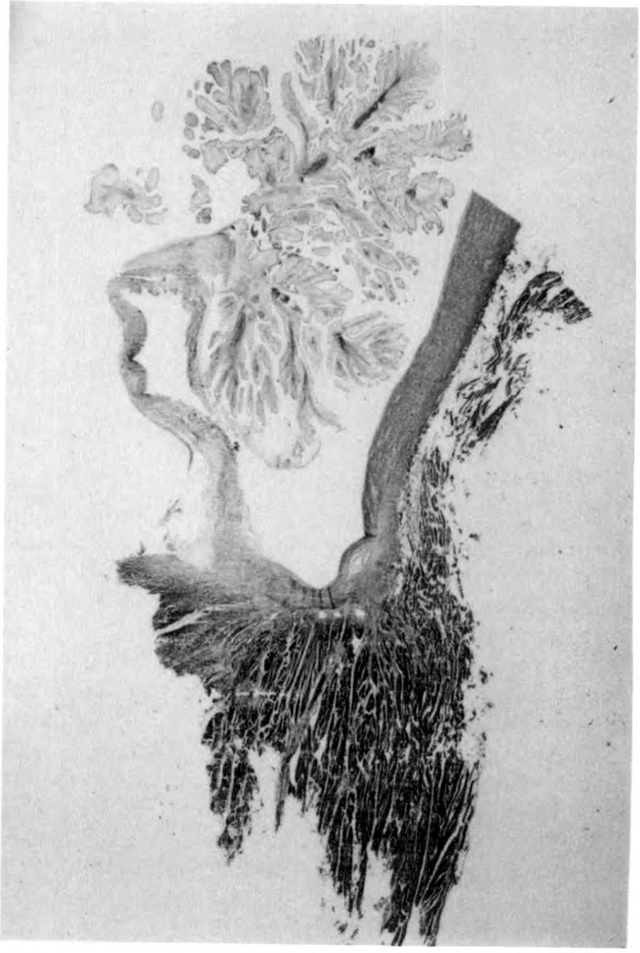

Fig 6. Microscopic appearance of papillary fibroelastoma. The tumor consists of multiple papillary fronds arranged on a stalk which blends into the substance of the valve. (Hematoxylin and eosin, ×10 before 3% enlargement.)

Comparison With Nonvalvular Cardiac Tumors

To see how these tumors differed from the more common extravalvular cardiac tumors, we compared our series against the 407 patients having nonvalvular solid cardiac tumors reported in the Armed Forces Institute of Pathology series [2].

The most common nonvalvular tumor was, of course, the myxoma, and the most common valvular tumor in our series was the papillary fibroelastoma. Other major differences are outlined in Table 2. In general, valve tumors are significantly more likely to occur in adults and in male patients. In addition, they are more likely to be benign, asymptomatic, and discovered incidentally at autopsy. There was no significant difference in the frequency of multiple tumors or the occurrence of sudden death.

Table 2. Comparison of Characteristics

Variable	Valvular	Nonvalvular[a]	p Value
Male	79.2% (42/53)	57.1% (76/133)[b]	<0.001
Pediatric	1.8% (1/53)	21.1% (86/407)	<0.001
Malignancy	7.6% (4/53)	30.7% (125/407)	<0.001
Asymptomatic	47.2% (25/53)	14.3% (58/407)	<0.001
Sudden death	7.6% (4/53)	9.6% (39/407)	NS
Multiple	9.4% (5/53)	13.5% (55/407)	NS

[a] From the Armed Forces Institute of Pathology series [2] of 407 patients with solid nonvalvular cardiac tumors. [b] Where specifically stated (76 males/133 cases).

NS = not significant ($p > 0.05$).

Clinical Implications

Primary cardiac valve tumors are less likely to produce serious sequelae as compared with nonvalvular tumors. Nevertheless, these tumors have been associated with sudden death, embolic events, and heart failure in a number of well-documented cases [1, 10, 12–16]. Perhaps because of its typically small size, the papillary fibroelastoma has been considered an innocuous lesion in the past [2, 3, 10, 11], but these tumors have a significant propensity to embolize [9, 10, 12–16]. The embolic material may arise from either fragments of the tumor or thrombus that not uncommonly forms around the tumor [9, 12].

The potential for life-threatening complications indicates that cardiac valve tumors should be managed surgically. This is particularly advisable given the ability to repair rather than replace many affected valves. Once the diagnosis of a valvular tumor is made, it appears reasonable to begin systemic anticoagulation and carry out a prompt surgical resection of the tumor.

Limitations of the Study

More than 70% of these tumors are papillary fibroelastomas, so that generalizations regarding valvular tumors are necessarily skewed to reflect the characteristics of fibroelastomas. Also, most of the tumors in this series were discovered at autopsy, so treatment recommendations are drawn from the management of a relatively small number of patients. In addition, the military population of this series may not represent a typical cross-section of the general population.

Conclusions

Cardiac valve tumors constitute a distinct clinical entity with important differences from extravalvular cardiac tumors. The valves are affected with approximately equal frequency, and compared with extravalvular tumors, they are more likely to be benign and asymptomatic. We have found that valve tumors are quite uncommon in the pediatric population and that male patients appear to have a higher propensity for the development of these tumors than do female patients. The most common of the valve tumors is the papillary fibroelastoma, which is characteristically a small tumor having a small but definite propensity to produce embolic sequelae.

The clinical behavior of these tumors is somewhat less aggressive as compared with the more common extravalvular tumors. Nevertheless, there is considerable evidence that valvular tumors can cause life-threatening complications. Because of this, we believe that the diagnosis of a cardiac valvular tumor mandates prompt surgical resection of the tumor with preservation of the native valve.

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Very Small Pulmonary Arteries: Central End-to-Side Shunt

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Between 1980 and 1989, 28 patients with pulmonary atresia, ventricular septal defect, and very small pulmonary arteries with major aortopulmonary collateral arteries underwent direct central end-to-side shunts as staging procedures. Age range was 2 months to 32 years, with 19 patients less than 1 year of age. Pulmonary artery diameters ranged from 1 to 4 mm, with 24 less than 3 mm. Two patients (7%; 70% confidence limits, 2% to 16%) died after the shunt, and there were two further deaths after subsequent staging or correction. Acute shunt complications included congestive cardiac failure (mild to moderate, n = 8; severe, n = 3) and endocarditis (n = 1). Proximal right pulmonary artery stenoses have occurred in 75% of patients and left pulmonary artery stenoses, in 50%. Satisfactory pulmonary artery growth was achieved, however, in 16 of 24 hospital survivors investigated postoperatively. Twelve patients have proceeded through unifocalization to biventricular repair (in 2 the ventricular septal defect patch was subsequently fenestrated) with one death (8.3%; 70% confidence limits, 1% to 25%). Eight patients are still in staging, and 4 have been excluded from the program because of inadequate unifocalization. The direct central end-to-side shunt has proven satisfactory in attaining pulmonary artery growth in patients with very small central pulmonary arteries.

(Ann Thorac Surg 1991;52:1132–7)

Pulmonary atresia plus ventricular septal defect (VSD) with very small pulmonary arteries (PAs), arborization defects, and major aortopulmonary collateral arteries (MAPCAs) is a complex congenital malformation that is not amenable to a primary corrective procedure. Where a diminutive PA system is present, staging procedures have been employed in an attempt to attain PA growth. These procedures include various types of shunts as well as right ventricular outflow tract reconstruction [1–9]. At the Royal Children's Hospital, a direct central end-to-side shunt has been developed as a staging procedure for a subgroup of patients with very small PAs. Herein, the sequelae of this shunt procedure are reviewed.

Patients and Methods

Patients

Between 1980 and 1989, 28 patients with pulmonary atresia plus VSD, MAPCAs, and very small PAs (usually with arborization defects) underwent a central end-to-side shunt as a staging procedure. Age range was 2 months to 32 years, with 19 patients less than 1 year of age (Table 1). The central shunt was the initial staging procedure in 27 of 28 patients, 1 patient having previously undergone a right modified Blalock shunt.

Twenty-six of 28 patients had a PA system consisting of a short main PA with right pulmonary artery (RPA) and left pulmonary artery (LPA) in continuity. Two patients had no LPA, so the shunt was between the ascending aorta and RPA. Pulmonary artery diameter was estimated at preoperative angiography and also at operation (Table 2).

Technique

A median sternotomy was used in 23 of 28 patients, and a left thoracotomy in the remaining 5. A left thoracotomy was used if it was thought that both the central shunt and left sided unifocalization (MAPCA transplantation or ligation or both) could be performed simultaneously. Cardiopulmonary bypass support was required in 2 very hypoxic patients.

The ascending aorta and the diminutive main and branch PAs were mobilized, and Silastic (Ethicon, Somerville, NJ) snares were placed around left and right PAs. Heparin (1 mg/kg) was given intravenously, and the left side of the ascending aorta was side-clamped as posteriorly as possible. A button of aortic wall was excised and the mobilized main pulmonary artery ("fishmouthed" on the right side) was anastomosed end-to-side to the aorta using continuous 7-0 polypropylene. The clamp was released and the heparin was not reversed (Fig 1).

Results

Mortality

Two patients died after the central shunt (7%; 70% confidence limits [CL], 2% to 16%). There were two further deaths associated with subsequent unifocalization or correction. The first death occurred in a patient with an absent LPA who also had a somewhat hypoplastic left ventricle. Sudden death occurred on the third postoperative day, presumably owing to an arrhythmia. The second

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Table 1. Age Distribution of Patients With Central End-to-Side Shunt

Age (y)	No. of Patients
0–6 mo	8
6–12 mo	11
1–2 y	0
2–3 y	1
3–4 y	1
6–12 y	5
30–35 y	2
Total	28

patient, 3 months old, had severe cyanosis. An emergency peripheral shunt was abandoned because the RPA was too small, and a central shunt using a diminutive main pulmonary artery was performed. A low output, hypoxic, and acidotic state persisted and the child died 48 hours postoperatively. The small PAs, and probably inadequate MAPCAs, constituted an anatomical basis for the failure of the procedure.

There were two further deaths after subsequent procedures. The first late death was due to a mesenteric embolus after a fourth preparatory operation (open mitral valvotomy for mitral stenosis). The second late death occurred in a 4-month-old patient with episodic severe hypoxia, who had major hemodynamic instability after a central shunt. He had required emergency reexploration in the early postoperative period, at which time the shunt diameter was reduced with vascular clips. This had the effect of severely narrowing the LPA and led to a sequence of attempted repair and then fenestration of the VSD patch because of systemic right ventricular pressures. This patient also had severe tracheobronchomalacia, which eventually led to management being withdrawn 3 months postoperatively.

Shunt Complications

Early congestive cardiac failure related to excessive pulmonary blood flow developed in 11 patients. Failure was mild to moderate with no serious hemodynamic instability in 8 patients and responded to antifailure therapy within 48 hours. These patients were all discharged for further investigation and staging. Severe failure developed in 3 patients, who remained ventilator dependent postoperatively. Their staging was accelerated, and all underwent unifocalization in the 2 weeks after shunting and correction within a month without operative mortality, but with one late death 3 months after repair, related to severe bronchomalacia (Figs 2–4).

Table 2. Pulmonary Artery Diameter Distribution

PA Diameter (mm)	No. of Patients
1–1.9	7
2–2.9	15
3–3.9	4
Absent left PA, right PA 2–3 mm	2
Total	28

PA = pulmonary artery.

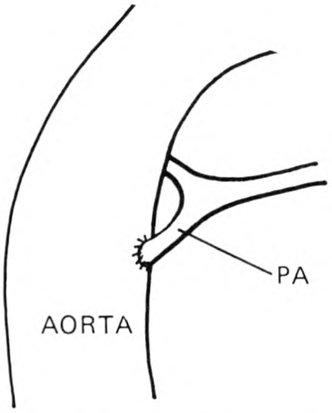

Fig 1. Central end-to-side shunt. The main pulmonary artery is anastomosed to the ascending aorta as posteriorly as possible.

Infection

A false aneurysm at the anastomosis, related to infective endocarditis, developed in 1 patient. Intervention was required 7 months after the initial shunt. The RPA had grown to 8 mm in diameter and the LPA to 6 mm in diameter from initial diameters of 2.5 mm. Therefore the false aneurysm, which had destroyed 1.5 cm^2 of the left wall of the ascending aorta, was completely excised. The aorta was oversewn, the branch PAs were reconstructed,

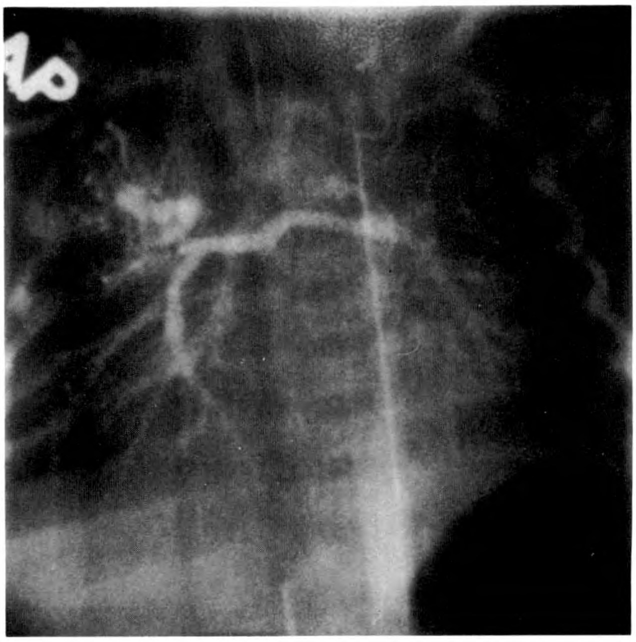

Fig 2. Pulmonary artery wedge injection outlining the pulmonary arteries before central shunt in a 2-month-old child.

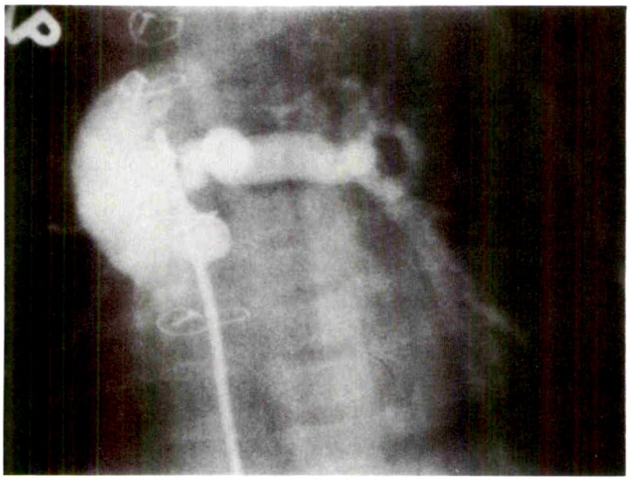

Fig 3. Angiogram of same patient as in Figure 2 three weeks after the central shunt demonstrating substantial pulmonary artery growth (2–3 mm to 7.5 mm).

and total correction was undertaken using an 18-mm valved homograft conduit. Six months after correction the child is asymptomatic and free of apparent infection.

Further Staging/Correction

Twelve patients have progressed to full correction (with the VSD patch subsequently requiring fenestration in 2) with one operative death (8.3%; CL, 1% to 25%) (Table 3). Eight patients are still in staging. Future repair is anticipated in 4 patients who meet criteria for correction (projected ratio of right ventricular to left ventricular pressure <0.7; no remaining sizeable MAPCAs; > ⅔ of lung segments connected to the native PAs, and a pulmonary to systemic flow ratio > 1) [10]. Three other patients have had problems identified with regard to these criteria at postshunt catheterization. Suitability for final correction has therefore not yet been attained. One patient is still in staging and has not yet had postshunt investigations.

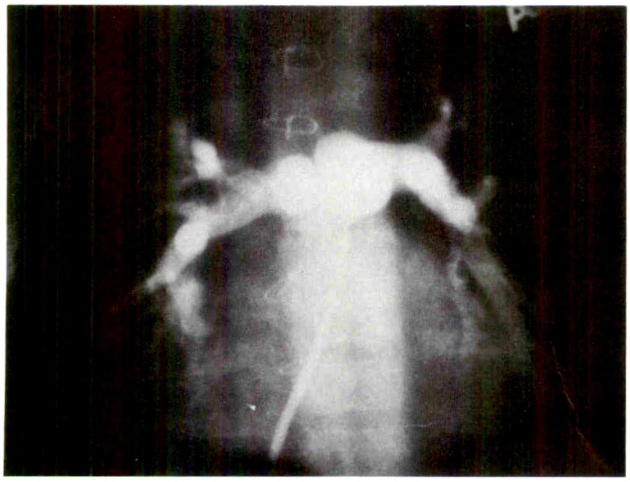

Fig 4. Pulmonary artery injection in same patient as in Figures 2 and 3 after total correction.

Table 3. Procedures After Central End-to-Side Shunt in 26 Survivors

Patient No.	Procedure No.	Procedures
1	2	L thoracotomy ML1
	3	Correction
2	2	R thoracotomy MT1, ML1
	3	L thoracotomy MT1, ML1
	4	Correction
3	2	Correction
4	2	R thoracotomy MT1, ML1
	3	L thoracotomy MT1, ML1
	4	Correction
5	2	R thoracotomy MT1, ML1
	3	L thoracotomy MT1, ML1
	4	Correction
6	1	L thoracotomy central shunt, MT2
	2	R thoracotomy MT1
	3	Correction
7	2	L thoracotomy ML2
	3	R thoracotomy MT1
	4	Correction
8	2	R thoracotomy MT1, ML2
	3	Correction
9	2	R thoracotomy MT2
	3	L thoracotomy MT1, ML1
	4	Correction
10	1	L thoracotomy central shunt, ML1
	2	R thoracotomy MT1, ML1
	3	Correction
11	1	L thoracotomy central shunt, ML1
	2	R thoracotomy MT1, ML2
	3	Correction
12	1	L thoracotomy central shunt, ML1
	2	R thoracotomy MT2
	3	Correction
13	2	2nd sternotomy aorta–RPA (direct)
	3	R thoracotomy MBTS
	4	3rd sternotomy central PA patch reconstruction, central Gore-Tex shunt
14	2	L thoracotomy MT1
	3	R thoracotomy ML1
15	. . .	No further procedure
16	2	2nd sternotomy aorta–RPA (direct)
	3	R thoracotomy MAPCA–aorta
17	2	L thoracotomy MT1, ML1
19	2	R thoracotomy MT1
20	1	L thoracotomy central shunt, ML1, for further staging
21	. . .	For further staging
22	. . .	For further staging
23	. . .	For further staging
24	. . .	For further staging

Table 3. Continued

Patient No.	Procedure No.	Procedures
25	2	R thoracotomy MT2
	3	L thoracotomy MT1, ML2
	4	VSD fenestration after repair
26	. . .	No further procedure
28	2	R thoracotomy MT1
	3	L thoracotomy MT1
	4	2nd sternotomy mitral valvotomy, atrial septectomy

L = left; MAPCA = major aortopulmonary collateral artery; MBTS = modified Blalock-Taussig shunt; ML = MAPCA ligation; MT = MAPCA transplantation; PA = pulmonary artery; R = right; RPA = right pulmonary artery; VSD = ventricular septal defect.

Four patients have been excluded from the program for the present. One patient had an absent LPA and a central end-to-side shunt to the RPA. Postoperative catheterization showed poor RPA growth. Another patient originally underwent a central end-to-side shunt to a very small (1 to 1.5 mm) PA system. Recatheterization showed a blocked RPA, and at reoperation a direct end-to-side RPA to ascending aorta anastomosis was performed. Further catheterization studies showed that both PAs were still very small. The third patient underwent further staging with a right thoracotomy and MAPCA transplantation using an azygos vein conduit [11]. Recatheterization showed severe long-segment LPA hypoplasia and systemic pressures in the RPA to the point of the MAPCA transplant. Pulmonary vascular disease is probably present in the right lung. The fourth patient, 31 years of age with progressive cyanosis at the time of the central shunt, was much improved by the operation and does not wish to have a further operation.

Pulmonary Artery Growth

The PAs have been assessed using cardiac catheterization in 25 (12 corrected, 13 uncorrected) of the 25 survivors of initial hospitalization (Table 4). Proximal RPA stenosis has been identified in 18 patients (72%; CL, 60% to 82%) and proximal LPA stenosis has been seen in 12 patients (48%; CL, 36% to 60%). Overall satisfactory PA growth, as

Table 4. Pulmonary Artery Growth After Central Shunt[a]

	RPA (mm)		LPA (mm)			
Age at Central Shunt	Pre Shunt	Post Shunt	Pre Shunt	Post Shunt	Time (mo)	Progress
3 y 6 mo	2	Occl	2	3.5	10	Staging
8 mo	2	8	2	8	6	Staging
8 mo	1	Occl	1	3	13	Staging
9 mo	2.5	. . .	3	. . .	No cath	Staging
11 y	3	6	3	6	16	Staging
10 mo	2	3	2	3	9	Staging
2 mo	2.5	4	2	5–6	22	Staging
9 mo	2.5	5.5	2.5	8.5	9	Staging
12 mo	2	7	2	3	12	Not for correction
31 y	3.5	8	3.5	8	36	Not for correction
10 y	2.5	2.5	Absent	. . .	13	Not for correction
2 mo	1	1	1	4	11	Not for correction
3 y 6 mo	1.5	5.5	1.5	7	7	Late death
6 y 10 mo	2	8	2	8	26	Correction
3 y 8 mo	3	15	3	10	29	Crorection
11 mo	2	10	2.5	10	21	Correction
9 mo	2	5.5	3	5.5	11	Correction
7 y 4 mo	3–4	14	3–4	14	29	Correction
34 y	3–4	10.5	5	10	10	Correction
4 mo	2	7	2.5	7.5	21	Correction
2 mo	2.5	7	2.5	7.5	1	Correction
2 y 11 mo	2.5	8	2.5	6	7	Correction
7 mo	2	5	3	8	1	Correction
12 mo	3	8.5	3	8.5	10	Correction
8 y	2	3	2	6	24	Correction/VSD patch fenestrated

[a] Pulmonary artery diameters measured between mid-pulmonary artery and hilum.

Cath = catheterization; LPA = left pulmonary artery; Occl = occluded; RPA = right pulmonary artery; VSD = ventricular septal defect.

Table 5. Pulmonary Artery Pressures After Central Shunt

Patient No.	PAP (mm Hg)	Progress	Interval (mo)	PA Size (mm)	
				RPA	LPA
1	55/32	Correction	26	8	8
2	59/36	Correction	29	15	10
4	LPA 12/8	Correction	11	5.5	5.5
5	60/48	Correction	29	14	14
6	40	Correction	11	10	10
7	28/12	Correction	21	7.5	7.5
11	16/6	Correction	1	5	8
12	47/28	Correction	10	8.5	8.5
14	RPA 85/45	PVD	12	7	3
16	35/28	Staging	26	8	8
24	32/18	Staging	7	8	6
28	45/24	Late death	7	5.5	7

LPA = left pulmonary artery; PA = pulmonary artery; PAP = pulmonary artery pressure; PVD = pulmonary vascular disease; RPA = right pulmonary artery.

assesssed by PA diameter measured between the mid-PA and hilum, was achieved in 72% (CL, 60% to 82%).

Pulmonary Hemodynamics

Selective cannulation of the central shunt has proved difficult at postoperative catheterization as the catheter tends to rebound from the central shunt into the aorta. This has meant that PA pressure measurements to date are available in 12 patients only (Table 5). One patient had systemic pressures in the main pulmonary artery and RPA and probably has pulmonary vascular disease in his right lung and is not suitable for correction. Systolic PA pressure has ranged from 12 to 60 mm Hg in the other 11 patients. Four patients had a systolic PA pressure between 45 and 60 mm Hg and all have successfully undergone correction.

Comment

Patients with pulmonary atresia, VSD, very small PAs, MAPCAs, and arborization abnormalities require central PA growth as well as unifocalization of pulmonary blood flow before a corrective procedure can be undertaken. Two different approaches have been used in an effort to attain PA growth. In the published literature the most common approach has been the use of right ventricular outflow tract reconstruction [1–7]. In the Mayo Clinic series (actual PA sizes not specified) approximately 50% of patients treated with right ventricular outflow tract reconstruction have gone on to complete repair [2, 3, 12]. The remaining patients were not suitable for complete repair because of inadequate PA enlargement or insufficient arborization of the central PAs. Other studies have also reported only moderate success in attaining PA growth using this technique [1–4]. As well, serious PA stenosis and right ventricular outflow tract aneurysms from patch enlargement have been reported [1, 4]. The results of right ventricular outflow tract patching, particularly in patients with very small PAs, were similar also in our own unit, leading to a change in approach. We currently employ the direct end-to-side central shunting technique for all patients with very small PAs in this setting. We prefer this to a peripheral shunting technique because we believe that the chances of attaining symmetric growth without iatrogenic stenosis from the shunt are higher.

The problem of excessive pulmonary blood flow from too large a shunt needs to be addressed when selecting patients for a direct central shunt, and also while monitoring their postoperative care. This is especially true if the MAPCA supply is well developed. Forty percent of our patients had signs of congestive failure, which, although mild to moderate in the majority, did require antifailure treatment.

The other common problem in the medium term is proximal PA stenosis, which occurs frequently as an associated congenital lesion in pulmonary atresia [13]. Where proximal PA growth has been suboptimal owing to such stenoses, correction is not precluded as long as the PA size at the hilum is satisfactory. However, extensive PA reconstruction has usually been required, often out into the hilum and occasionally into the lower lobe branch, to obtain satisfactory PA dimensions. Eleven of 13 patients coming to corrective operation have required PA reconstruction. Proximal PA stenoses have affected the RPA more than the LPA. Where there has been moderate growth the RPA can be reconstructed, but in 3 patients the RPA was virtually occluded. This has proved to be a major problem because reoperation for direct RPA–aorta anastomosis has uniformly failed to achieve RPA growth. We have consequently abandoned this approach in favor of direct reconstruction of the PA bifurcation. It is likely that some RPA stenoses can be avoided by placing the anastomosis as posteriorly as possible to avoid tension or kinking of the RPA. In other patients, even in those with very small PAs, bifurcation stenosis has been identified at initial angiography.

A potential problem from direct central shunting is pulmonary vascular disease due to systemic pressure in the pulmonary vessels. This has occurred in only 1 patient, precluding correction. In the other patients in whom pressures have been measured, the peak systolic PA pressure has always been subsystemic.

The majority of central shunts have been performed in patients less than 12 months of age. This philosophy of early entrance into a staged surgical program evolved because of the disappointing experience with unifocalization in some of the older patients with thick sclerotic MAPCAs and hypertensive lung segments. Analysis of the overall experience of 58 patients from this unit revealed a 78% incidence of suitability for correction in patients less than 5 years of age compared with 50% for the older patients ($p < 0.05$) [14].

Our current indications for use of the central end-to-side shunt in pulmonary atresia plus VSD with MAPCAs is the presence of very small confluent PAs. In patients less than 1 year of age, this means RPA and LPA ranging from 1 to 3 mm in diameter. If the PAs are not visualized on echocardiographic and catheterization studies (an unusual occurrence), exploration may still be carried out through a sternotomy. We prefer to establish this shunt before unifocalization of MAPCAs. The shunt should ideally be performed in the first year of life, even in asymptomatic patients. Timing of further staging and biventricular repair may vary considerably according to individual anatomical features. Subsequent staged bilateral thoracotomies are used for unifocalization of MAPCAs. This is complemented by catheter occlusion (coils) of MAPCAs with major connections to the native PA system, usually discovered after complete repair.

In conclusion, our overall experience with the direct central shunt in this difficult group of patients highlights certain points.

1. This shunt is suitable only for patients with very small PAs. These patients need careful supervision in the early postoperative period because of their prospensity to cardiac failure. In the majority, cardiac failure can be successfully treated with an antifailure regimen. If it cannot, early catheterization is warranted because the PAs will most likely have grown and the patient can be placed on an accelerated staging program with a view to early correction.
2. The shunt must be placed as posteriorly as possible on the aorta to reduce RPA distortion.
3. Although proximal PA stenoses have been common after a direct central shunt, correction is possible as long as the PA diameter at hilar level and beyond is appropriate for the size of the child.
4. Overall, we believe that early intervention for staging in these patients will allow better results from shunting and unifocalization procedures and will maximize the number of patients who will be suitable for biventricular repair. Ideally, surgical staging should commence in the first year of life.

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Transcutaneous Iodine Absorption in Infants Undergoing Cardiac Operation

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Povidone-iodine is an effective antiseptic, but its topical use has been associated with a number of adverse reactions in burn patients and in neonates as a result of transcutaneous absorption. In particular, high plasma iodine concentrations are known to cause renal failure, metabolic acidosis, and thyroid suppression. Because of the permeable nature of the skin in small infants and the large areas cleaned before cardiac operations, it is possible that significant transcutaneous iodine absorption might occur in this situation. We have studied 17 infants, less than 3 months of age, who were undergoing closed cardiac or thoracic procedures. After povidone-iodine skin preparation in 15 (covering 20% to 30% of body surface area), plasma total iodine concentrations rose fourfold (range, 160% to 1,440%). This increase was significantly different from the preoperative level at 6, 12, 18, and 24 hours. There was no increase in plasma iodine concentration in 2 patients who were not exposed to povidone-iodine or any other iodine-containing compound. We discuss the implications for a topical antisepsis policy in infants.

(Ann Thorac Surg 1991;52:1138–40)

Povidone-Iodine is an extremely effective antiseptic agent, active against bacteria, viruses, and fungi. Topical application in burn patients has, however, been associated with significant transcutaneous absorption and a number of adverse effects including renal failure, metabolic acidosis, hypernatremia, hyperosmolarity, and hypothyroidism [1–5].

In neonates, the skin is very thin and permeable [6], and although it is intact, significant transcutaneous absorption has been reported [7]. Chabrolle and Rossier [6] described 5 cases of goiter and hypothyroidism after iodine skin preparation in newborn infants; subsequent studies have confirmed this finding [8, 9]. Because of the serious implications for growth and development, in most neonatal units povidone-iodine either is no longer used for skin preparation or is immediately washed off at the end of the procedure.

In adults, the skin is much less permeable, and there have been only two reports of transcutaneous iodine absorption, both involving massive exposure [10, 11].

Pediatric cardiac operations involve the preparation of substantial areas of skin, providing the potential for significant iodine absorption. In adults and older children (with less permeable skin), transcutaneous absorption is unlikely to be significant, but in small infants this may not be true and the documented complications of such absorbance could aggravate the inherent risks of cardiac operation in these very young patients. In particular, our own studies (unpublished results) have demonstrated a sharp decrease in triiodothyronine and thyroxine concentration in the first 48 hours after cardiopulmonary bypass, then slow recovery over the next 3 to 5 days. Because triiodothyronine is an inotrope and has an important role in maintaining myocardial energy stores, and because high plasma iodine concentrations are known to have an inhibitory effect on thyroid hormone production [12], these changes could have serious implications for postoperative cardiac performance.

We have therefore studied plasma iodine concentrations in term infants less than 3 months of age receiving povidone-iodine skin preparation for cardiothoracic procedures not involving cardiopulmonary bypass.

Material and Methods

Approval for this study was granted by the local ethics committee, and informed parental consent was obtained for each child who participated.

Seventeen term infants were studied. These were 6 male and 11 female infants with a mean age of 26 days (range, 1 to 84 days) and a mean body weight of 3.66 kg (range, 2.50 to 5.00 kg). Fifteen patients underwent thoracotomy and 2, sternotomy. Surgical procedures were a systemic to pulmonary artery shunt in 6, coarctation repair in 6, ligation of a patient ductus arteriosus in 2, lobectomy in 1, resection of a gastric duplication in 1, and mediastinal toilet (after a previous Blalock-Taussig shunt) in 1.

Skin preparation was carried out in a standard fashion. In 15 infants 10% aqueous povidone-iodine (Betadine; Napp Laboratories Ltd, Cambridge, UK) was used. The surface area covered was estimated by a single observer, using a standard pediatric burn chart, as being between 20% and 30% of the total body area (mean, 26%). For the duration of the operation, the exposed skin around the wound was covered with a Steri-Drape (3M Health Care,

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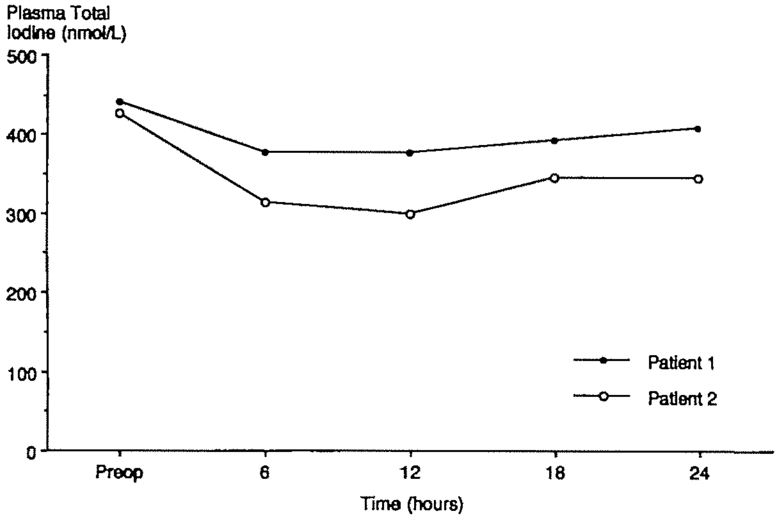

Fig 1. Plasma total iodine concentrations in 2 infants after chlorhexidine skin preparation.

Loughborough, UK). In 5 infants this was iodine-free, and in 9 it was impregnated with iodophor. In 2 children the skin was prepared with a 0.5% alcoholic solution of chlorhexidine (Hibisol; ICI Pharmaceuticals, Macclesfield, UK). Iodine-free Steri-Drapes were also used.

No child had been exposed to topical iodine-containing preparations within the previous 48 hours, and there was no further contact with povidone-iodine or any other iodine-containing compound over the following 24 hours. Blood samples were taken from arterial catheters immediately before skin preparation and at 6, 12, 18, and 24 hours after. Total plasma iodine concentrations were assayed by a modification of a spectrophotometric method described by Barker and associates [13].

Results

All children made a satisfactory postoperative recovery.

Preoperative total plasma iodine concentrations ranged from 260 to 900 nmol/L (mean, 634 nmol/L). In the 2 patients in whom chlorhexidine skin preparation was used, there was no increase in plasma total iodine concentration over the 24-hour period after operation (Fig 1). In contrast, after povidone-iodine skin preparation there was a rapid increase of between 160% and 1,440%, reaching a peak at 12 to 18 hours (mean, 2,579 nmol/L). This increase was apparent at 6 hours and sustained for more than 24 hours, and was significantly greater than the preoperative value at each sample point (6 hours, $p < 0.02$; 12 hours, 18 hours, 24 hours, $p < 0.01$ by t test). The results are summarized in Figure 2.

There was no difference in iodine absorption between those patients using plain or iodophor-impregnated Steri-Drapes ($p > 0.05$ by Wilcoxon test).

Comment

Total rather than protein-bound iodine concentration was measured to avoid any distortion due to fluctuations in plasma protein binding. Similarly, only patients undergoing closed cardiac procedures were studied to avoid the difficulty of interpreting biochemical changes after cardiopulmonary bypass but allow the investigation of patients having similar incisions and skin preparation.

Preoperatively, we observed a mean plasma total iodine concentration of 634 nmol/L, approximately half that previously reported in newborn term infants [7, 14]. Nevertheless after skin preparation with povidone-iodine there was a fourfold increase in plasma concentration, maximal at 12 to 18 hours, as found elsewhere [7]. There was no increase, however, in the 2 patients who did not have any contact with topical iodine, either as a skin preparation or as an iodophor-impregnated wound dressing. This clearly demonstrates that the elevated plasma iodine concentrations in the povidone-iodine–treated group were the result of transcutaneous absorption.

High plasma iodine concentrations are known to cause a transient inhibition of thyroid hormone production and a significant decrease in serum thyroxine level. This

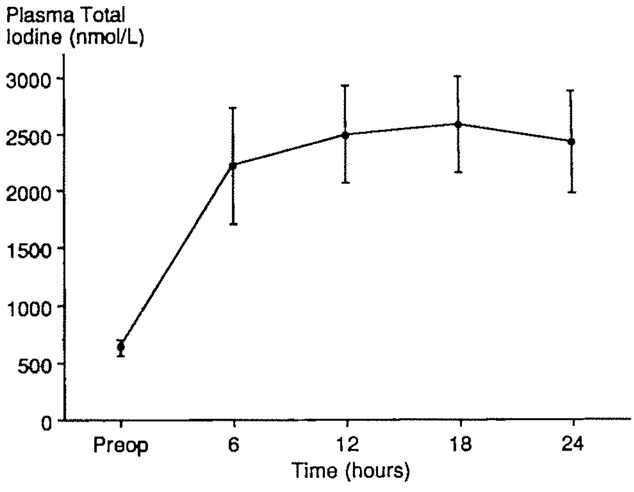

Fig 2. Plasma total iodine concentrations in 15 infants after skin preparation with povidone-iodine (mean and one standard deviation).

phenomonen, known as the Wolff-Chaikoff effect [12], is thought to be mediated by high intrathyroid concentrations of inorganic iodide. It usually lasts only 48 hours, even if plasma iodine levels remain high. However, in a minority of patients, prolonged hypothyroidism does occur, possibly due to HLA histocompatibility antigens and an inherited inability to counteract the Wolff-Chaikoff effect by down-regulating the thyroid/plasma iodide pump, so reducing thyroid iodine uptake [15]. In addition to an inherited defect, prematurity may also be associated with difficulty in overcoming the Wolff-Chaikoff effect, although in this series all infants were born at term.

After cardiopulmonary bypass, plasma triiodothyronine and thyroxine concentrations are known to decrease and remain low for 5 to 7 days. Replacement therapy in adults has been shown to increase stroke volume, reduce systemic and pulmonary vascular resistance, and significantly reduce the requirements for conventional inotropes [16]. There is, therefore, considerable short-term benefit in maintaining normal circulating concentrations of thyroid hormones after cardiac operations, quite apart from the long-term benefits for calorie regulation and for growth and development.

We have previously demonstrated that the triiodothyronine and thyroxine response to cardiopulmonary bypass is more profound in infants than in adults (unpublished results), and yet the long-term consequences of hypothyroidism are more important in this age group. In addition, the more complex nature of the operation in infants often leads to a higher rate of rethoracotomy in the first 48 hours and consequently, further iodine exposure. Because iodine excretion is predominantly renal, any degree of renal failure may also contribute to increased plasma concentrations. This is particularly important in neonates, in whom the glomerular filtration rate is lower than in adults.

In summary, we have demonstrated significant transcutaneous absorption of iodine after skin preparation with povidone-iodine in infants undergoing cardiac operation. Because of the potential consequences of this in terms of metabolic dysfunction, exacerbated hypothyroidism, and an increased risk of renal failure, we believe that a skin preparation that does not contain iodine should be used in infants less than 3 months of age. If this is not acceptable for reasons of antisepsis, then we believe that the iodine should be washed off immediately with sterile saline solution.

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Surgical Treatment of Supraventricular Tachycardia: Experience With 61 Patients

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Sixty-one patients underwent operation for supraventricular tachycardia: 52 had tachycardia associated with the atrioventricular accessory pathways; 9 patients had other forms of tachycardia. Accessory pathways were successfully divided in 92.3% of the patients. Classic endocardial approach was used in 30 patients; closed heart ablation of the accessory pathways was performed in 22 patients. There were two reoperations for return of conduction through the accessory pathways. Six patients underwent cryoablation of the His bundle through the right atrial approach. Four patients underwent attempts at curative operations for atrioventricular node reentry tachycardia, atrial flutter, and atrial ectopic tachycardia. Concomitant cardiac surgical procedures were performed in 14 patients. Operative mortality was 1.6%. A satisfactory result, without tachycardia and without medication, was achieved in 93.4% of all patients.

(Ann Thorac Surg 1991;52:1141–4)

During the past two decades surgery has become an acceptable mode for the treatment of supraventricular tachycardia. A variety of operative techniques are used in ablation of the accessory pathways (APs) [1–6] as well as in operation for other tachycardias [7, 8]. This report describes our initial experience with 61 patients who underwent surgical treatment of supraventricular tachycardia.

Material and Methods

Between January 1987 and May 1990 a total of 61 patients underwent investigations and operations for supraventricular tachycardia at our institution. All patients had medically refractory tachyarrhythmia.

Indications for operation included a reentry tachycardia using the APs in 49 patients and atrial fibrillation with atrioventricular (AV) conduction through APs in 2. One patient had both tachycardia caused by APs and tachycardia due to AV node reentry. Nine patients had supraventricular tachycardias not involving APs. Four of them had paroxysmal atrial flutter, 2 had chronic atrial fibrillation with rapid ventricular response, 2 had AV nodal reentrant tachycardia, and 1 patient had ectopic atrial tachycardia.

The patients' ages varied from 4 to 68 years, and 16 of them had some other cardiac disorder.

All patients underwent preoperative electrophysiological study; intraoperative cardiac mapping was performed in all patients. Free wall connections were found in 24 patients (right free wall in 13 patients and left free wall in 11 patients). Eighteen patients had posterior septal APs and 2 had anterior septal connections. Eight patients had multiple APs; 4 of them had right free wall and posterior septal connections, 2 patients had multiple right free wall APs, and 1 had multiple left free wall APs. One patient had both anterior and posterior septal APs.

Right free wall and anterior septal APs were ablated by closed technique on the beating heart without cardiopulmonary bypass. The heart was exposed through a median sternotomy. After completion of the epicardial mapping, dissection of the fat pad in the AV grove was performed. Wide dissections were used to mobilize coronary vessels. The fat pad was separated from the atrial wall toward the top of the external wall of the ventricle. The small vessels were divided. Once dissection has been completed, a cryoprobe with an active part 4 mm in diameter and 15 mm long was applied through the entire length of the dissection to incorporate part of the atrial wall, the adjacent AV junction, and the adjacent ventricular wall. The number of applications varied from 4 to 7. The zone of freezing was extended 2.5 to 3 cm on each side of the determined location of the AP. Free mobilization of the fat pad allowed avoidance of contact between cryoprobe and coronary vessels during cryothermia.

For ablation of anterior septal pathways we mobilized the AV fat pad in the right coronary fossa. The tricuspid valve annulus was exposed toward the AV junctional area. Then the tip of the cryoprobe was put into the space created between the atrial wall and the aortic root, and cryoablation (−70°C for 2 minutes) was applied.

Left free wall APs in 6 patients were ablated by a closed heart technique similar to that used for right free wall APs. The left anterolateral thoracotomy was employed in these cases. In 5 patients we used a transatrial approach for interruption of left free wall APs. The surgical technique used was analogous to the technique developed by Sealy and Gallagher [9]. The only distinction was appli-

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cation of the cryoprobe on the top of the left ventricle after completion of the dissection of the AV groove.

Posterior septal connections were divided through the right atrium. These operations were performed with the use of cardiopulmonary bypass and cold cardioplegia. We used the most commonly employed technique [3, 8, 10] including supraannular incision in the posterior septal area with consequent extensive dissection.

In all patients in whom correction of concomitant cardiac lesions was performed, a transatrial approach for interruption of an AP was used.

Before the chest was closed, epicardial mapping was performed repeatedly to assess antegrade and retrograde conduction. During the first 8 to 9 days after operation, AV and ventriculoatrial conduction were checked daily with the aid of temporary wire electrodes.

In 2 patients with AV nodal reentry tachycardia, discrete cryolesions were applied around the borders of the triangle of Koch according to the technique proposed by Holman and associates [7]. In 1 of those patients two APs were divided.

Six patients underwent cryoablation of the His bundle through the right atrial approach: 3 for paroxysmal atrial flutter, 2 for chronic atrial fibrillation, and 1 for AV nodal reentry tachycardia. Four of those patients had concomitant cardiac lesions that required surgical correction, and 1 patient had thrombi in the left atrium that resulted in multiple embolic episodes. For these reasons surgical ablation of the His bundle was performed instead of a transvenous catheter procedure.

Excision of an ectopic focus in the right atrium was performed in 1 patient with ectopic tachycardia.

In 1 patient with paroxysmal atrial flutter and an atrial septal defect we made an attempt to identify a possible circle of reentry on the basis of a consequence of atrial activation and the localization of a conduction delay zone. One part of this circle was cryoablated, and the other part was excised.

Concomitant cardiac surgical procedures were performed in 14 patients including repair of atrial septal defects in 4 patients, correction of Ebstein's anomaly in 3, mitral valve replacement in 2, mitral valvotomy in 2, and other procedures in 3.

Results

Table 1 demonstrates the operative results in 52 patients with APs. As can be seen in Table 1, the APs were successfully interrupted in 90% of patients. Epicardial mapping before completion of the operation demonstrated that conduction through the AP was absent in all patients. Return of conduction over an AP occurred in 5 of them within the first week postoperatively: in 2 patients with posterior septal APs ablated by endocardial dissection and in 2 patients with left free wall and 1 with right free wall connections ablated by the epicardial approach. Two of those 5 patients (one with posterior septal and 1 with left lateral AP) underwent successful reoperation.

There was one postoperative death in a patient who underwent interruption of a left free wall AP and open mitral valvotomy. This patient died of pulmonary insufficiency. One patient had permanent complete heart block because of interruption of the posterior septal AP. Postoperative complications are listed in Table 2.

Table 1. Results of Operation for Interruption of Accessory Pathways in 52 Patients

Localization of AP	No. of Patients	No. of Patients With Divided AP
Posterior septal	18	16
Right free wall	13	12
Left free wall	11	9
Multiple	8	8
Anterior septal	2	2
Total	52	47 (90.4%)

AP = accessory pathway.

Thus, all accessory AV and ventriculoatrial connections were successfully divided in 92.3% of patients. All patients in whom conduction through the AP was absent before discharge were free of tachycardia in follow-up.

In 5 of 6 patients who underwent cryoablation of the His bundle, permanent complete heart block was achieved; in 1 patient AV conduction was only modified but it slowed significantly the heart rate during atrial fibrillation.

Two patients who underwent perinodal cryoablation for AV nodal reentry tachycardia remained in sinus rhythm. The tachycardia could not be initiated in both patients by atrial and ventricular stimulation at electrophysiological study after operation. On follow-up they were free of symptoms.

In 1 patient who underwent curative operation for atrial flutter, the atrial stimulation after operation initiated an atrial tachycardia with cycle length of 330 milliseconds (the cycle length of atrial flutter before operation was 220 milliseconds). The paroxysms after operation did not occur spontaneously during a 10-month period without any medication.

In the patient with ectopic atrial tachycardia, epicardial mapping after excision of the ectopic focus showed an activation begun from sinus node region. He remains fully asymptomatic more than 1 year after operation.

Table 2. Complications After 54 Operations for Interruption of Accessory Pathways in 52 Patients[a]

Complication	No. of Patients
Temporary low cardiac output	4
Severe pneumonia necessitating ventilation	2
Wound infection	1
Lymphorrea necessitating reopening	1

[a] Overall incidence of complications was 14.8%.

Table 3. Results of Operation for Tachycardias Not Associated With Accessory Pathways

Variable	No. of Patients
No. of patients	10[a]
No. clinical SVT	10
Postoperative complications	0
Operative mortality	0
Late mortality	0

[a] Including 1 patient in whom curative operation for atrioventricular nodal reentrant tachycardia was performed in addition to ablation of an accessory pathway.

SVT = supraventricular tachycardia.

Results of operations for supraventricular tachycardias not associated with APs are summarized in Table 3.

Comment

Today the most experienced centers report excellent results of surgical treatment of the Wolff-Parkinson-White syndrome with success rate greater than 90% and mortality less than 1% [3, 8, 11]. The endocardial approach is the most commonly employed, but there are surgeons who use a closed heart technique [2, 4–6]. Is the ablation of APs by closed heart technique reasonable?

We obtained return of conduction over an AP in 2 (6%) of the 30 patients who were operated on by the open heart technique and in 3 (13%) of 22 who underwent external ablation of the AP. In the former group both recurrences occurred after ablation of posterior septal APs. Among patients who were operated on by the closed heart technique, all three recurrences occurred after ablation of free wall APs. Thus, when ablation of free wall connections was performed by the endocardial approach we had no recurrences, whereas in 20 patients with free wall APs operated on by the epicardial approach the frequency of recurrences was 15%.

It may be concluded that the endocardial dissection technique for ablation of free wall APs is more successful than the epicardial one. However, we have considerations that do not allow us to reject epicardial dissection as method of ablation of free wall APs. In our clinic the operations with cold cardioplegic cardiac arrest are associated with greater mortality and morbidity in comparison with those without cardiopulmonary bypass. In our series all complications occurred in patients who were operated on with cardiopulmonary bypass. Therefore, the ablation of the APs by a closed heart technique seems to us more safe than by an open heart technique.

The analysis shows that in 2 patients with recurrences after closed heart operations we made obvious errors. In 1 patient the zones of freezing did not overlap each other. In the other patient the ventricular wall near the valve annulus had been perforated by the tip of the cryoprobe. After repair of the defect, application of the cryoprobe around its borders were not done. In all cases of recurrence there was no evidence of conduction over APs immediately after operation. It reappeared from 20 hours to 7 days postoperatively. An epicardial mapping during reoperation in 1 of the patients who underwent closed heart operation and repeated electrophysiological study in another patient showed that conduction in both occurred through the same AP as before the first operation. Thus there was no error in initial localization of the AP and there were no other concomitant obscured APs. The cause of recurrence was insufficient "traumatization" of the AP. It seems that if the all steps of the procedure are carefully performed, closed heart ablation of free wall APs can be used.

In our little experience with anterior septal pathways we did not observe AV conduction disorders either in the early or in the late follow-up. In 1 patient temporary second-degree AV block occurred during freezing. Freezing was interrupted, the tip of the cryoprobe was moved more externally, and then freezing was resumed. We suppose that in both patients with anterior septal pathways we really ablated anterior septal APs. The cryomapping at the place of the earliest right atrial activation adjacent to the aortic root did not interrupt conduction over the AP, which indicates that the AP coursed more internally. The freezing in the space created after dissection resulted in the interruption of conduction through the AP.

Our first successful attempts at curative operation for AV nodal reentry tachycardia and atrial flutter allow us to employ these methods more widely. We now use ablation of the His bundle only in patients with atrial fibrillation who require correction of concomitant cardiac lesions.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1994 may begin the recertification process in 1992. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is May 1, 1992. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Thoracoscopy in the Diagnosis and Management of Recurrent Pleural Effusions

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Between April 1984 and July 1988, 102 patients with persistent or recurrent pleural effusions investigated by conventional methods in whom a diagnosis had not been achieved were submitted to thoracoscopy under general anesthesia. A positive diagnosis was obtained in 80.3% of the patients. The procedure was well tolerated, and although some of these were seriously ill patients, there were no procedure-related deaths.

(Ann Thorac Surg 1991;52:1145–7)

Thoracoscopy became popular early in the century for the division of tuberculous pleural adhesions. In 1925 Jacobaeus [1] suggested a diagnostic role for the thoracoscope, but little use was made of it. More recently thoracoscopy has regained popularity and has been a useful procedure in the etiological diagnosis of pleural effusions [2, 3]. The diagnosis of the cause of the pleural effusions is obtained in a large proportion of cases by simple aspiration and examination of the pleural fluid. Percutaneus needle biopsy of the pleura and bronchoscopy will add to the diagnostic yield, but there remains a group of patients with recurrent or persistent pleural effusions in whom these techniques have given a high percentage (up to 37.5%) of negative results [4]. The question addressed in this report is whether thoracoscopy will add further to the diagnosis in these patients in whom previous diagnostic methods have failed to achieve a histological diagnosis at the time of referral to the thoracic unit. Therefore, rigid thoracoscopy under general anesthesia has been used as the investigation of choice in 102 patients at our center.

Material and Methods

Thoracoscopy was carried out on patients who had had a recurrent pleural effusion that had been aspirated at least once with or without pleural biopsy and in whom these investigations had been nondiagnostic. There were 76 male (74.6%) and 26 female (25.4%) patients, ranging in age from 21 years to 86 years (mean age, 60.2 years). Sixty-four patients had a pleural effusion in the right pleura (62.7%), 37 patients had a left-sided pleural effusion (36.2%), and 1 patient (0.9%) had bilateral pleural effusions. The mean duration of pleural effusion was 7.2 weeks (range, 1 to 36 weeks). Table 1 shows the number of previously attempted pleural aspirations and needle biopsies; it is interesting to note that 1 patient had eight pleural aspirations and two percutaneous biopsies, all of which failed to give a histological diagnosis. (Thoracoscopy in this patient confirmed a diagnosis of malignant mesothelioma.) In 2 of the 102 patients, there was a suspicion of malignancy but there was insufficient material for accurate diagnosis. Bronchoscopy had been carried out in 37 (36.2%) patients with suspected hilar or pulmonary shadow and had been diagnostic in 5 patients, showing endobronchial adenocarcinomas in 2 and squamous cell carcinomas in 3. Thoracoscopy was not used for talc pleurodesis, but chemical pleurodesis was routinely performed in patients with recurrent pleural effusions using either diluted tetracycline (1 g) in case of chronic inflammatory disease or bleomycin (60 mg) in patients with malignant pleural effusions.

Thoracoscopy is carried out under general anesthesia with a single-lumen endotracheal intubation. Upon insertion of thoracoscope the lung falls away from the chest wall and an adequate view is obtained, thus making the use of a double-lumen endotracheal tube unnecessary. The patient is positioned in the full lateral position. The pleural cavity is aspirated with a wide-bore needle to confirm the presence of the effusion. Pleural fluid is sent for cytological and bacteriological examination and for direct smear for tuberculosis when suspected. A trocar and cannula is inserted and the effusion is sucked out. A rigid thoracoscope (Genito Urinary Company) is inserted and the pleural cavity is inspected with a 30-degree angled telescope. Adhesions and loculations may be broken down with diathermy or biopsy forceps to obtain an adequate view of the pleura, diaphragm, pericardium, lung, and mediastinum. Biopsy of abnormal areas is performed under direct vision. Appearances of abnormal tissues vary from inflammatory infiltration, loss of sheen on the pleura, diffuse thickening, and fibrous exudate covering local areas of thickening to frankly malignant seedling deposits, which may be up to 1 or 2 cm in diameter. All removed specimens are sent for histology. At the end of the procedure a wide-bore intercostal catheter is inserted and connected to underwater seal drainage and the lung is allowed to reexpand.

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Table 1. Previous Negative Diagnostic Investigations (n = 102)

Procedure	Number
Aspirated once	32
Aspirated twice	22
Aspirated three times or more	27
Percutaneous biopsy + 1 to 3 pleural aspirations	15
Percutaneous biopsy + 4 to 8 pleural aspirations	6

Results

A definitive diagnosis was obtained in 82 of the 102 patients (80.3%). In 20 patients thoracoscopy was nondiagnostic. In the diagnostic group, 21 patients had an inflammatory pathology; the remainder had malignancy in the form of secondary carcinoma in 28 patients, mesothelioma in 31, and primary adenocarcinoma and lymphoma in 1 patient each (Table 2). Thoracoscopy has allowed direct and excellent visualization of the thoracic cavity with generous biopsy specimens. Upon confirmation of diagnosis in 82 patients an appropriate treatment could be started without delay. The need for formal diagnostic thoracotomies was substantially decreased, because before the use of the thoracoscope in April 1984 all these patients would have been submitted to a minithoracotomy. Of these 102 patients, 7 required an open pleural biopsy to establish a histological diagnosis after thoracoscopy. The average hospital stay of the patients was 8.8 days (range, 3 to 62 days) and average duration of intercostal tube drainage required was 4 to 8 days (range, 2 to 28 days).

Histology at Thoracoscopy

Table 2 shows the positive histological diagnoses obtained at thoracoscopy. In the "inflammatory and other" group, the 8 patients with "diagnosis by exclusion" were those patients who had proven malignancy elsewhere and were strongly suspected of having malignant pleural effusions but in whom a diagnosis of benign pathology was confirmed at thoracoscopy. Of the 20 patients who had nondiagnostic thoracoscopies, 7 were investigated further by a minithoracotomy and open pleural biopsy, and a diagnosis of squamous cell carcinoma was confirmed in 4 and adenocarcinoma in 3. Additional bronchoscopy was performed in 3 other patients; 2 of them were found to have squamous cell carcinoma and 1 of them, an adenocarcinoma. A diagnosis of metastatic lymphoma was obtained by laparotomy in 1 patient and by skin nodule biopsy in 1 other patient. In the remaining 6 patients the primary pathology was attributed to the cause of the recurrent pleual effusion. In 2 patients the diagnosis of mesothelioma and squamous cell carcinoma of lung was obtained at postmortem examination. Table 3 shows the final diagnosis in those patients who had nondiagnostic thoracoscopy.

Table 2. Positive Histology at Thoracoscopy (n = 82; 80.3%)

Histology	Number
Inflammatory and other	
Nonspecific	13
Unknown cause: "diagnosis by exclusion"[a]	8
Secondary carcinoma	
Adenocarcinoma	14
Squamous cell carcinoma	4
Anaplastic carcinoma	6
Oat cell carcinoma	2
Lymphoma	1
Clear cell carcinoma	1
Malignant mesothelioma	31
Primary adenocarcinoma	1
Lymphoma	1

[a] See text for explanation.

Table 3. Diagnosis by Other Methods After "Nondiagnostic" Thoracoscopies (n = 20)

Diagnosis	Number
Squamous cell carcinoma	7
Adenocarcinoma	4
Chronic lymphatic leukemia	1
Pulmonary embolism	1
Left ventricular failure	1
Meigs' syndrome	1
Hereditary nephritis (Alport's syndrome)	1
Ankylosing spondylitis	1
Metastatic lymphoma	2
Mesothelioma	1

Complications

The procedure has been low in complications. An empyema developed in 1 patient, and mesothelioma at the thoracoscopy puncture site developed in 1 patient with mesothelioma. Although there were no procedure-related deaths, 11 patients died (10.7%) of their disease within 30 days of thoracoscopy. All but 1 had advanced malignant disease.

Comment

Thoracoscopy has provided a positive diagnosis in a high proportion of pleural effusions in whom a diagnosis had not been achieved by conventional investigations. The major advantage of thoracoscopy is that it gives the operator an opportunity to perform biopsy on suspicious-looking pleural lesions and nodules on the surface of the lung under direct vision. It is also possible to get good views in loculated pleural effusions because of the ability to break down the loculi, either with diathermy or with biopsy forceps. In addition, it is possible to carry out chemical pleurodesis at the same time.

Weissberg and Kaufman [5] used a mediastinoscope to perform thoracoscopy in 114 of their 127 patients satisfactorily. We found a dedicated instrument such as a Storz thoracoscope, with its specially designed diathermy and biopsy attachments, extremely useful. The most commonly employed procedure for obtaining an etiological diagnosis of pleural effusion is aspiration and needle biopsy of the pleura. Unfortunately, its reliability and diagnostic yield ranges between 30% and 64% [6, 7]. A plain roentgenogram and computed tomography of the chest help to locate a mediastinal mass, but a computed tomographic scan cannot provide a histological diagnosis. The main disadvantage of the needle biopsy, even if guided by computed tomography, is that only a small and sometimes insufficient amount of tissue is obtained for the histological examination. A limited thoracotomy with open pleural biopsy is a major procedure compared with a thoracoscopy. The risk of tumor seedling implantation in the incision is well known, and in 1 of our patients with malignant mesothelioma, implantation of the tumor seedling was found at the site of thoracoscopy.

Thoracoscopy is well tolerated, and in some cases it has been preferable to percutaneous biopsy and aspiration under local anesthesia. Thoracoscopy under local anesthesia with the flexible fiberoptic bronchoscope [4, 8] has been recommended, but the disadvantage here is the difficulty of obtaining biopsy material when the pleura is fibrous and the pleural nodules are hard. In addition, it is only possible to take small biopsy specimens, which makes the histological diagnosis, particularly of mesothelioma, difficult in the extreme.

In patients with recurrent pleural effusion, chemical pleurodesis was carried out, but only after the drainage had become minimal and there was radiological evidence that the lung had reexpanded to come into contact with the chest wall.

The high incidence of mesothelioma reflects the difficulty of diagnosis by conventional methods. Our catchment area for thoracoscopy includes several dockyards and other industrial establishments where asbestos was extensively used.

Many patients are unwell and have died of progression of their disease within 30 days of thoracoscopy, but there have not been any procedure-related deaths as such. The high diagnostic yield obtained at thoracoscopy justified the risk in these unwell patients.

In conclusion, thoracoscopy has provided a positive diagnosis in 80.3% of patients with recurrent pleural effusions not diagnosed by repeated thoracentesis, pleural biopsy, or bronchoscopy. In those patients in whom thoracoscopy was nondiagnostic, an open pleural biopsy gave a definitive diagnosis in 6.8%, but there still remains a minority of patients in whom a diagnosis will not be made before postmortem examination. Our success rate is comparable with that of other reported series [9]. Thoracoscopy proved effective in the diagnosis of recurrent pleural effusions, especially when malignancy was strongly suspected. The use of the thoracoscope substantially decreased the necessity for formal diagnostic thoracotomy.

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Evaluation of the Damus-Kaye-Stansel Operation in Infancy

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Thirteen patients, 12 of whom younger than 2 years, underwent a Damus-Kaye-Stansel procedure for complete transposition of the great arteries, ventricular septal defect, or double-outlet right ventricle and subpulmonary ventricular septal defect. In 6 patients, associated cardiac anomalies caused systemic flow obstruction. There were six hospital deaths (mortality rate, 42%). In a mean follow-up period of 57 months, 5 of 7 survivors required relief of right ventricular hypertension through conduit replacement or enlargement (4 patients) or conduit valve balloon dilation (1 patient). The aortic valve became regurgitant in 2 patients in whom it had been left in potential connection with the right ventricle. One patient has moderate pulmonary valve regurgitation. The main advantage of the Damus-Kaye-Stansel procedure is that it avoids coronary relocation; also, the spatial relationship of the great arteries and the coronary anatomy do not affect its feasibility. One drawback is the need for a conduit in infancy. Our present indication for Damus-Kaye-Stansel procedure is confined to double-outlet right ventricle with subpulmonary ventricular septal defect; 5 of 6 patients survived repair in this series. Possible indications are for patients with associated subaortic obstruction or unusual coronary arrangements. Fresh or cryopreserved homografts as extracardiac conduits and primary closure of the subaortic area may reduce the need for reoperation after Damus-Kaye-Stansel procedure.

(Ann Thorac Surg 1991;52:1148–53)

The advantage of anatomically corrective procedures [1–5] for patients with complete transposition of the great arteries and ventricular septal defect (VSD) is generally accepted. Early and midterm results of intraatrial repair and VSD closure are, in fact, unsatisfactory [4–6]. Since its introduction in 1976 [1], the arterial switch operation has been widely accepted as the procedure of choice for anatomical correction. For patients with associated left ventricular outflow tract obstruction, the Rastelli [7] and REV [8] procedures are also important in the surgical repertoire.

For editorial comment, see page 1033.

Fewer reports have appeared on the procedure advocated separately by Damus, Kaye, and Stansel [9–12]: essentially an arterial switch operation without coronary reimplantation. The pulmonary trunk is anastomosed end-to-side to the ascending aorta in order to constitute an outlet from the left ventricle, and continuity between the right ventricle and the bifurcation of the pulmonary trunk is established by an extracardiac valved conduit. Part of this procedure has been successfully applied as a means to bypass subaortic obstruction in certain types of single ventricle [13, 14].

The Mayo Clinic group [15] has published the largest series of patients with simple transposition, transposition with VSD, and double-outlet right ventricle with subpulmonary VSD treated by the Damus-Kaye-Stansel (DKS) procedure. Their results indicated a high mortality rate in patients less than 18 months of age. We report on a smaller sample of infants and children treated with the same procedure to highlight the effect of age on immediate survival.

Material and Methods

Patients

In a 7-year period we operated on 13 patients. Seven of them had complete transposition and VSD, and 6 had double-outlet right ventricle with subpulmonary VSD (Taussig-Bing anomaly). Mean age and weight at operation were 8.2 months (range, 12 days to 39 months) and 5.2 kg (range, 3 to 12.5 kg), respectively.

In 6 patients, associated anomalies resulted in obstruction of systemic flow. Aortic arch interruption was present in 2 patients and coarctation in 2 others; 2 patients had subaortic obstruction, in 1 associated with hypoplastic aortic arch and coarctation. Previous palliative procedures are indicated in Table 1.

Surgical Technique

Standard hypothermic cardiopulmonary bypass with crystalloid cardioplegia was used in 9 patients; in 4 others, deep hypothermia and circulatory arrest were employed. The aorta was dissected from the pulmonary trunk and cannulated as far distally as possible. Each caval vein was cannulated separately through the atrium. A single atrial

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Table 1. Clinical Characteristics and Surgical Results

Patient No.	Age at Operation (mo)	Diagnosis	Weight (kg)	Associated Malformations	Previous Operations	Mode of Aortic Valve Closure	Outcome	Follow-up (mo)	Re-operation	Semilunar Valve Fate
1	0.5	TGA, VSD	3.5	AAI	AAI repair	VSD patch	Died	. . .	. . .	. . .
2	3	TGA, VSD	4.3	. . .	. . .	VSD patch	Alive	30	No	. . .
3	5	TGA, VSD	4.5	SubAo St	. . .	VSD patch	Alive	60	Yes	. . .
4	6	TGA, VSD	4.2	. . .	PAB	Separate patch	Died	. . .	. . .	. . .
5	7	TGA, VSD	5.6	LSVC	. . .	Separate patch	Died	. . .	. . .	. . .
6	7	TGA, VSD	4.5	CoAo	Subcl flap	Separate patch	Died	. . .	. . .	. . .
7	8	TGA, VSD	5.4	. . .	. . .	VSD patch	Died	. . .	. . .	. . .
8	2	TB	3	CoAo, SubAo St	. . .	VSD patch	Died	. . .	. . .	. . .
9	3	TB	4.3	. . .	. . .	VSD patch	Alive	73	Yes	. . .
10	7	TB	6.2	AAI	AAI repair, PAB	. . .	Alive	54	Yes	AI
11	8	TB	4.3	. . .	. . .	Leaflet suture	Alive	86	BD; yes	AI
12	11	TB	5.8	. . .	A sept, PAB	Leaflet suture	Alive	87	BD	PI
13	42	TB	12.5	CoAo	Subcl flap, PAB	VSD patch	Alive	9	No	. . .

A sept = atrial septectomy; AAI = aortic arch interruption; AI = aortic insufficiency; BD = balloon dilation of conduit valve; CoAo = coarctation of the aorta; LSVC = left superior vena cava; PAB = pulmonary artery banding; PI = pulmonary valve insufficiency; SubAo St = subaortic stenosis; Subcl flap = subclavian flap aortoplasty; TB = Taussig-Bing type of double-outlet right ventricle; TGA = transposition of the great arteries; VSD = ventricular septal defect.

cannula was placed if circulatory arrest was planned. Bypass time averaged 155 minutes (range, 127 to 217 minutes) and aortic cross-clamp time, 77 minutes (range, 49 to 143 minutes). During the cooling period, the pulmonary trunk was transected close to its bifurcation or at the site of previous banding; the proximal stump was tailored obliquely to face the ascending aorta.

We introduced two modifications of the original technique: first, the aortic pathway was routinely occluded (except in 1 patient whose aortic valve was left open and connected with the right ventricle). Second, after the aorta–pulmonary artery anastomosis was completed (Fig 1A), the pulmonary confluence was closed either directly or with a pericardial patch, the arteriotomy was extended to the main right pulmonary branch (Fig 1B), and the valved conduit was anastomosed to it (Fig 1C). This maneuver allows the conduit to be positioned to the right of the ascending aorta and the aorta–pulmonary anastomosis to lie posterior and to the left of the ascending aortic segment, so that it is more accessible for control of hemostasis.

Various techniques were employed for the first described modification (Table 1). In the last 6 patients, the VSD patch was extended to the subaortic region, thereby placing the semilunar valve in communication with the left ventricle in a similar fashion to the Rastelli procedure (Fig 2). A wide-open subaortic tunnel is not necessary, as the left ventricle has an unobstructed outlet through the pulmonary trunk.

Heterograft valved conduits, size 12 or 14, were used to complete the operation. In 1 patient, a hypoplastic aortic arch associated with coarctation required pericardial patch reconstruction extended beyond the coarctation site.

Results

Six patients did not survive the immediate postoperative period (early mortality rate, 42%). Of 6 patients with double-outlet right ventricle, 5 survived.

One 12-day-old baby in desperate condition was operated on 24 hours after repair of concomitant interruption of the aortic arch; the pulmonary trunk had not been banded in this patient. One patient died of respiratory insufficiency after massive blood transfusion in the operating room. The pulmonary trunk was paper-thin after pulmonary artery banding and profuse bleeding occurred from the aorta–pulmonary anastomosis.

Acute myocardial failure followed an untimely attempt at extubation of another, apparently stable, child. In another patient, coronary air embolism occurred before the institution of cardiopulmonary bypass; this was the only patient in the sample whose left ventricular systolic pressure was less than systemic (ratio, 0.68) despite the presence of a ventricular septal defect.

Two deaths followed *Staphylococcus aureus* septicemia. In 1 patient endocarditis occurred after an otherwise uneventful postoperative recovery. Mediastinitis developed in the other patient after urgent DKS procedure; his condition deteriorated after cardiac catheterization complicated by iliac vein thrombosis.

In retrospect, technical mistakes or errors of judgment resulted in the death of three patients. The role of an inadequately prepared left ventricle in causing a fatal outcome in 1 patient is debatable.

Follow-up to date averages 57 months (range, 9 to 87 months). No patient died in this period. Of the 7 survivors, 4 required further surgical treatment. The right ventricle–pulmonary artery conduit became stenotic and was replaced with an aortic homograft (2 patients) or

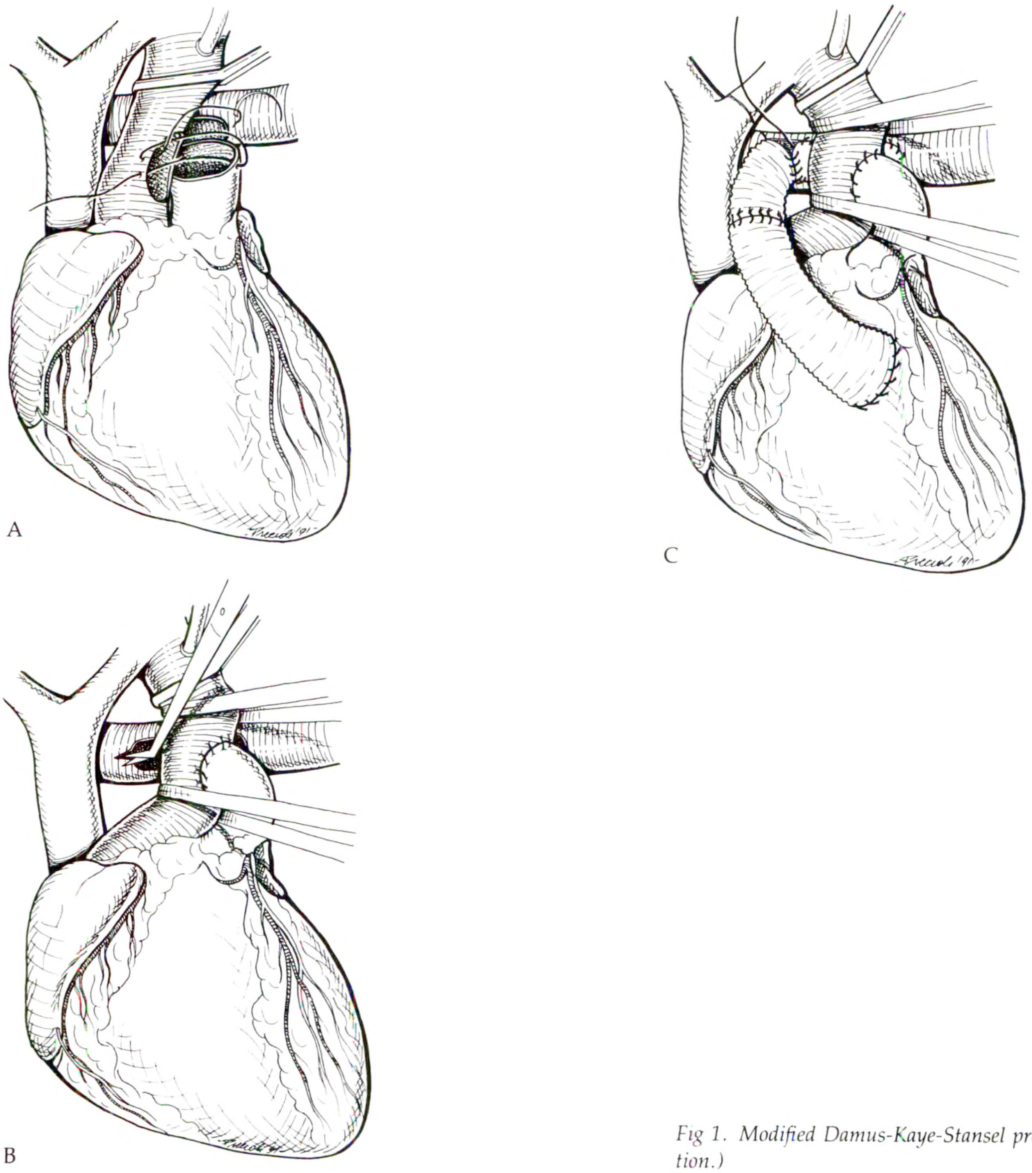

Fig 1. Modified Damus-Kaye-Stansel procedure. (See text for explanation.)

enlarged with a pericardial roof (1 patient) at 6, 7, and 3 years after the DKS procedure. One patient required three reoperations because of conduit stenosis and native aortic valve regurgitation. An aortic homograft was implanted at the second procedure but had to be replaced after 8 months owing to angulation under the sternum.

Conduit valve stenosis was identified as the main cause of right ventricle–pulmonary artery gradient and was treated by balloon dilation in 2 patients: in both the gradient was partially relieved (from 75 to 41 mm Hg and from 45 to 25 mm Hg). In 1 of these patients the conduit was replaced 2½ years later, whereas the other continues to do well, but has moderate right ventricular hypertension and dysfunction. Finally, 2 patients, both operated on in the last 2 years, are well with no evidence of right ventricular outflow tract obstruction. On the whole, 5 of 7 early survivors needed some type of right ventricular decompression.

Semilunar valve regurgitation was found in 3 patients: in 1 there was moderate pulmonary valve insufficiency, and in 2 there was aortic insufficiency. In the latter 2 patients the aortic valve was left anatomically connected to the right ventricle, with sutured leaflets in 1. This patient required reoperation for repeat suture of the aortic valve leaflets. One other patient has pulmonary insufficiency; despite this problem and associated right ventricular outflow tract obstruction he is currently asymptomatic. Pulmonary artery banding had previously been performed in this patient. The angiographic and cross-sectional echocardiographic appearance of the left and right outflow tracts after the DKS procedure are shown in Figures 3 and 4. Figure 5 shows the left ventriculogram of a patient in whom a double outlet from the left ventricle was surgically created. With this technique a wash-out effect of the sinuses of Valsalva and coronary ostia is obtained through the natural pathway.

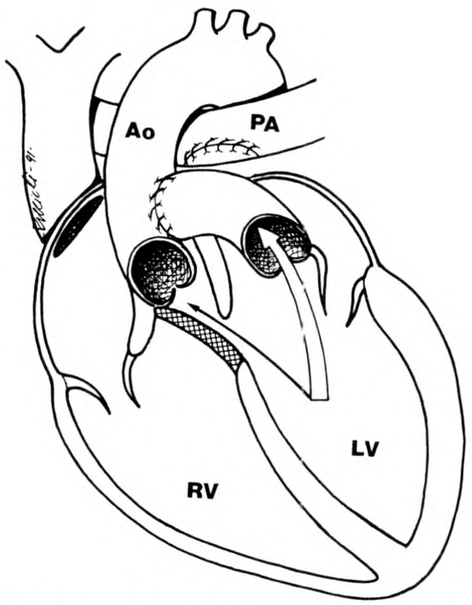

Fig 2. "Limited connection" technique: the right ventricle–pulmonary artery conduit is omitted for clarity of illustration. (See text for explanation.) (Ao = *aorta;* LV = *left ventricle;* PA = *pulmonary artery;* RV = *right ventricle.)*

Comment

In this small sample the hospital mortality was high, reflecting the complexity of the repair during the learning curve. These statistics are similar to the first results of the arterial switch operation. The early mortality in infants younger than 18 months was 50%; the Mayo Clinic group obtained similar results ($p = 0.77$, not significant). Low age and weight in our experience did not preclude surgical success, but the use of a prosthetic conduit for right ventricle–pulmonary artery connection substantially contributed to morbidity and, in some cases, mortality. From this point of view the procedure must be considered no more than palliative. The negative impact of residual defects such as pulmonary outflow tract stenosis, pulmonary insufficiency, or a combination of the two on late survival must be kept in mind. In this sample, all patients with a longer follow-up interval than 36 months had to undergo further operations. Some of them still have mild or moderate right ventricular outflow tract pressure gradients and right ventricular dysfunction.

In most reoperations fresh or cryopreserved homografts replaced the original conduits; in an "adult" size, these may offer a satisfactory performance for an indefinite number of years [16].

Another cause of reoperation was aortic incompetence, which occurred when this valve was left communicating with the right ventricle. Because of the peculiar physiology of the DKS procedure, aortic insufficiency results in varying degrees of left-to-right shunt and is, therefore, poorly tolerated [17].

The native pulmonary valve, as in the Jatene operation (arterial switch), will always work under systemic pressure load. Late development of regurgitation has been observed after arterial switch operation [3–5] and palliative pulmonary–ascending aorta anastomosis [17, 18]. This complication occurred in 1 of the patients in our sample (14% of early survivors). Its cause is unclear; if the

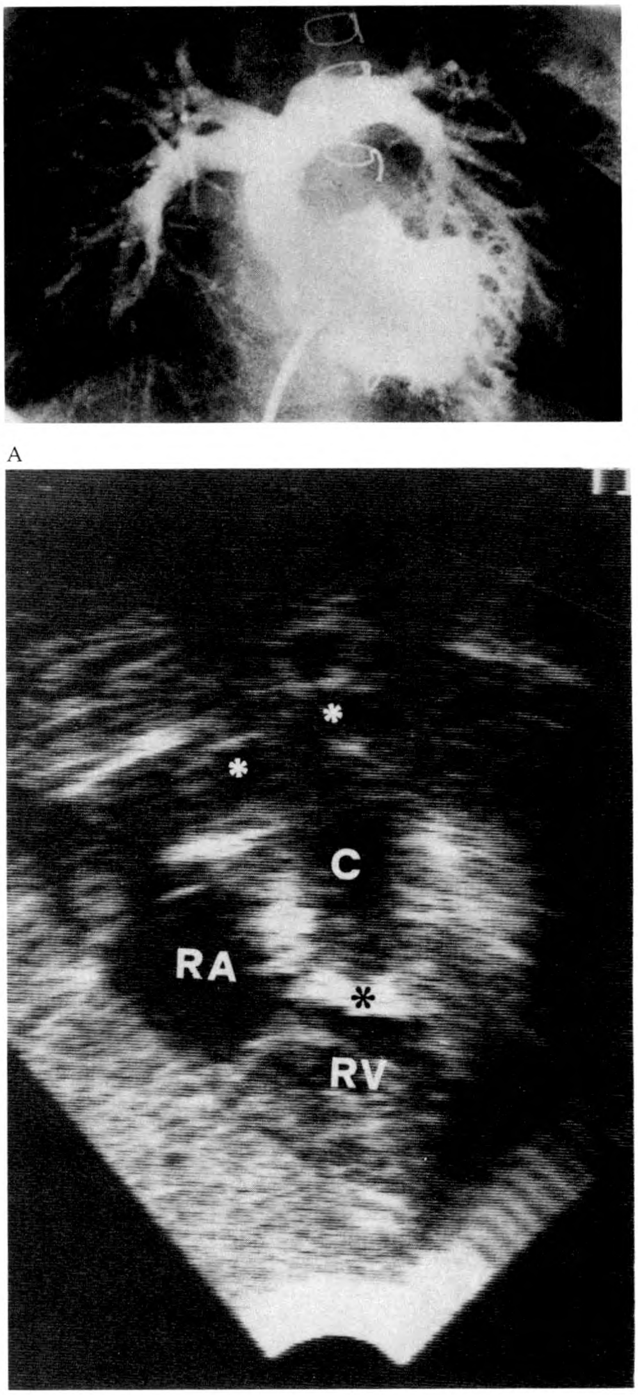

Fig 3. Angiographic (A) and cross-sectional echocardiographic (B) appearance of the right ventricular outlet after the Damus-Kaye-Stansel procedure; the black asterisk *marks the conduit valve, and the* white asterisks *mark the main pulmonary branches.* (C = *conduit;* RA = *right atrium;* RV = *right ventricle.)*

valve were intrinsically unable to function as the aortic valve, the incidence of regurgitation should be similar for the two arterial switch procedures.

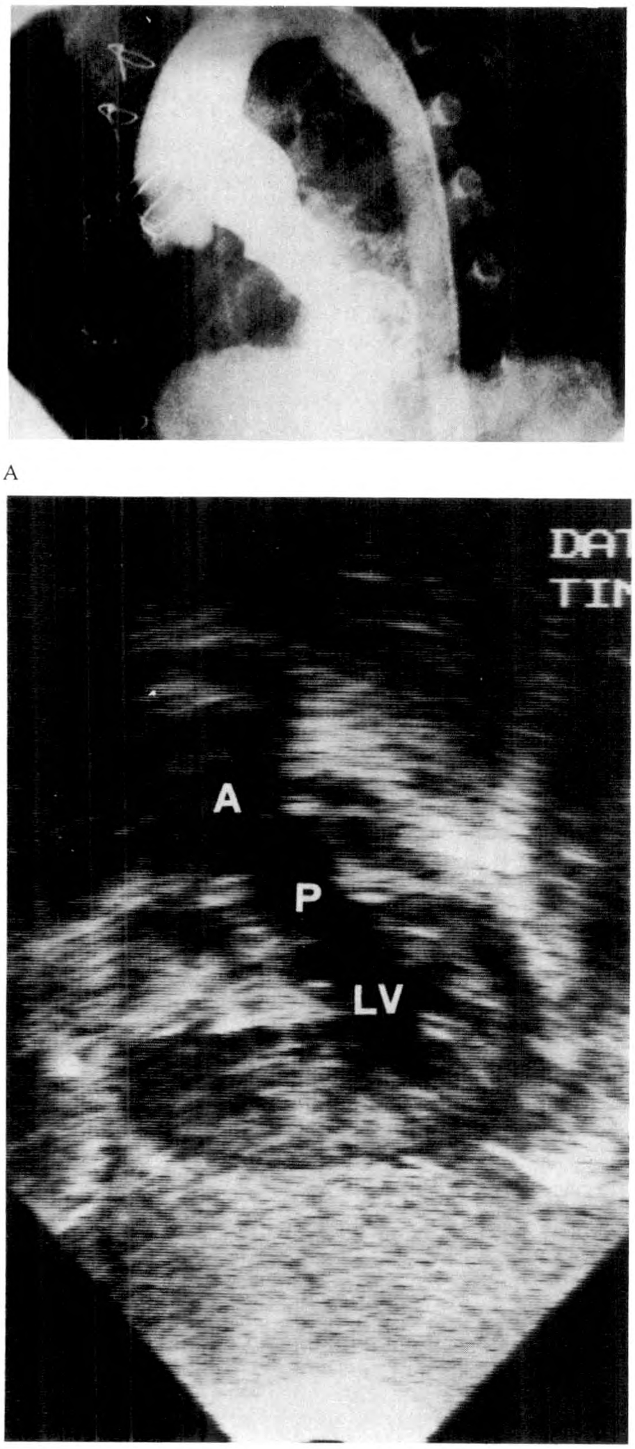

Fig 4. Angiographic (A) and cross-sectional echocardiographic (B) appearance of the left ventricular outlet after Damus-Kaye-Stansel procedure. (A = *ascending aorta;* LV = *left ventricle;* P = *pulmonary trunk.)*

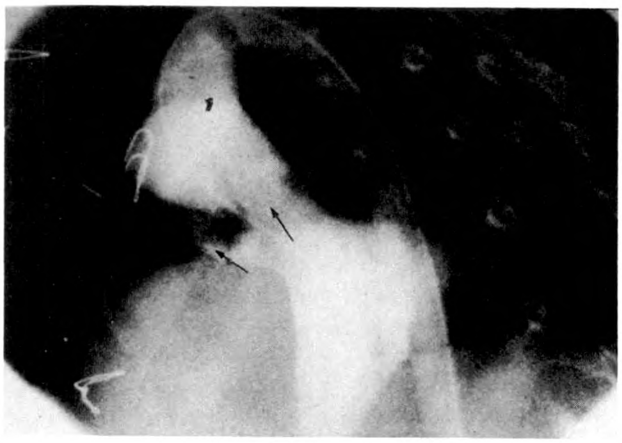

Fig 5. Left ventricular contrast injection; in this patient the subaortic region was excluded from the right ventricle by extension of the ventricular septal defect patch A double-outlet left ventricle is demonstrated (arrows).

The presence of a pulmonary band, in our experience, complicated the procedure by producing scarring and shortening of the pulmonary trunk. Hemostasis of the aorta–pulmonary anastomosis was time-consuming. We now believe that, with the exception of newborns with associated aortic arch interruption, banding should not be performed if a DKS procedure is planned.

Our current indications for this operation concentrate on the Taussig-Bing anomaly, in which our experience and that of other groups is favorable. Thirteen of 14 reported patients, ours included, survived [17, 19–23].

Patients with complete transposition and VSD form a fairly homogeneous population in which a Jatene procedure may now be safely performed. Nevertheless, select patients might be, in our view, considered for a DKS procedure. The association of subaortic obstruction makes these patients less than ideal candidates for the Jatene procedure [23, 24]. The association of aortic coarctation might suggest the possibility of subaortic stenosis, even in the absence of pressure gradients; this is often the case in a double-outlet right ventricle of the Taussig-Bing type [25]. One should also be aware of particular arrangements of the coronary system that would require complex repairs [25, 26] or contraindicate the Jatene operation [27]. This is true for the intramural course of a major coronary vessel [26].

In conclusion, the DKS operation permits an anatomical repair of transposition defects. It is always feasible, independent of great vessel position or coronary arrangement; furthermore, coronary artery manipulation is avoided. Its drawbacks are the need for ventriculotomy and subsequent conduit replacement(s). The use of fresh or cryopreserved homografts during the initial procedure would respond in part to this consideration. Immediate aortic valve occlusion or "limited connection" with the systemic ventricle should also help reduce the number of reoperations required by this procedure.

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Ivor Lewis Operation for Epidermoid Carcinoma of the Esophagus

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One hundred patients, 95 men and 5 women with a mean age of 59 years (age range, 35 to 77 years), were treated by the same initiate surgeon in practice from 1982 to 1988 for epidermoid carcinoma of the lower two-thirds of the esophagus using the Ivor Lewis procedure. Fifty-eight tumors were located in the middle third of the esophagus and 42, in the lower third. Postoperative staging revealed 30 stage I/II and 70 stage III carcinomas (ie, tumors extending beyond the esophageal wall or lymph node extension). Operative procedure was considered curative in 70 patients and palliative in 30 patients. The same procedure has been used for all patients. In all patients we were able to perform extended esophagectomy with anastomosis located 3 to 7 cm under the pharyngoesophageal junction. Postoperative mortality was 4%. Morbidity due to leakage was 7%; proper drainage enabled spontaneous healing in 5 patients. Fifteen patients had pulmonary complications, none of which fatal. Median actuarial survival was 17 months. Actuarial survival at 24 months is significantly higher for patients in stage I and II (68.4%) than for patients in stage III (23.2%) ($p < 0.01$). The Ivor Lewis procedure is a safe surgical approach for the treatment of the esophageal carcinoma that has a high survival rate and allows a good quality of life.

(Ann Thorac Surg 1991;52:1154–7)

In 1946, Ivor Lewis [1] proposed an operation for the surgical treatment of the middle and lower thirds of the esophagus. Extensive mobilization of the stomach and duodenum through a laparotomy is performed and is followed by a right thoracotomy for resection and reconstruction in the apex of the chest. This operation has been the main choice in our university unit over the last decade. Our aim in this report is to review the experience of a new surgeon in practice with his 100 initial esophagectomies. Conclusions should be drawn from this experience on what can be expected from these operations.

Material and Methods

Between 1985 and 1988, 95 men and 5 women with a mean age of 59 years (age range, 35 to 77 years) were operated on. Sixty-eight patients smoked more than 10 cigarettes per day, and 70 patients were considered to have heavy alcohol intake with consumption of more than 80 g of alcohol per day. Of 65 patients in whom liver biopsies were performed during the abdominal stage of the operation, 31 (49%) were found to have alcohol-related hepatitis and steatosis. Four patients had cirrhosis. These 100 patients represent 48% of all patients seen with a diagnosis of esophageal carcinoma. During the same period 46 patients with upper localization had the Akiyama procedure.

Thirteen patients had associated carcinomas of the mouth, pharynx, or larynx. Eight had been previously treated and healed, whereas 5 had synchronous lesions diagnosed during preoperative evaluation. Eighty-one percent of the patients had dysphagia, and the mean weight loss was 6% (range, 0% to 20%).

Surgical Procedure

In each patient, a median laparotomy and a right thoracotomy were performed. Through an epigastric laparotomy incision, the stomach was fully mobilized, preserving the right gastric artery from its origin at the level of the pylorus.

Mobilization of the head of the pancreas and pylorus was completed in all patients. Pyloroplasty was added in the last 33 patients. Liver biopsy was performed in 65 instances. One splenectomy was required due to an iatrogenic laceration. A feeding jejunostomy tube was added in all patients for the postoperative period.

The right thoracotomy incision was through the sixth intercostal space. Esophagectomy and lymphadenectomy were conducted without pulmonary exclusion. The stomach was then brought up into the chest. The esophagogastric anastomosis was completed in an end-to-side fashion using 25-mm EEA (Auto-Suture) or ILS (Ethicon) staplers. The instrument was introduced through a gastrotomy on the lesser curve, and the anastomosis was made on the posterior wall of the stomach. No dilation of the esophageal stump was performed. Once the anastomosis was completed, closure of the interposed stomach was accomplished using the TA 90 stapler placed on the smaller curvature of the gastroplasty. The gastrotomy site was then resected along with the lesser curve to a point just under the third branch of the left gastric artery. The gastroplasty was routinely attached to the mediastinal pleurae at the apex of the chest and excluded to prevent any eventual contamination of the pleural cavity. A posterior drainage tube was laid parallel to the plasty, its

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Table 1. Morbidity and Mortality in 100 Patients

Complication	No.	Deaths
Leakage	7	2
Hemothorax	3	0
Chylothorax	1	0
Recurrent nerve palsies	7	0
Pulmonary complications	15	0
Cardiac failure	2	1
Enterocolitis	1	1

superior tip being at the level of the anastomosis. The length of the esophageal remnant was estimated to be 3 to 7 cm after the anastomosis. The total time of operation was between 3 and 6 hours (mean, 4 hours).

Postoperative Care

All patients had respiratory assistance for periods varying between 24 hours and 60 days (mean, 4 days). Enteral nutrition by means of the jejunostomy began on the third day after operation. Antibiotics were administered in a preventive fashion using cefazolin and metronidazole.

The chest tube was removed when less than 50 mL drained over a 24-hour period. Oral feeding was resumed if no leakage was suspected after Gastrografin examination on the sixth day. The anastomosis was assessed endoscopically 3 weeks after the operation.

Adjuvant Therapy

Twenty-four patients received chemotherapy preoperatively (5 patients) or during the postoperative period (19 patients). A combination of cisplatinum and 5 fluorouracil was administered to all 24 patients who agreed to be part of a multicenter prospective and randomized trial.

Results

Staging of Results

Patients were classified into three groups according to the TNM American Joint Committee for Cancer staging system [2] as follows: stage I: T1 N0 M0, 19 patients; stage II: T2 N0 M0, 11 patients; stage III: T3 N0 M0, 21 patients, T2 N1 M0, 12 patients, and T3 N1 M0, 37 patients. All lesions were epidermoid carcinomas, 58 located in the distal third and 42 in the middle third of the esophagus. Thirty resections were considered palliative according to the following pathological documentation: part of the tumor was unresected due to direct extension, macroscopically positive lymph nodes were left in place, or a histologically positive margin was noted.

Morbidity and Mortality

Follow-up was available in all but 1 patient. Patients were assessed every 4 months for the initial year and every 6 months thereafter.

Early morbidity and mortality are summarized in Tables 1 and 2. There were 4 in-hospital postoperative deaths. One acute pulmonary edema and one necrotizing enterocolitis of unknown cause were lethal in 2 patients. Two patients had a lethal fistula: 1 had cirrhosis and died on the 75th day although the fistula was well drained, and the other had a palliative resection and died of severe cachexia on the 35th day.

A total of seven fistulas occurred in this series, in three of 70 curative resections and four of 30 palliative resections. Two fistulas were truly anastomotic, whereas the others were located on the stapler line of the gastric interposition. One patient had to be reoperated on to ensure proper drainage of the fistula. Of the six remaining fistulas, four healed spontaneously thanks to 16 to 40 days of appropriate drainage and enteral or parenteral alimentation.

Nine patients required a second operation, 3 for intrathoracic bleeding (gastric resection line, 1; heparin therapy complication, 1; and after removal of the chest tube, 1). Thoracic duct ligation was required for 1 patient with chylothorax. Two laparotomies were required owing to complications at the jejunostomy site: one small bowel volvulus and one intraabdominal leakage from the jejunum. Three eviscerations occurred with one thoracic wound dehiscence.

Pulmonary complications were the most frequent nonsurgical complications, occurring in 15 patients. For 8 patients these complications led to ventilatory support for 1 to 3 months; none was fatal. Cardiac complications were one myocardial infarction and the massive pulmonary edema that was fatal in 1 patient.

Mean hospital stay was 29.5 days (range, 14 to 140 days).

Functional Results

There were 16 anastomotic strictures, easily controlled by one dilation for 12 patients; 4 patients required two dilations. When we used EEA staplers, we observed stenosis in 32%; the rate was 11% when we performed the anastomosis with the ILS stapler (not significantly different). Strictures developed a few weeks after operation and were all benign.

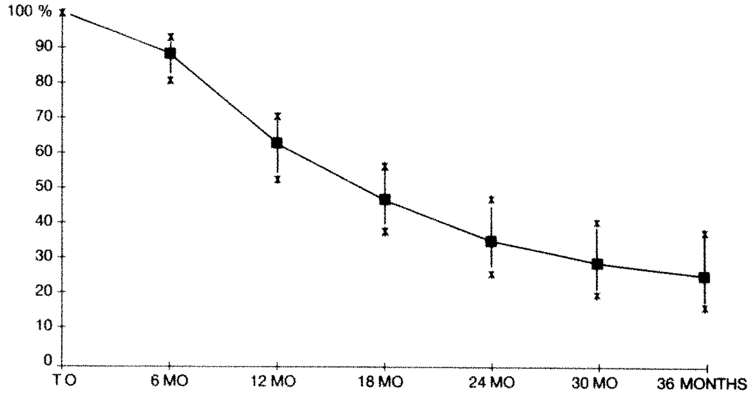
Major gastric emptying dysfunction occurred in 9 patients. A pyloroplasty had to be performed on 2 of those up to 6 months after the esophageal resection. One patient felt hardly improved after this late reintervention. All emptying complications were observed exclusively in the 67 patients who initially did not have pyloroplasty. No gastric emptying problems were observed in the 33 patients who received a pyloroplasty ($p < 0.05$).

Table 2. Early Results of Curative and Palliative Resections

Variable	Curative	Palliative	*p* Value
No.	70	30	
Deaths	3	1	NS
Leakage	3	4	NS
Wound dehiscence	1	5	<0.05
Pulmonary complications	4	11	<0.001

NS = not significant.

Fig 1. Overall actuarial survival after Ivor Lewis operation in 100 patients with carcinoma of the esophagus.



Recurrences

Twenty-one recurrences were documented; 7 were anastomotic, all confirmed by endoscopy in patients with dysphagia. Twelve were mediastinal, with bronchial infiltration in half of the cases and asymptomatic gastric infiltration in 2 patients. One patient had a pyloric stricture at the level of the esophageal hiatus.

A recurrence or a second esophageal cancer occurred in 1 patient 17 months after operation, documented immediately below the upper sphincter. Radiotherapy controlled this lesion with no evidence of disease 4 months after the diagnosis.

Survival

Median actuarial survival is 17 months (Fig 1). Actuarial survival at 24 months is significantly higher for patients in stage I and II (68.4%) than for patients in stage III (23.2%) ($p < 0.01$) (Fig 2). In this series there is no difference in survival among stage III patients with T3 N0 and T3 N1 tumors.

Comment

Several procedures are used for the treatment of epidermoid carcinoma of the esophagus. They may vary in radicality (en bloc versus blunt) or in exposure of the level of esophagogastric reconstruction (cervical or intrathoracic). The main purpose of the treatment remains an attempt at a curative resection while offering at the same time good palliation of the dysphagia. The main interest of our series is its homogeneity and the fact that these 100 patients represent the starting experience of a young surgeon beginning his practice.

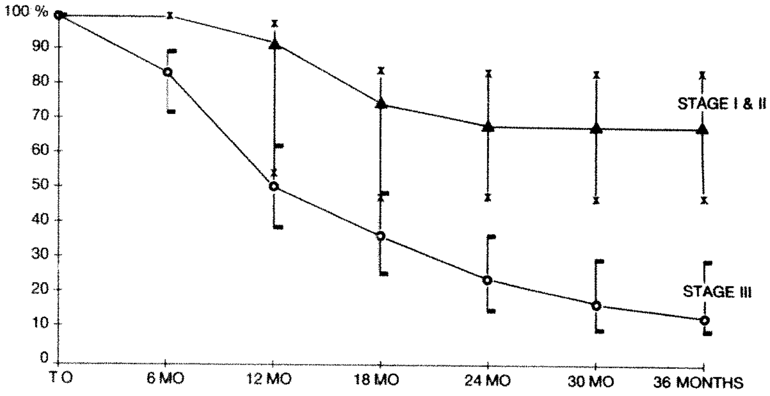
A standardized procedure has been used in every patient without major changes in operative or postoperative management. The observation is limited in time (7 years), and the follow-up is complete for 99% of the group. Only epidermoid carcinomas are considered because this is an entity quite different from adenocarcinomas of the esophagogastric junction, both in scope and in evolution.

The initial reported mortality of the Ivor Lewis operation varied from 8.7% to 28% [3, 4]. Since the early 1980s, the mortality of reported series has ranged between 3% and 14.3% [5–7]. Mathisen and associates [8] reported no mortality and no fistula formation in 104 patients in whom 40 Ivor Lewis operations were performed with a two-layer hand-made anastomosis in the right chest. In the study by King and co-workers [5], there were three deaths within 30 days of operation.

The in-hospital mortality observed in our study is 4% for the first 100 patients treated by a surgeon starting in practice. Fifty percent of the deaths were related to anastomotic complications in patients with a poor overall status. We conclude that the mortality to be expected in an initial experience with esophagectomy may come from technical or judgmental errors. Unforeseen complications may also play a role in increasing morbidity and mortality. There was no difference in mortality between those patients who underwent curative operation (3 of 70) and those who received only a palliative resection (1 of 30).

Morbidity is related to the anastomosis and to respiratory function. Reduction in these complications could be obtained, in our opinion, by following certain technical details. Construction of the anastomosis and intrathoracic completion of the thoracic gastric tube seems an impor-

Fig 2. Comparative actuarial survival of patients with stage I or II (30 patients) or stage III tumors (70 patients).



tant time saver. The 7% incidence of radiologically documented fistulas may seem high. It is an accurate number in a group of patients who were all evaluated with the objectivity of radiology. No fistula appeared after it was initially ruled out by the esophagogram on the sixth postoperative day. The incidence of fistulas may be reduced by completing the esophagogastric reconstruction at a lower level on the gastric tube, where the blood supply is known to be more adequate [9]. The widest possible margin should be left between the anastomosis and the stapling line on the lesser curvature of the stomach. Five of the seven fistulas were actually gastric perforations in this specific area or on the most proximal portion of the gastroplasty. Exclusion of the anastomosis from the pleural cavity in the thoracic inlet may help in avoiding any traction on the reconstruction by suspending the stomach by separate stitches on the apical pleura. Proper positioning of the chest tube and removal of the tube only after complete exclusion of all fistulous complications ensures the most appropriate drainage if such a complication becomes manifest.

The performance of a high chest anastomosis raises two questions: is the length of resected esophagus adequate, and does this approach result in a higher anastomotic recurrence rate? With systematic endoscopic documentation of the esophageal remnant at 3 weeks, the level of the anastomosis was shown to be 3 to 7 cm under the pharyngoesophageal junction, with a mean length of 5 cm. This observation confirms that of Richelme and associates [10], who did not find major resection length differences between patients reconstructed through the chest or through a cervical approach. True anastomotic recurrences were seen in 7 patients, and in those the resection showed a margin of 7.4 cm from the tumor (range, 5 to 12 cm). As Wong and others [11, 12] emphasized, the margin from the tumor shows a direct correlation with anastomotic recurrence.

Selection of patients probably remains the most difficult task, for the young surgeon as well as for more experienced clinicians. Thirty percent of our patients had a palliative operation. Mortality and fistula formation were not higher in this group when compared with the curative resection group. Pulmonary complications, however, were significantly more frequent in this group (37%) than in the group undergoing curative operation (6%) ($p < 0.001$).

Owing to the short survival expectancy if the procedure is determined preoperatively to be only palliative, severe emphysema and poor respiratory function have become strong selection criteria for exclusion of these high surgical risks.

In this series, survival is strongly dependent on the stage of the disease. Lymph node involvement plays an important role, although for stage III tumors there is no significant difference between T3 N0 M0 and T3 N+ M0 in terms of survival, suggesting that for large tumors extending beyond the esophageal wall, lymph node involvement has little importance with respect to the poor prognosis. Owing to relatively short follow-up in our series, the 5-year survival could not be calculated. The 2-year actuarial survival is 38.8% and compares favorably with reports in the literature where the Ivor Lewis procedure was used for epidermoid carcinoma [13, 14]. Recently, Chasseray and colleagues [7], in a prospective study, reported no difference between the Ivor Lewis and Akiyama techniques in terms of 2-year survival (47% for both groups). Akiyama and co-workers [14] achieved a 5-year survival of 34.7% using an abdominal, thoracic, and cervical approach.

In conclusion, the Ivor Lewis operation for esophageal carcinoma has proven to be a safe procedure for a surgeon starting in practice. A meticulous and well standardized technique will result in adequate curative and occasionally palliative resections. Morbidity should be reduced to a minimum, and satisfactory control of dysphagia should be obtained.

We thank André Duranceau, Professor of Surgery, Hotel-Dieu, Montreal, Canada, for his invaluable help in the writing of this article.

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Propranolol for Intractable Hemolysis After Open Heart Operation

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Postoperative intravascular hemolysis occurring in 2 patients was alleviated by propranolol. One patient underwent mitral valve replacement and had development of intractable hemolysis due to a paravalvular leak. The other patient underwent ventricular septal defect closure and had hemolysis caused by the Dacron patch. Both patients were given oral propranolol, and the degree of hemolysis decreased substantially. Although the exact mechanism of the propranolol effect on mechanical intravascular hemolysis is unclear, propranolol is thought to reduce the shearing stress between erythrocytes and the foreign material by slowing the velocity of the circulation.

(Ann Thorac Surg 1991;52:1158–60)

Intravascular hemolysis after open heart operations often results from mechanical destruction of erythrocytes due to interference between intracardiac foreign bodies and residual cardiac defects. Further operation is usually indicated in such patients unless the hemolysis spontaneously subsides. We treated 2 patients whose intractable intravascular hemolysis after open heart operation was alleviated with the use of propranolol.

Case Reports

Patient 1

A 58-year-old woman with a typical history of rheumatic fever had undergone open mitral commissurotomy for mitral stenosis when she was 51 years old. Seven years later exertional dyspnea developed, and cardiac catheterization revealed moderate mitral stenosis, mild mitral regurgitation, and severe tricuspid regurgitation. At operation, a severely deformed mitral valve was replaced with a St. Jude Medical prosthesis (27 mm) using 2-0 polypropylene (Prolene; Ethicon, Somerville, NJ) continuous sutures. The annulus of the tricuspid valve was reduced from 60 mm to 40 mm using DeVega's technique. The postoperative course was compromised by a moderate degree of low cardiac output syndrome. She left the intensive care unit on the 10th postoperative day.

On the 20th postoperative day, her urine was found to be black. A weak pansystolic blowing murmur was heard at the left sternal border. The hemoglobin value was 73 g/L, the maximum serum lactate dehydrogenase level was 7,525 IU, and the plasma free hemoglobin level was 879 mg/L. There was moderate hepatic and renal dysfunction. Peripheral blood examination for hemolysis excluded intrinsic erythrocyte abnormalities. Although left ventriculography, performed on the 39th postoperative day, did not detect a paravalvular leak around the mitral valve prosthesis, mechanical hemolysis due to a paramitral prosthetic valve leak was highly suspected, and 30 mg of propranolol (0.8 mg/kg) was prescribed on the 131st postoperative day.

Six days after propranolol administration was started, her urine became clear, the lactate dehydrogenase level declined to 1,480 IU, and the plasma hemoglobin was 52 mg/L. However, the patient did not tolerate the propranolol, and her cardiac size increased and lungs became congestive. Administration of propranolol was discontinued on the 186th postoperative day. Her urine immediately became black again, and the lactate dehydrogenase and plasma hemoglobin levels rose to 4,240 IU and 498 mg/L, respectively. A second postoperative left ventriculogram disclosed a tiny paravalvular leak. At reoperation, a 2-mm paravalvular leak was detected and closed with direct suture. Postoperatively, there was no evidence of intravascular hemolysis and she was discharged from the hospital. Her clinical course is shown in Figure 1.

Patient 2

A 2-year-old boy was diagnosed with a ventricular septal defect, coarctation of the aorta, patent ductus arteriosus, and pulmonary hypertension soon after birth. When he was 2 months old, he underwent subclavian flap repair for coarctation, ductus ligation, and pulmonary arterial banding in our hospital. At the corrective operation, a large subarterial ventricular septal defect was closed through a main pulmonary arteriotomy with a Dacron double-velour patch (Meadox, Oakland, NJ) using eight buttressed polyester sutures (Ethibond; Ethicon). After excision of the short, narrowed, and scarred segment of the main pulmonary artery, leaving the most posterior portion, the main pulmonary artery was reconstructed with direct anastomosis using 6-0 polypropylene sutures (Prolene).

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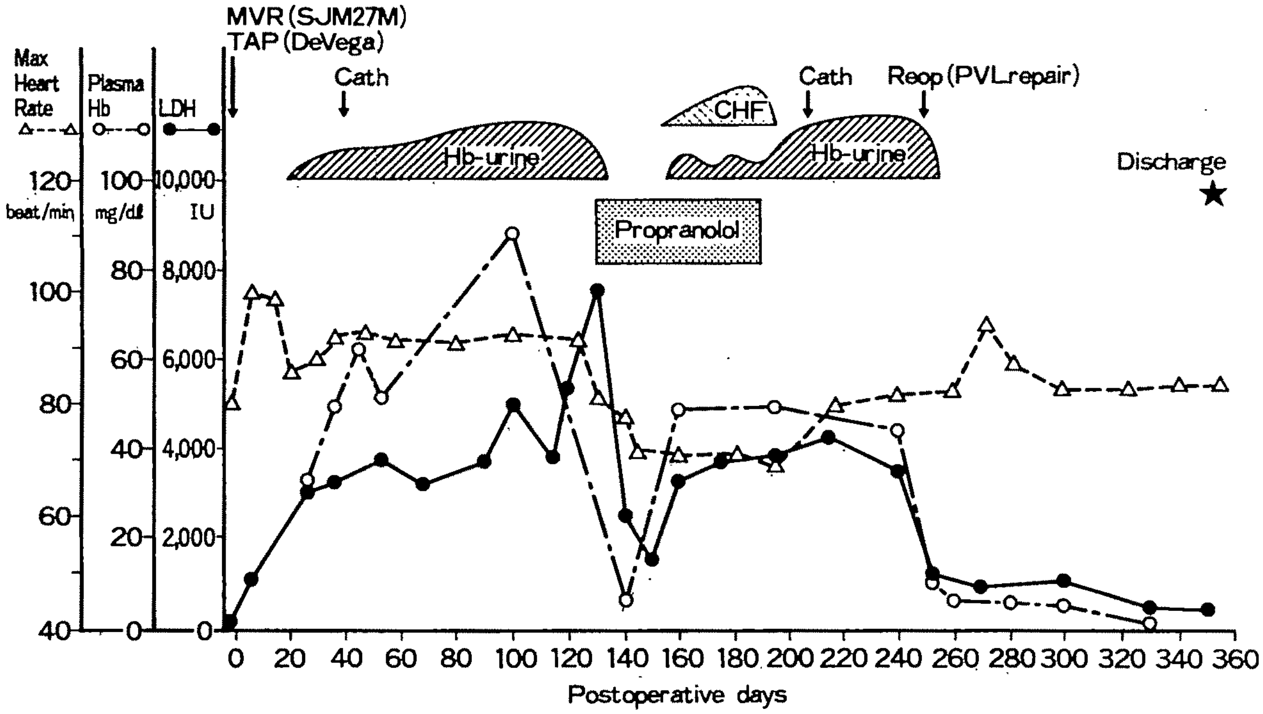

Fig 1. Clinical course of patient 1, a 58-year-old woman with mitral stenosis and minor regurgitation, tricuspid regurgitation, and minor aortic regurgitation who had undergone open mitral commissurotomy. (Cath = *catheterization;* CHF = *congestive heart failure;* Hb = *hemoglobin;* LDH = *lactate dehydrogenase;* Max = *maximum;* MVR = *mitral valve replacement;* PVL = *paravalvular leakage;* SJM = *St. Jude Medical;* TAP = *tricuspid annuloplasty.*)

Although postoperative hemodynamics were satisfactory except for tachycardia, a harsh pansystolic murmur was heard, and his urine became dark red on the day of operation. The lactate dehydrogenase level was 3,105 IU and the plasma hemoglobin level was 1,549 mg/L. Postoperative Doppler echocardiography revealed no residual shunt and a pressure gradient of 15 mm Hg in the right ventricular outflow tract across the ventricular septal defect patch. There was no pressure gradient in the left ventricular outflow tract. Propranolol administration was started at 30 mg (2.7 mg/kg) daily, and his urine became clear on the following day. Thereafter his recovery was progressive. Propranolol administration was discontinued on the 16th postoperative day, and he was discharged on the 23rd day. His clinical course is shown in Figure 2.

Comment

Mechanical destruction of erythrocytes by intracardiac foreign bodies after cardiac operations has been a well-known phenomenon since Sayed and associates [1] reported that Teflon felt could cause hemolysis after correction of an endocardial cushion defect. The pathogenesis of this mechanical hemolysis, as reported by Rubinson and colleagues [2] and Crexells and co-workers [3], is that the shearing stress generated between the foreign body surface and the erythrocyte destroys the erythrocyte membrane. Nevaril and associates [4] demonstrated experimentally that a shearing stress of more than 3,000 dynes/cm^2 increases hemolysis tremendously. Using Bernoulli's equation, stenosis with a pressure gradient of 50 mm Hg can generate a shearing stress of 4,000 dynes/cm^2. According to Schlichting [5], shearing stress is directly proportional to the square of the velocity of blood passing through the stenosed area. Consequently, paravalvular leakage after artificial valve replacement has a great potential for causing hemolysis. As reported by Rogers and Sabiston [6], mechanical hemolysis associated with paravalvular leakage has occurred predominantly in aortic valve replacement. We [7] reported serious hemolytic anemia caused by paravalvular leakage in mitral valve replacement using St. Jude Medical prostheses.

In a subarterial ventricular septal defect, the noncoronary and right cusps are deviated anteriorly due to conal malposition. Prosthetic patch closure of a large ventricular septal defect in the subarterial position inevitably causes infundibular stenosis [8]. In our patient, both the pressure gradient across the patch and the surface of the Dacron patch were considered to generate a greater shearing stress on the erythrocytes. Anzai and associates [9] reported a similar experience.

Theoretically, propranolol can reduce the velocity and quantity of blood flow and slow the intravascular mechanical hemolysis. Ishii and associates [10] reported a patient with mechanical hemolysis who underwent mitral valve replacement. In their patient, the grade of hemolysis fluctuated according to the heart rate. We postulated that propranolol could alleviate the mechanical hemolysis in a such a patient. Unfortunately, the first patient could not tolerate the propranolol trial because of congestive heart

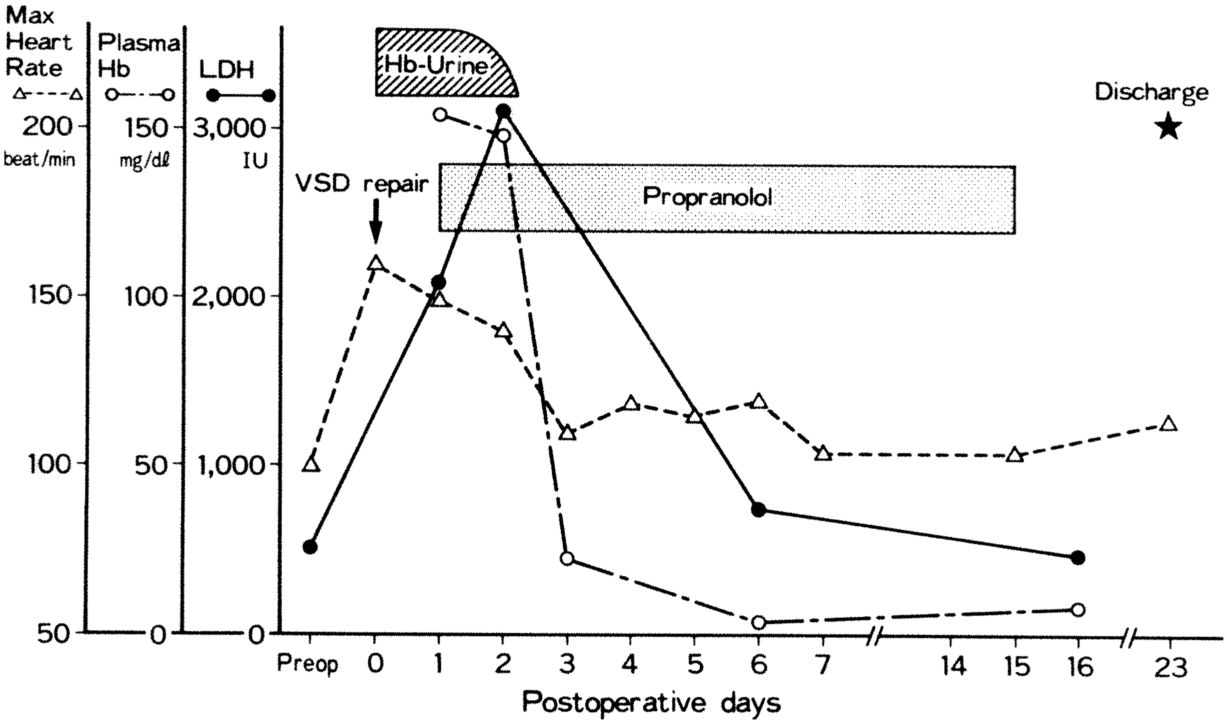

Fig 2. Clinical course of patient 2, a 2-year-old boy with a ventricular septal defect who had undergone subclavian flap repair and pulmonary arterial banding. (Preop = *preoperation; other abbreviations are listed in Figure 1.)*

failure. However, the hemolysis did transiently respond to propranolol. Hemolysis that occurs after closure of a subpulmonary ventricular septal defect, as in our second patient, usually spontaneously resolves within several weeks. Propranolol certainly reduced the duration and degree of the hemolysis. Although propranolol is no substitute for repair, we believe that propranolol is sometimes effective for intractable mechanical hemolysis after an open heart operation.

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Skin Grafting for Repair of a Cardiac Laceration

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Rupture of the right ventricle has been reported as a complication of closed catheter irrigation in poststernotomy mediastinitis. We report the case of a right ventricular rupture that was repaired with a deepithelialized dermal skin graft. The technique is described and management options for these difficult wounds are discussed.

(Ann Thorac Surg 1991;52:1161–3)

Sternal wound complications and mediastinal infections after median sternotomy are rare but devastating problems in cardiac surgery. Rupture of the right ventricle has been reported as a catastrophic event in this setting [1]. Right ventricular rupture may occur as a result of erosion of the infected sternum, ribs, or irrigation catheters through the ventricular free wall. A number of tissues including pericardium, pectoralis major muscle, and extrathoracic muscle transfers have been used to repair ventricular defects in the face of mediastinal infection. We present the case of a patient in whom acute mediastinitis developed after coronary revascularization with the internal mammary artery. A laceration of the right ventricle was repaired with a deepithelialized dermal skin graft. The patient recovered uneventfully and continues to do well 6 months after operation without infections or cardiac complications.

A 72-year-old man with a history of hypertension, diabetes mellitus, and multiple myeloma was seen at an outlying community hospital in January 1990 with an acute myocardial infarction. Postinfarction angina pectoris persisted, and he underwent cardiac catheterization, which revealed triple-vessel coronary disease. The patient's chest pain persisted, and on February 13, 1990, he underwent coronary artery bypass grafting with a left internal mammary artery graft to the left anterior descending artery as well as saphenous vein grafts to the right coronary artery and the diagonal branch of the left coronary artery. On postoperative day 7, a sternal wound infection developed that grew coagulase-negative staphylococci. The patient was treated with antibiotics and underwent wound debridement; however, the infection progressed to acute mediastinitis, which required reexploration and open povidone-iodine (Betadine; Purdue Frederick, Norwalk, CT) dressings. On postoperative day 21, a massive hemorrhage developed from bony erosion of the sternum into the right ventricle. This defect was repaired with a Dacron patch and the chest was closed. In spite of postoperative Betadine irrigation the wound dehisced due to persistent infection and the patient was transferred to Yale for definitive care.

The sternal wound was open, exposing a Dacron patch graft that was grossly purulent (Fig 1). The defect was approximately 10 × 6 cm. A partial thickness skin graft was harvested from the left thigh using the Padgett dermatome to remove the epidermis (set at 0.012 inches). The dermal graft was then excised manually. Cardiopulmonary bypass was initiated with normothermia by cannulation of the femoral artery and vein. The infected Dacron patch was dissected and excised creating a 10 × 6-cm defect in the anterior wall of the right ventricle. The deepithelialized dermal graft was then sutured circumferentially to close the defect with an interrupted, monofilament suture (Fig 2). The sternum and ribs were reapproximated after mobilization of the pectoralis major muscles and skin to cover the mediastinum. The mediastinum was irrigated with dilute Dakins' solution through a single irrigation and two drainage catheters for 7 days. His postoperative course was complicated by a right pleural effusion, which was drained by tube thoracostomy. He completed a 5-week course of antibiotics before his discharge on postoperative day 38. Follow-up echocardiography in the fifth postoperative month has demonstrated a slightly enlarged right ventricle with akinesis of the anterior and lateral ventricular walls but only trace tricuspid insufficiency. He continues to do well 6 months after operation.

Comment

The incidence of sternal wound complications and mediastinitis after median sternotomy is reportedly between 0.4% and 5% [2]. Several perioperative and postoperative factors have been implicated as predisposing to an increased incidence of postoperative mediastinitis. These include postoperative hemorrhage leading to mediastinal hematoma, operative time greater than 3 hours, postoperative resternotomy, previous sternotomy, low cardiac output syndromes, pneumonitis, and the presence of a tracheostomy [2, 3]. Mobilization of the internal mammary arteries for revascularization has also been associated with a higher incidence of infection in some series [4]; however, this has not been a universal observation. In general, most authors believe that several of these risk factors may be important in the eventual development of postoperative mediastinitis rather than a selected single factor.

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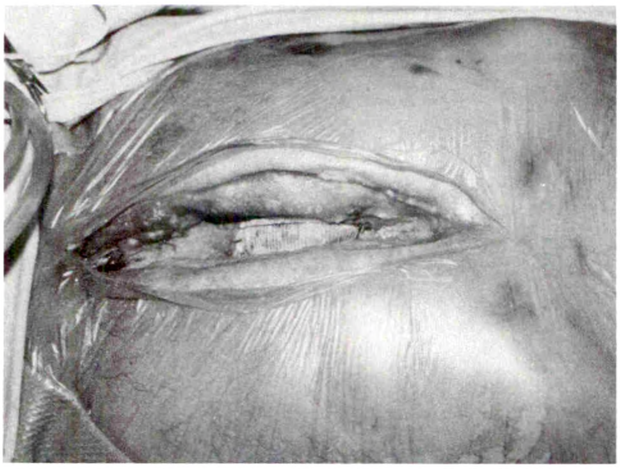

Fig 1. Open sternal wound with grossly purulent Dacron patch.

The successful treatment of mediastinitis requires immediate drainage with debridement of devitalized tissues and systemic antibiotics appropriate for the cultured organism. Historically, two approaches have been used by a number of investigators in large series [2, 3]. The "closed" technique involves surgical debridement and closed mediastinal irrigation with antimicrobial agents and tube drainage for 10 to 14 days [5, 6]. "Open" techniques require debridement and open packing of the mediastinum and sternal wound with secondary or delayed closure using muscle flaps. This method has been reserved for patients who fail closed techniques or those who have suppurative mediastinitis at the time of initial presentation. More recently, Nahai and associates [7] have recommended a more aggressive approach to the management of infected sternotomy wounds, citing persistent infection and increased mortality in those patients who undergo catheter irrigation. They reported improved results with *primary* debridement and muscle flap closure, which led to a fourfold decrease in mortality and increased success in their experience.

Piwnica and co-workers [1] reported additional problems with catheter drainage techniques. In their experience, rupture of the right ventricle developed in 6 patients with postoperative mediastinitis. Two patients had rupture during revisions of closed drainage systems and subsequently died. Four additional patients had rupture while coughing. Three of these patients went on to survive with successful repair of the defects by direct suture techniques or pericardial patch with pectoral muscle flap for tissue coverage.

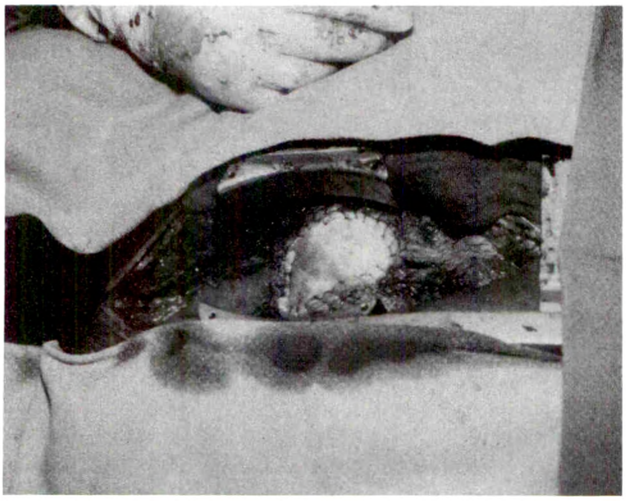

Fig 2. Operative view of deepithelialized dermal graft.

In our case, initial repair of the cardiac rupture with a Dacron patch was a life-saving measure. In general, the presence of prosthetic materials in patients with purulent mediastinitis has required removal of the material with replacement by autogenous tissue. Some rare cases of prosthetic graft salvage have been reported with closed povidine-iodine irrigation [8, 9]. These prosthetic patches often become infected and commonly disrupt along suture lines, leading to hemorrhage or pseudoaneurysm formation. Orringer and colleagues [10] reported their experience in this situation in which they replaced an infected Dacron patch with fascia lata in a child. Cohen and associates [11] recently reported the repair of a full-thickness right ventricular erosion using fascia lata buttressed by a rectus abdominis flap.

A number of authors have accrued considerable experience with infected median sternotomy wounds that were managed with muscle transposition, reducing the risk of prosthetic infection. In one of the largest series, 211 patients underwent debridement and primary wound closure with muscle or omental flap transposition [7]. Reconstruction was completed with pectoralis major, rectus abdominis, latissimus dorsi muscle, or omentum. The authors of that study emphasize that the blood supply to the sternum and the medial pectoralis major and rectus abdominis muscles is based on the thoracoacromial artery and the internal mammary artery. They caution that if the internal mammary artery is not intact other options may be required such as a contralateral or "segmental" pectoralis muscle flap.

Clinical success of vascularized muscle transplants to *directly* repair cardiac defects may be limited by the inherent thrombogenicity of muscle exposed to the blood elements. Autogenous tissue such as fascia lata, pericardium, or dermal skin grafts allows the repair to be performed with tissue that provides a smooth endocardial surface.

Our technique requires preoperative selection of a graft site adequate to cover the defect. With the dermatome set for maximum width, a flap of epidermis 0.012 to 0.015 inches in thickness is raised. The dermal skin graft is then manually excised to provide the appropriate graft size. This graft may require debridement of subcutaneous fat on the underside. The epidermal flap can then be replaced on the donor site and sutured in place or the graft site can be close primarily with proper mobilization. The graft is then sutured to the cardiac defect with the deepithelialized side away from the cardiac chamber. The major advantage of a dermal skin graft is that it allows a large quantity of autogenous tissue to be harvested for adequate coverage. When the graft is transplanted it receives blood supply from all surfaces within 3 to 5 days. Some authors have concluded from their experience that the graft remains unchanged as collagen and elastic fibers, which resist infection [12]. There may be some epidermal elements remaining in the graft in spite of deepithelialization. Theoretically, these epidermal elements could lead to the formation of an epidermal inclusion cyst. This has not been apparent during the early follow-up period in our patient. The graft is also a noncontractile tissue, as demonstrated by our postoperative echocardiogram. It is unlikely that this will have any long-term clinical significance in the right ventricular free wall with normal tricuspid valve function. Careful postoperative follow-up including biyearly echocardiography will be necessary to assess the long-term integrity of the graft.

In summary, we present the successful repair of a right ventricular laceration in a patient with mediastinitis using a full-thickness dermal skin graft. This autogenous tissue graft provided ample tissue for direct cardiac repair in the face of mediastinal infection. Deepithelialized dermal skin grafts may be an important alternative for the repair of cardiac defects in patients with mediastinitis.

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Partial Anomalous Pulmonary Venous Connection to the Azygos Vein With Atrial Septal Defect

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A patient undergoing successful repair of partial anomalous pulmonary venous connection to the azygos vein associated with a secundum atrial septal defect is presented.

(Ann Thorac Surg 1991;52:1164–5)

Partial anomalous pulmonary venous connection (PAPVC) is frequently associated with a sinus venosus type of atrial septal defect and right upper lobe pulmonary venous drainage to the superior vena cava [1]. Three patients with PAPVC to the azygos vein with an intact atrial septum have been reported [2–4]. We report here a patient with PAPVC to the azygos vein and an associated atrial septal defect who was managed successfully by complete surgical correction.

A 16-year-old girl was referred for evaluation of increasing fatigue and shortness of breath on exertion. The child had previously been well but had been noted to have a heart murmur in infancy. Physical examination revealed a grade 2/6 systolic ejection murmur heard maximally at the upper left sternal border, and loud fixed splitting of the second heart sound. A chest roentgenogram (Fig 1) showed moderate cardiomegaly, mild pulmonary plethora, and a prominent sharply circumscribed density at the level of the right tracheobronchial bifurcation. Two-dimensional echocardiography outlined a 17-mm secundum-type atrial septal defect with left to right shunting, right ventricular enlargement, and PAPVC near the superior vena caval–right atrial junction. A transesophageal echocardiogram confirmed these findings. Selective pulmonary arterial angiograms demonstrated the right superior pulmonary vein entering the azygos vein. There was then flow of contrast material into an enlarged right superior vena cava and right atrium (Fig 2A). The left pulmonary veins entered the left atrium (Fig 2B), as did the right inferior pulmonary vein.

She underwent operation on July 18, 1990. Through a median sternotomy incision, cardiopulmonary bypass was established using bicaval cannulation. The heart was arrested with warm induction blood cardioplegia followed by myocardial cooling to 15°C, and moderate systemic hypothermia was employed. The atrial septal defect was enlarged by incising the superior rim of the septum as far as the foramen ovale, and autologous pericardium was employed to baffle the flow from the enlarged azygos vein to the atrial septal defect. The right pleural cavity was opened and the azygos vein ligated inferior to the right superior pulmonary venous connection. The supreme intercostal vein draining into the azygos vein was also ligated. After repair, venous oxygen saturations were similar in the superior vena cava cephalad to the azygos vein, right atrium, and pulmonary artery.

The postoperative course was uncomplicated. A perfusion lung scan performed 1 week postoperatively showed 71% of the flow going to the left lung and 29% to the right. Cardiac catherization was repeated and showed unobstructed flow from the right superior pulmonary vein through the azygos and baffle and into the left atrium. Flow through the superior vena cava was also unobstructed. Follow-up 6 months postoperatively revealed she had remained asymptomatic and she was not taking any medications.

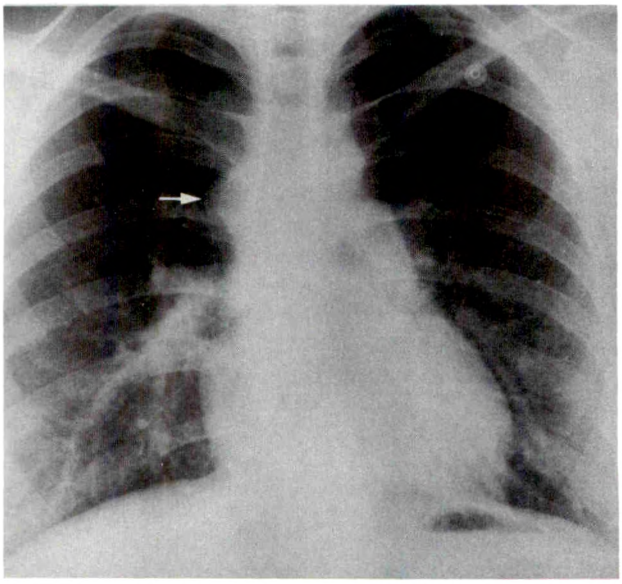

Fig 1. Chest roentgenogram showing moderate cardiac enlargement, increased pulmonary vascularity, and prominence of the azygos vein (arrow).

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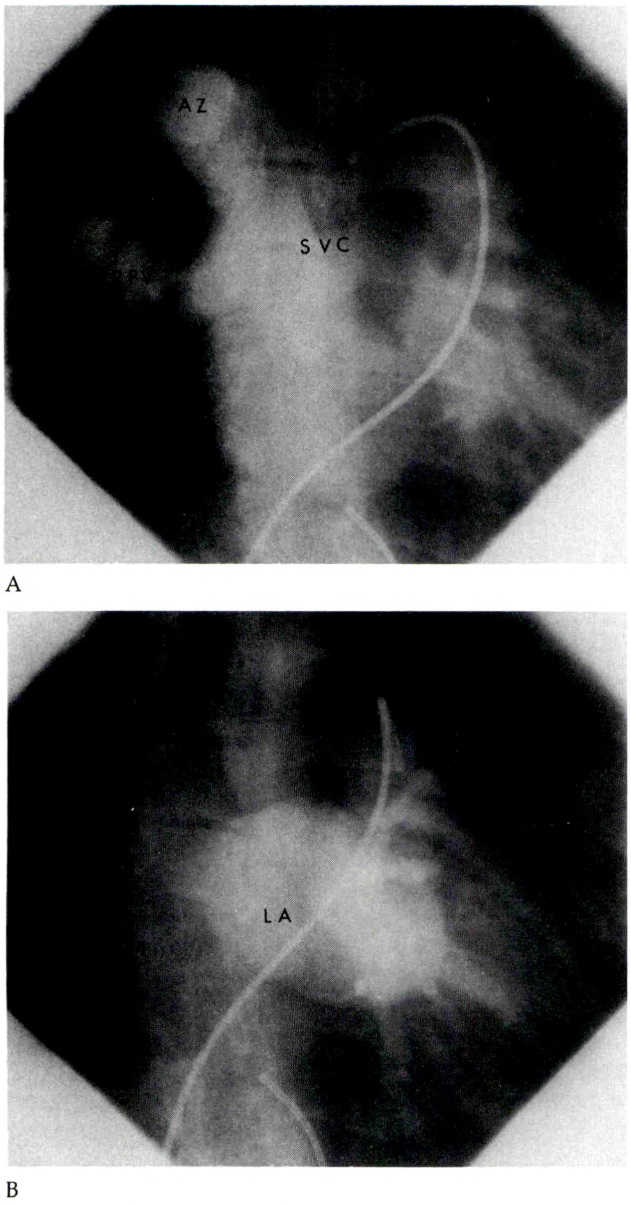

Fig 2. Selective cardiac angiograms (levophase) with catheter in the right (A) and left (B) pulmonary arteries. The right superior pulmonary vein (RPV) *drains superiorly into the azygos vein* (AZ) *and then inferiorly into the superior vena cava* (SVC)*. The normal left pulmonary veins are seen entering the left atrium* (LA)*.*

Comment

Partial anomalous pulmonary venous connection to the azygos vein is uncommon. Three cases of PAPVC and intact atrial septum have previously been reported [2–4], and in 2 of these surgical repair was successfully performed [2, 3]. Cases exhibiting bilateral total anomalous pulmonary connection have previously been described [5]. The present case is unusual, having unilateral partial pulmonary venous connection to the azygos vein plus an atrial septal defect.

The diagnosis of PAPVC to the azygos vein is difficult to make by transthoracic or transesophageal echocardiography. The atypical appearance of the PAPVC in the region of the superior vena caval–right atrial junction prompted selective cardiac catheterization, which was critical in making an accurate diagnosis.

Surgical correction was similar to the procedure used in patients with PAPVC to the superior vena cava [6, 7]. The atrial septal defect was first enlarged and then a pericardial baffle was constructed to tunnel the blood flow from the azygos vein to the left atrium. The other systemic venous tributaries of the enlarged azygos vein were divided to prevent a subsequent right to left shunt and possible "paradoxical" emboli. In the 2 previous surgically corrected cases [2, 3] this was not done, but difficulties from the resultant small shunt were not reported.

Partial anomalous pulmonary venous connection to the azygos vein requires an index of suspicion and cardiac catheterization to diagnose. The condition may or may not be associated with an atrial septal defect. Complete repair is feasible and can be accomplished safely.

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Cogan's Syndrome With Aortitis, Aortic Regurgitation, and Aortic Arch Vessel Stenoses

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Cogan's syndrome of interstitial keratitis and vestibuloauditory dysfunction is rare. Systemic vasculitic manifestations occur, and 10% of patients with this syndrome have aortic valvular disease. A patient with Cogan's syndrome is presented who had aortitis of the ascending thoracic aorta, severe aortic valve regurgitation, orificial stenosis of the right coronary artery, and orificial stenoses of all three aortic arch vessels. Histopathology confirmed aortitis. Aortic valve replacement combined with coronary and aortic arch vessel reconstruction was required for correction.

(Ann Thorac Surg 1991;52:1166–7)

In 1945 Cogan [1] reported nonsyphilitic interstitial keratitis with vestibuloauditory symptoms, progressing to deafness. Subsequently Cogan's syndrome has been reported in association with vaccination, sarcoidosis, vasculitis, and polyarteritis nodosa [2]. Response to steroids has been variable. Vascular involvement has affected large vessels of the extremities and viscera [3, 4], the aortic wall, and aortic valve leaflets, resulting in aortitis, aortic regurgitation, cardiac failure [5], and requirement for aortic valve operation [6].

A 35-year-old man with prior asthma and thyrotoxicosis became ill 10 years before this report. He experienced dizziness, vomiting, red painful eyes, and bilateral sensorineural deafness. There was normal middle ear, cerebellar, and cranial nerve function, except for the deafness related to the eighth nerve. Blood examination, erythrocyte sedimentation rate, temporal bone roentgenograms, and electrolytes were all normal. Full syphilis serology was negative. A diagnosis of Cogan's syndrome was made. Treatment with corticosteroids was unhelpful.

Two years later, his erythrocyte sedimentation rate was 69 mm/h, and an immunological work-up revealed lymphocytosis, marked rouleaux, raised serum immunoglobulin level, and positive antinuclear and smooth muscle antibody titers. An asymptomatic aortic systolic murmur was now present.

Five years later exertional dyspnea developed. Examination revealed a slim, deaf man with bilateral keratitis but normal visual acuity. Pulse was regular, but the blood pressure was different in the arms, 130/40 mm Hg on the left and 90/70 mm Hg on the right. The systolic blood pressure in the legs was 160 mm Hg. Both carotid arteries were of small volume with palpable thrills and bilateral bruits, extending on the right to the shoulder. There was a loud cardiac systolic murmur and a loud, long diastolic murmur of aortic regurgitation. Electrocardiography showed mild left ventricular hypertrophy. Chest roentgenogram showed a normal-sized heart and aorta and normal lungs. Doppler echocardiography showed severe aortic regurgitation.

Cardiac catheterization and aortography revealed an aortic valve gradient of 20 mm Hg, severe aortic regurgitation, mild left ventricular enlargement, and a 95% ostial stenosis of the right coronary artery. The ascending aorta appeared rigid and narrowed proximally; distally it was relatively dilated. There was an 80% orificial stenosis of the innominate artery, a 90% orificial stenosis of the left common carotid artery, and a 50% proximal stenosis of the left subclavian artery. The carotid arteries in the neck were normal (Fig 1).

At operation, the ascending aorta was of normal external diameter, but in the proximal 2 cm, the wall was thick (5 mm) and rigid. The aortic valve leaflets were thickened and retracted, the annulus was small, and the right coronary orifice was pinpoint in size. The innominate artery stenosis was relieved by an 11-mm woven Dacron tube graft from the aorta to the origin of the right common carotid and subclavian arteries. The graft was first preclotted with 25% albumin and autoclaved. The stump of the innominate artery was oversewn.

After commencement of bypass by right atrial and right femoral cannulation, the aorta was clamped below the new Dacron graft. Cold crystalloid potassium cardioplegia, given through the left coronary orifice, and topical cooling were used for cardiac protection. Saphenous vein bypass was performed to the right coronary artery, which was normal distally. The valve was replaced with a 19-mm St. Jude Medical mechanical valve prosthesis (St. Jude Medical Inc, St. Paul, MN). After the aortotomy was closed and the aortic cross-clamp was removed, there was insufficient room on the ascending aorta to place a side clamp, and hence the proximal end of the vein graft was attached to the side of the aorta–innominate Dacron graft. His postoperative course was uneventful, without neurologic sequelae. He was anticoagulated with warfarin sodium.

Histopathology of the innominate artery wall showed vascularized fibromyxoid connective tissue with a chronic

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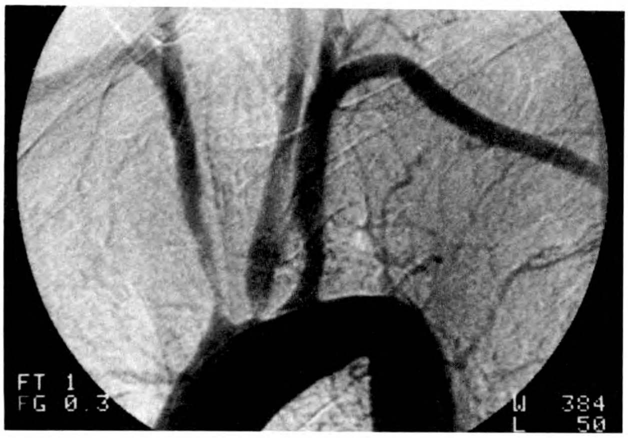

Fig 1. Digital subtraction angiogram of the aortic arch showing 80% orifice stenosis of the innominate artery, 90% orifice stenosis of the left common carotid artery, and 50% proximal stenosis of the left subclavian artery.

inflammatory infiltrate. The aortic wall section showed marked intimal inflammation, fibrosis, and thickening. The valve cusps showed thickening of the free edge, with extensive myxoid degeneration, but no evidence of acute inflammation.

Three years postoperatively he remains well, active, and at work. Examination revealed a soft systolic murmur, and the blood pressure difference between the arms was reversed. Repeat angiography demonstrated patency of the saphenous vein and Dacron grafts.

Comment

Cogan's syndrome is rare. The ocular symptoms can be variable, but visual loss is unusual. In contrast, progression to complete deafness is common, with little or no response to corticosteroids.

An underlying vasculitis is likely in all patients with Cogan's syndrome, although it may be variable in severity and extent. The histology in this patient seems similar to that previously described in patients with aortitis and aortic regurgitation who underwent valve operations [5, 6]. The aortic tissue showed marked inflammatory reaction, both acute and chronic, foci of fibrinoid necrosis, giant cell formation, neovascularization of the intima, and arteriolar changes of endothelial proliferation, medial hypertrophy, and perivascular fibrosis. However, there is less acute inflammatory component in the present case.

Heart disease is found in 15% to 25% of patients with Cogan's syndrome [7, 8], predominantly manifesting as aortic regurgitation and often progressing to severe aortic regurgitation and heart failure over a number of weeks or months. The median time from the onset of the illness to diagnosis of aortic regurgitation was 29 months.

This patient's aortic involvement also resulted in orificial stenoses of the right coronary artery and all arch vessels, least severe in the left subclavian artery. Although coronary orificial stenosis has been described [6], involvement of the aortic arch vessels is extremely rare and not clearly documented.

The surgical approach to the problem warrants comment. The patient was asymptomatic from these stenosing lesions, and the operative risk and the risk of stroke would have been high from complete arch reconstruction. A less aggressive course was taken to restore normal flow in the innominate artery on the presumption that if the left common carotid artery did occlude at a later time, collateral flow through the now "normal" right side would be satisfactory, or that it would be separately reconstructed later if symptoms pertaining to it evolved.

The small aortic valve annulus, which would accept only a 19-mm St. Jude Medical valve, raises the question about the adequacy of a small prosthesis and the size of any residual gradient. As the patient weighed only 58 kg and had a small surface area of 1.7 m^2, it was believed that the hemodynamic performance of the low-profile bileaflet valve would allow reasonable cardiac output at moderate workloads. The presence of the abnormal, thickened, rigid aortic wall, affected by "vasculitis," would have made aortic root enlargement by transannular patch difficult and dangerous. In fact, the patient's activity has not been limited by the size of the valve prosthesis.

Reversed saphenous vein was used for the right coronary artery bypass graft because of the known disease affecting the innominate and right subclavian arteries, because of the unknown quality of the right internal mammary artery, and to simplify the operation.

Patients with aortic regurgitation in association with Cogan's syndrome who have not undergone a corrective operation (aortic valve replacement) have usually died within a few months of the detection of the murmur [7]. Hence early operation to correct aortic regurgitation and associated severe ostial coronary and aortic arch vessel lesions is advocated.

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Successful Management of Aortoesophageal Fistula Due to Thoracic Aortic Aneurysm

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Aortoesophageal fistulas due to atherosclerotic thoracic aneurysms are usually fatal, with few reported survivors. We report an aortoesophageal fistula managed successfully in one stage by resection and replacement of the aortic aneurysm with a prosthetic graft and total esophageal resection. Immediate esophageal reconstruction was attained using orthotopic gastric interposition with omentopexy around the prosthetic aortic graft.

(Ann Thorac Surg 1991;52:1168–70)

Thoracic aortic aneurysms may, by extrinsic compression of the esophagus, cause ischemic necrosis of the esophageal wall, which eventually results in an aortoesophageal fistula. Rupture into the esophagus is a terminal event in up to 27% of atherosclerotic and dissecting aortic aneurysms [1, 2]. Patients with aortoesophageal fistulas are seen with midthoracic pain, sentinel arterial hemorrhage, and finally exsanguination after a symptom-free interval [2].

There are few reports of successful surgical management of aortoesophageal fistulas caused by atherosclerotic aneurysms [2]. Patients dying after operation invariably have ongoing mediastinal sepsis [1].

A 67-year-old man with multiple myeloma in remission, was admitted with a 3-week history of dysphagia, central chest pain, and hematemesis. A chest radiograph showed a posterior mediastinal mass. Barium study of the esophagus demonstrated displacement of the esophagus by the mass and extravasation of contrast medium into the posterior mediastinum (Fig 1A). An aortogram showed a fusiform descending thoracic aortic aneurysm with an additional separate false aneurysm in the mediastinum (Fig 1B). Aortography did not demonstrate active bleeding or a fistulous connection with the esophagus.

The atherosclerotic true aortic aneurysm was resected 8 hours later and replaced with a 22-mm Dacron prosthesis during an ischemic cross-clamp time of 30 minutes. Examination of an intraoperative swab taken from the false aneurysm revealed pus cells and gram-positive organisms (bacteriologic culture later demonstrated *Streptococcus salivarius*). The infected false aneurysm, residual aneurysmal wall, and surrounding mediastinal inflammatory tissue were resected. Both end-to-end aortic anastomoses were performed as far distally and proximally as possible, and the graft was made purposefully long to position it away from the potentially infected area. The esophageal wall surrounding the 3 × 1-cm perforation was soft, necrotic, and unsuitable for any reparative procedure. A total esophagectomy was performed, and the stomach as well as the greater omentum was mobilized and delivered through the esophageal hiatus into the chest. A cervical gastroesophageal anastomosis was done, and the omentum was wrapped around the vascular prosthesis (Fig 2).

This patient was ventilated postoperatively, extubated the following day, and maintained on intravenous antibiotics for 12 days. Recovery was uneventful, and 2 weeks after operation he was discharged. He remains well 14 months after operation with no evidence of sepsis.

Comment

Aortoesophageal fistulas can occur as a result of thoracic aortic aneurysms [2], esophageal foreign bodies [3], reflux esophagitis with peptic ulceration [4], tuberculosis [5], carcinoma of the esophagus, or aortic operation [6]. An aortoesophageal fistula can be difficult to diagnose as aortography only confirms the presence of an aortic aneurysm and rarely demonstrates the fistula. An esophagogram is more likely to show a fistula but may also only show ulceration. Alternatively, endoscopic features are characteristic; however, catastrophic hemorrhage can be precipitated. There are few reported survivors of aortoesophageal fistulas [3, 4, 6], and only 2 patients have survived atherosclerotic aneurysm-induced fistulas [2]. To ensure a successful outcome, the planned surgical procedure should follow established principles for managing an aortic aneurysm, an esophageal perforation, and a potentially infected mediastinum.

If the aortoesophageal fistula is a result of an aortic aneurysm, the aneurysm must be resected and replaced with a prosthetic graft. However, anastomoses should be placed as far from the potentially infected field as possible, and consideration should be given to extraanatomical bypass. In contrast, primary repair of the aorta has only been successful if the fistula was caused by a foreign body [3] or reflux esophagitis [4].

Perforation of the esophagus is associated with substantial morbidity and mortality, regardless of cause [7]. An aortoesophageal fistula due to a localized perforation, such as that from a foreign body, has been managed by

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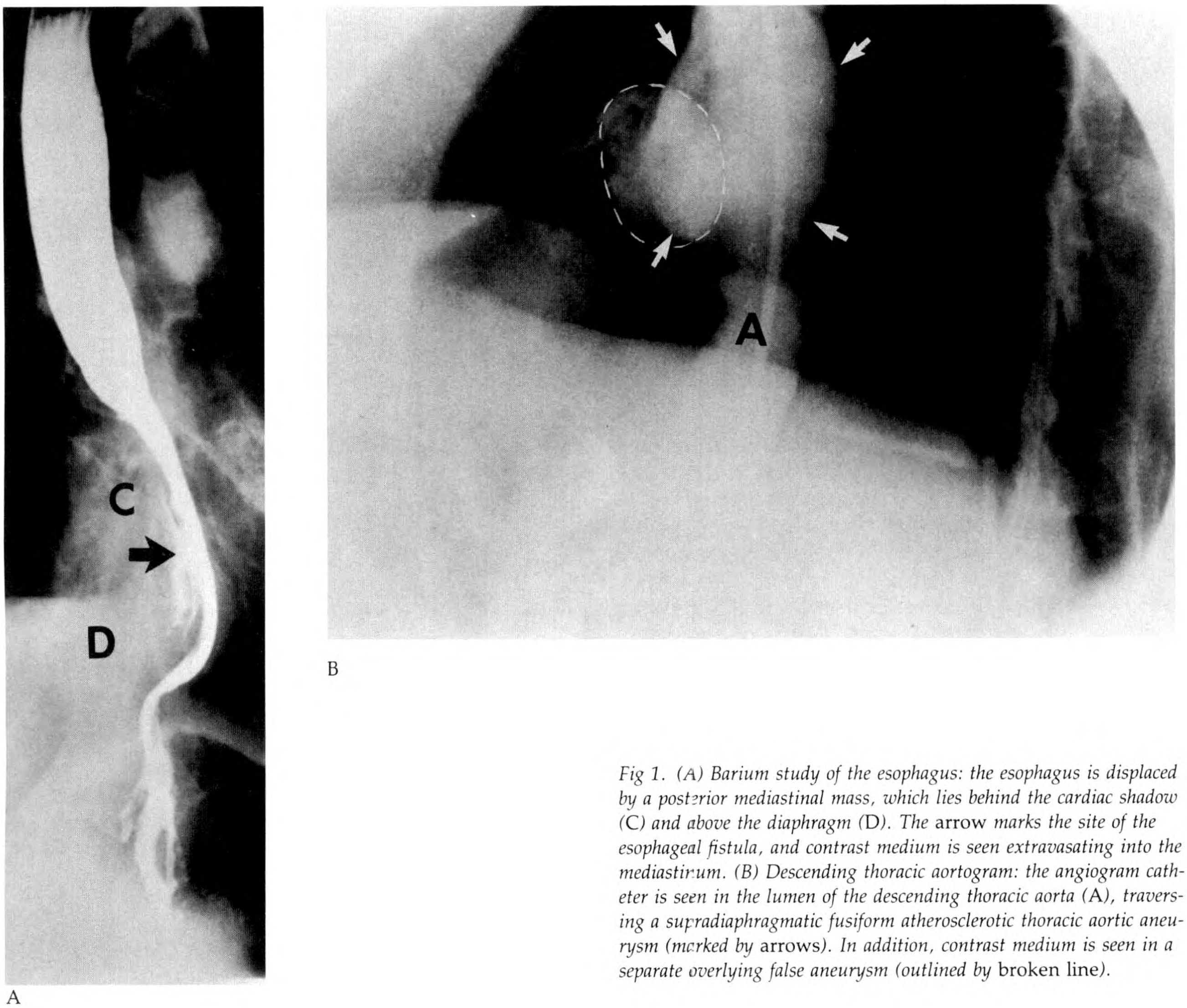

Fig 1. (A) Barium study of the esophagus: the esophagus is displaced by a posterior mediastinal mass, which lies behind the cardiac shadow (C) *and above the diaphragm* (D). *The* arrow *marks the site of the esophageal fistula, and contrast medium is seen extravasating into the mediastinum. (B) Descending thoracic aortogram: the angiogram catheter is seen in the lumen of the descending thoracic aorta* (A), *traversing a supradiaphragmatic fusiform atherosclerotic thoracic aortic aneurysm (marked by* arrows). *In addition, contrast medium is seen in a separate overlying false aneurysm (outlined by* broken line).

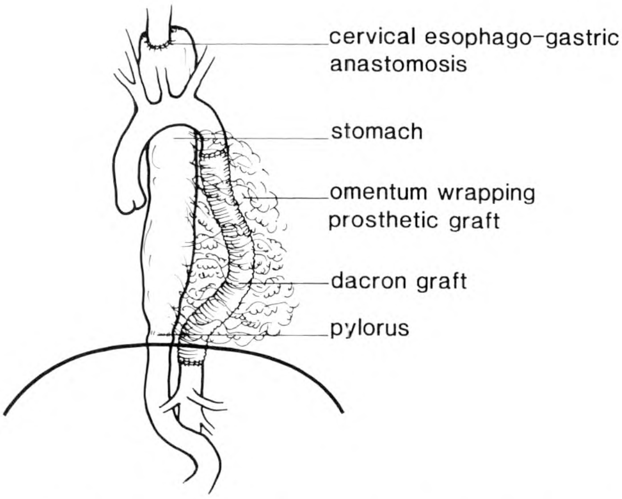
cervical esophago-gastric anastomosis
stomach
omentum wrapping prosthetic graft
dacron graft
pylorus

Fig 2. Surgical procedure: total esophagectomy and resection of true and false aneurysms have been performed. Reconstruction was by orthotopic gastric interposition. In addition, the omentum was left attached to the stomach and wrapped around the prosthetic aortic graft.

primary suture repair of the esophagus [3]. However, all other attempts at primary esophageal repair of aortoesophageal fistulas have been complicated by either dehiscence of the repair, reoperation, prolonged hospitalization, or death [2, 6]. In contrast, our patient and the other previously reported patient managed by immediate esophagectomy [2] made uneventful, rapid postoperative recoveries. Therefore, we believe a distinction must be made between an ischemia-induced fistula due to pressure necrosis and that due to a very localized perforation from a foreign body. We would advise total esophagectomy in the former.

Furthermore, immediate reconstruction of the esophagus by orthotopic gastric interposition obliterates the large posterior mediastinal space produced by resection of the true and false aneurysms. Obliteration of potential spaces is an important principle in the management of intrathoracic infections. In addition, the omentum has been previously shown to be very effective in combating infection, and we therefore believe it should be transposed into the thorax with the stomach.

The rapid postoperative recovery of our patient is probably also related to other factors, such as not requiring massive preoperative blood transfusions. However, it would appear that early aggressive management of the associated esophageal perforation by esophagectomy is of paramount importance.

We thank our colleagues in the Departments of Anesthesiology, Cardiology, and Haematology for their expert assistance.

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Blunt Traumatic Rupture of Pulmonary Vein, Left Atrium, and Bronchus

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A 22-year-old man fell 12.2 m (40 ft), injuring the right lower lobe bronchus, right inferior pulmonary vein, and left atrium. These injuries were not associated with fractures, cardiac tamponade, or pneumothorax. The severity of injury became apparent only upon right thoracotomy for persistent hemorrhage. This case presented special diagnostic and surgical challenges and suggests a role for the increased use of bronchoscopy in major blunt chest trauma.

(Ann Thorac Surg 1991;52:1171–2)

Injury to the heart, great vessels, and tracheobronchial structures from blunt trauma may not be suspected clinically but may occur in 10% to 15% of fatal accidents [1, 2]. Concomitant injuries and lack of clinical signs may obscure the diagnosis. We report this challenging case of a tear in the right inferior pulmonary vein and left atrium combined with transection of the right lower lobe bronchus.

A 22-year-old man was found pulseless and unconscious after falling from an overpass. MAST trousers were placed and resuscitation commenced at the scene. On arrival in the emergency room he responded to deep pain on the left side only. Deep tendon reflexes were absent. Pupils were dilated but reactive. Breath sounds were decreased on the right, with no evidence of subcutaneous emphysema or cardiac tamponade. Chest roentgenography showed opacification of the right hemithorax without mediastinal widening, pneumothorax, or fractures. A chest tube was placed with no obvious air leak. Head computed tomographic scan showed no pathology.

Emergency right thoracotomy was performed for persistent chest hemorrhage. A double-lumen endotracheal tube was placed and bronchoscopy was attempted to confirm correct positioning, but visualization was limited. When the chest was entered, a large amount of blood was present in the mediastinum. The pericardium had a large rent. A 7-cm tear extending from the inferior pulmonary vein into the left atrium was discovered after temporary manual control of hemorrhage was achieved (Fig 1). Large sutures were placed to repair the tear. This compromised the inferior pulmonary vein, necessitating a right lower lobectomy. The right lower lobe bronchus had an unexpected, almost complete traumatic transection.

After the repairs repeat bronchoscopy did not demonstrate further bronchial injuries. Computed tomographic scan 1 week later disclosed a hemorrhagic occipital infarct. The patient remained comatose and ventilator dependent for 2 weeks. Within 2 months dramatic recovery of speech and motor activity enabled transfer to a rehabilitation center.

Comment

Bright and Beck [3] launched early investigation into effects of blunt trauma to the heart. In 1958 Parmely and associates [4] found myocardial rupture in 64% of 546 postmortem cases of nonpenetrating trauma to the heart, but it was clinically suspected only once. Successful operative repair of a right atrial tear was reported by Desforges and co-workers in 1955 [5]. Subsequent reports describe isolated chamber rupture [1, 4, 6–8], multiple chamber rupture [4, 6–8], chamber rupture with or without pericardial tears [3, 4, 7, 8], myocardial rupture associated with large airway injuries [2, 8], and those with injuries to great vessels [2]. Death from cardiac rupture occurs from exsanguination or tamponade.

The clinical presentation is profound hypotension, hypovolemia, and massive hemothorax. Tamponade physiology is not always a reliable clinical indicator of cardiac trauma as 30% of patients have associated pericardial tears [1]. Blunt injuries to the airway are seen most often as tracheobronchial disruptions. In one study the postmortem incidence of tracheobronchial injuries was 2.8% after blunt trauma [2]. Bronchial disruption when in communication with the pleural space can produce symptoms of dyspnea, hemoptysis, cyanosis, subcutaneous and mediastinal emphysema [2]. Pneumothorax or mediastinal air may not be present if the tear is not in communication with the pleura.

Motor vehicle accidents are the most frequent cause of blunt chest trauma. Severe injury to intrathoracic structures may occur in the absence of fractures and can be attributed to several mechanisms: (1) indirect or "hydraulic ram" force from the abdomen and lower extremities, which increases intrathoracic pressure and produces rupture without a direct blow to the chest; (2) bidirectional compression between the sternum and vertebral bodies; (3) acceleration/deceleration effect on the fixed structures in the chest; and (4) blast forces. Intrathoracic structures

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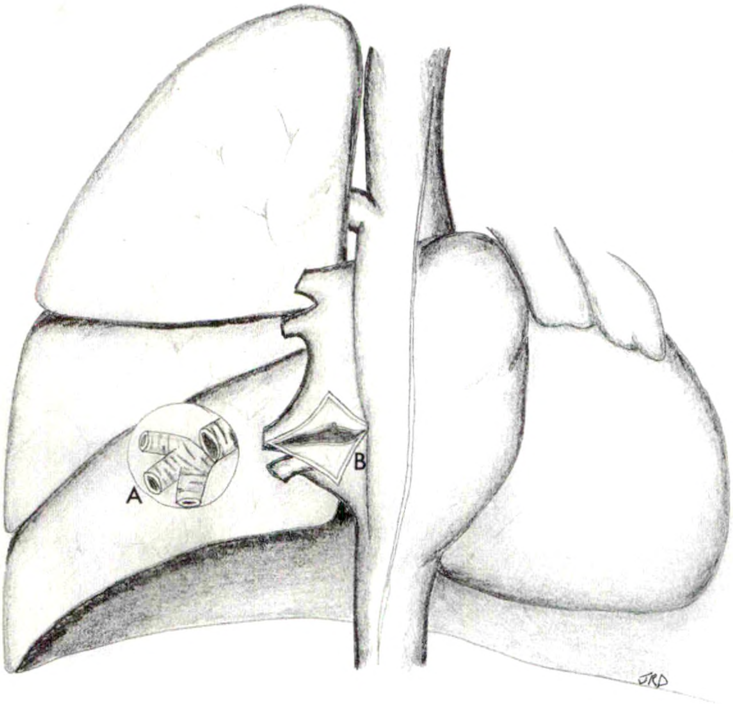

Fig 1. Almost complete transection of right lower lobe bronchus (A) *and a 7-cm tear extending from the inferior pulmonary vein into the left atrium* (B) *from blunt trauma.*

may be violated by a fractured rib or sternum [1–6, 8]; however, bony fractures of the thorax do not necessarily herald severe injuries. A subset of patients have serious intrathoracic injuries without rib fractures [2–4, 7, 8]. Energy not dissipated by ribs may target soft tissue rather than bone [7].

This case had several unusual features. Despite the height of the fall, bony injuries were absent and the initial head computed tomographic scan results were normal. Evidence of widened mediastinum or signs of tamponade were absent. Initially the right lung was poorly ventilated from the massive hemothorax. Reexpansion after drainage was confirmed by roentgenography. The bronchial tear was contained by the surrounding lung parenchyma. Lack of pneumothorax or air leak is peculiar with almost complete transection of the right lower lobe bronchus. This suggests the need for a more liberal and aggressive use of bronchoscopy in major blunt chest injuries. When the left atrial tear was discovered, suctioning of the surgical field was done cautiously to avoid air embolus. Surgical control of the extensive, rapidly bleeding site posed a major challenge as application of standard vascular clamps was impractical. Insertion of a Foley catheter with a large enough balloon to control the bleeding would have impeded venous return and filling of the left ventricle. The initial attempt to repair the tear without jeopardizing the inferior pulmonary vein was unsuccessful. Control was achieved using number 2 polypropylene suture with a large needle, facilitating the repair with the field essentially obscured by blood. This technique decreased the risk of air embolism and provided quick control of the massive hemorrhage but compromised the inferior pulmonary vein. Transection of the bronchus was not suspected clinically or by gross inspection of the lung. Only when lower lobe fissures were dissected was the extent of injury realized.

In summary, in this patient cardiac rupture and tears of a pulmonary vein and bronchus were not clinically suspected as rib fractures, subcutaneous emphysema, pneumothorax, and physiologic signs of tamponade were absent. This case report stresses a heightened index of suspicion for critical intrathoracic injuries with blunt chest trauma and advocates the more liberal use of bronchoscopy in these patients.

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Massive Hemoptysis Due to Broncholithiasis

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Massive hemoptysis due to broncholithiasis is rare. Such a case is presented here, and the literature is reviewed. Surgical resection is the preferred definitive therapy, as a lack of bronchial artery collaterals limits the utility of bronchial artery embolization.

(Ann Thorac Surg 1991;52:1173–5)

Hemoptysis secondary to broncholithiasis is rarely massive [1]. It has been termed "innocent hemoptysis" because of its low volume and self-limiting nature [2]. We report here a case of massive hemoptysis due to broncholithiasis and review the literature.

An 81-year-old man was admitted with new onset of hemoptysis. There was no prior medical history. Approximately 800 mL of blood was expectorated during his first 24 hours in the hospital. Abnormalities were not noted on physical examination. The admitting chest roentgenogram was notable for a left hilar broncholith (Fig 1).

The referring physician promptly performed bronchoscopy on the patient. This confirmed that the left lung was the source of hemorrhage; however, exact localization was not possible because of the limited suction capacity of the flexible bronchoscope. Subsequent aortography not only failed to reveal a source of bleeding but was remarkable for a paucity of enlarged bronchial arteries.

At emergent exploration, dense adhesions and numerous calcified and matted lymph nodes were encountered. This tissue reaction prevented secure vascular isolation of the lobar pulmonary artery, necessitating a left pneumonectomy. The pathologic specimen revealed the broncholith to be in close proximity to several 4- to 5-mm pulmonary veins (Fig 2).

Postoperatively the patient did well, leaving the hospital 7 days later. The patient has had no further episodes of hemoptysis.

Comment

Massive hemoptysis due to broncholithiasis is rare. Eleven such cases have been reported in the literature (Table 1), with only 4 cases having been reported in the past 35 years.

Broncholithiasis is due to the late tissue response to healing of granulomatous infections, especially histoplasmosis and tuberculosis [6, 8]. Compression or distortion of adjacent structures caused by the broncholith's presence leads to pressure necrosis of the walls of bronchial and pulmonary vessels. The resulting fistula is the source of signs and symptoms in this disease.

The majority of patients with massive hemoptysis secondary to broncholithiasis have rarely had prior pulmonary symptoms. Chest roentgenography coupled with flexible bronchoscopy can localize the side of bleeding. Rigid bronchoscopy, however, has been recommended because of the greater suction capabilities, which allow localization of the bleeding source to a single lobe [4, 9]. As in all cases of massive hemoptysis, protection of the contralateral lung is essential [4, 6, 8].

Most patients with massive hemoptysis have an underlying active inflammatory disease that promotes the development of bronchial artery collaterals. Thus, hemoptysis in the vast majority of patients originates from systemic rather than pulmonary arteries [10]. Broncholithiasis, by its very nature, does not lead to such an inflammatory response. This accounts for the paucity of bronchial arteries at aortography. More likely, patients with massive hemoptysis secondary to broncholithiasis bleed from erosion of the broncholith into adjacent pulmonary vessels. We would suggest that in patients with massive hemoptysis due to broncholithiasis, if the chest roentgenogram lacks evidence for other inflammatory diseases, then aortography may be omitted.

In the management of massive hemoptysis due to broncholithiasis, surgical intervention should rapidly fol-

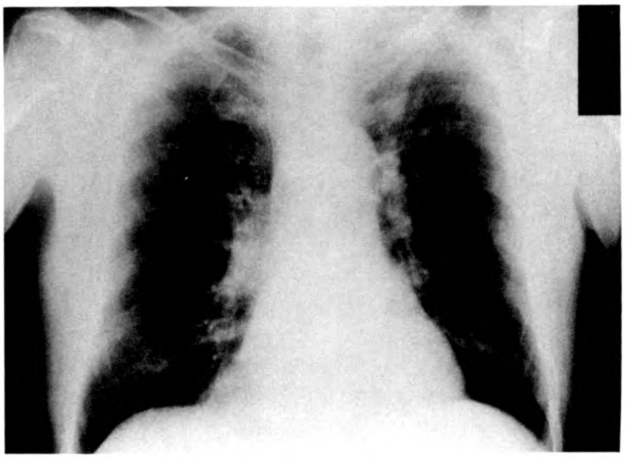

Fig 1. The patient's admitting chest roentgenogram: there is a 2 × 2-cm broncholith noted in the patient's left hilum. There is also a suggestion of segmental atelectasis extending superiorly from the broncholith to the periphery of the lung.

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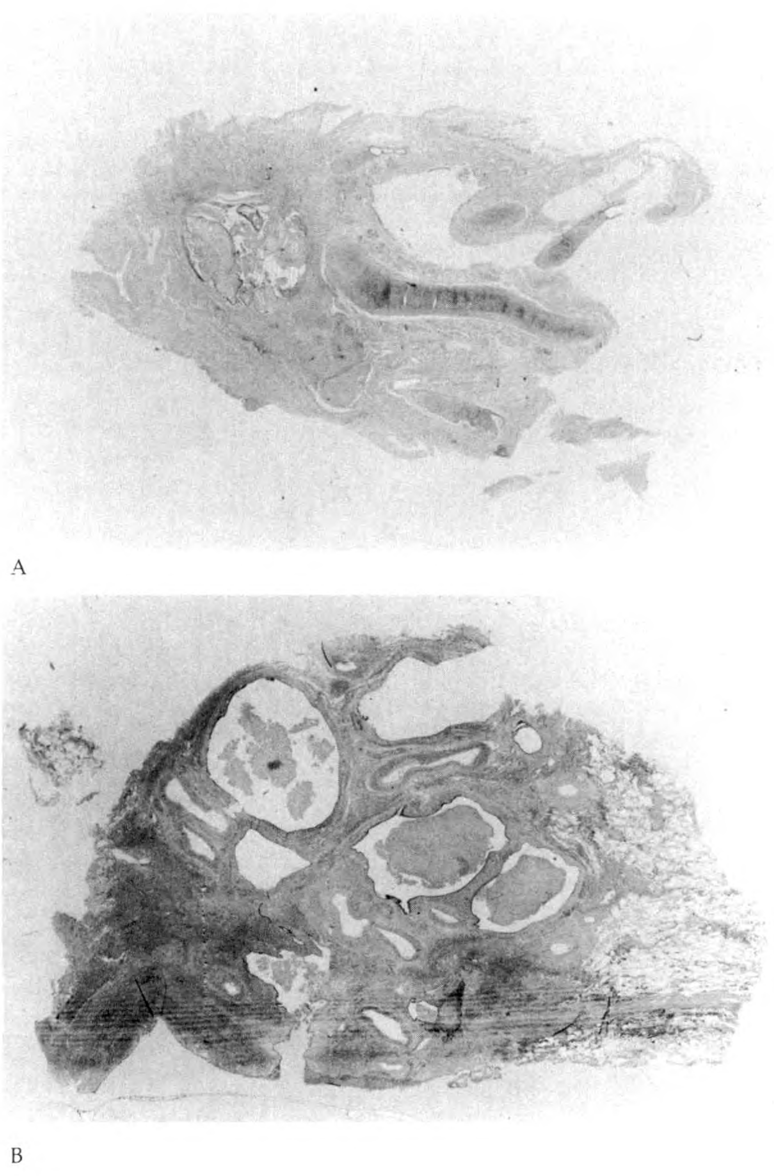
Fig 2. Representative microscopic sections: (A) The broncholith is demonstrated in close proximity to a bronchial lumen. (B) A histologic section near the area shown in (A) is reproduced. Note the large (4- to 5-mm) pulmonary veins. (Hematoxylin and eosin, ×10 before 38% reduction.)



low medical stabilization. Review of the literature demonstrates all medically managed patients died due to recurrent hemorrhage. At exploration the dense calcific hilar tissue reaction is more extensive than what might have been expected based on the preoperative chest roentgenogram alone [4]. Proximal control of the pulmonary artery has been recommended as a first maneuver to prevent fatal hemorrhage [4, 6]. Conservative resection should be the intraoperative goal. This can occasionally be accomplished by a local resection with or without a bronchoplastic procedure [4, 6, 8]. Adhesions generally make a lobectomy the most popular procedure [4]. Rarely, injury to the pulmonary artery necessitates a pneumonectomy [6].

In summary, broncholithiasis may result in massive hemoptysis. Patients with massive hemoptysis due to broncholithiasis are managed similarly to patients with massive hemoptysis from other causes. Surgical resection is the preferred method of definitive therapy. However, the value of bronchial artery embolization has to be questioned as bleeding is frequently from the lesser circulation rather than from systemic vessels.

Table 1. Massive Hemoptysis Due to Broncholithiasis

First Author	Year	No. of Cases	Management	Outcome
Pitkin [3]	1941	1	Pneumonectomy	. . .[a]
Ehrenhaft [4]	1955	6	5, lobectomy; 1, pneumonectomy	6 survivors
Bollengier [5]	1974	1	Medical	Death
Faber [6]	1975	1	Resection	. . .[a]
Lin [7]	1978	1	Medical	Death
McLean	Present Report	1	Pneumonectomy	Survived

[a] Insufficient data provided.

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INVITED COMMENTARY

A perusal of the literature yields many definitions for "significant" and "massive" hemoptysis. Certainly, the individual who expectorates 800 mL of bright red blood in a 24-hour period has massive hemoptysis. Although McLean and co-authors state that massive hemoptysis secondary to broncholithiasis is rare, it is certainly more frequently seen and perhaps less commonly reported by those who practice in endemic areas of histoplasmosis.

Plain chest roentgenograms showing characteristic hilar calcifications may be a clue as to the underlying cause of hemoptysis. The computed tomographic scan will in almost all instances document the precise location of the broncholith, and this examination should be done in all patients.

Endoscopy is, of course, mandatory. When bleeding is brisk the rigid bronchoscope permits efficient aspiration of blood and provides the opportunity for tamponade of the bleeding site. The fiberoptic instrument may be easily passed through the lumen of the rigid bronchoscope if better definition of the lesion is needed. Because the calcification may be covered by granulation tissue, the endoscopist may mistakenly believe that carcinoma is present. Thus, confirmation of calcium at this site by the computed tomographic scan is reassuring. In my experience, broncholithiasis involving the anterior segment of either upper lobe or the bronchus imtermedius has more commonly given rise to significant or massive hemoptysis. In almost every instance, bleeding occurs from erosion of a bronchial artery and rarely from a major pulmonary vessel. However, forceful attempts to carry out endobronchial extraction of calcium are ill-advised, for they may cause massive, even fatal, hemorrhage from a pulmonary arterial branch, because these vessels are usually intimately adherent to the other side of the partially extruded broncholith.

The necessity for emergency operation for hemoptysis secondary to broncholithiasis is indeed rare (only one instance in the last 32 years in my experience). Endoscopic control of bleeding permits a semielective procedure to be done. Thoracotomy is the treatment of choice for continuing massive hemoptysis. A double-lumen endotracheal tube is necessary, and after its placement the unaffected side of the tracheobronchial tree should be thoroughly cleansed before turning the patient to the lateral thoracotomy position. If bleeding can be controlled by the use of a tamponading catheter or by packing with quarter-inch gauze, a regular endotracheal tube may be used.

The hilar dissection of the patient with broncholithiasis secondary to histoplasmosis can be a technically difficult challenge to the surgeon. The individual ligation technique may not be possible because of dense scarring of the pulmonary vasculature and the bronchi. In these cases, temporary occlusion of the main pulmonary artery permits rapid transection and oversewing of segmental arteries, thus limiting the magnitude of pulmonary resection in emergency situations.

The role of bronchial artery embolization should perhaps be reserved for those patients with lesser degrees of hemoptysis and for those whose clinical status contraindicates major operative intervention.

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Thoracoscopic Neodymium : Yttrium-Aluminum Garnet Laser–Assisted Pulmonary Resection

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The recent explosion of interest in surgical endoscopic techniques has revived the application of an old thoracic surgical procedure—thoracoscopy. We report a case of a transthoracoscopic neodymium : yttrium-aluminum garnet laser resection of a limited-stage peripheral adenocarcinoma of the lung accomplished in an elderly man with serious chronic obstructive pulmonary disease.

(Ann Thorac Surg 1991;52:1176–8)

Increasing interest in minimally invasive surgical techniques has infiltrated most fields of surgery today. Laparoscopic cholecystectomy and appendectomy have dominated recent general surgical discussion and appear to be valuable adjuncts in the management of selected patients. A renewed interest in diagnostic and therapeutic thoracoscopy has also emerged [1, 2]. This report describes the successful transthoracoscopic neodymium : yttrium-aluminum garnet (Nd : YAG) laser–assisted resection of a 2.5-cm left lower lobe adenocarcinoma found in an elderly man with a synchronous stage I squamous cell carcinoma of the larynx and serious chronic obstructive pulmonary disease.

For editorial comment, see page 1036.

A 74-year-old man was referred for evaluation of a 2.3-cm noncalcified peripheral left lung nodule identified by chest roentgenography during the staging of a clinical stage I laryngeal carcinoma. Computed tomography of the chest and upper abdomen revealed a single lesion within the periphery of an emphysematous left lower lobe (Fig 1). Mediastinal lymph nodes of 1.0 to 1.5 cm in diameter were noted in the left lower paratracheal region and in the aortopulmonary window. The patient had a 60 pack-year history of smoking. Respiratory spirometric assessment was consistent with moderately severe chronic obstructive pulmonary disease (forced expiratory volume in 1 second, 1.4 L; forced vital capacity, 3.8 L). Room air arterial blood gas analysis further suggested moderate pulmonary functional impairment (pH, 7.43; oxygen tension, 61 mm Hg; carbon dioxide tension, 44 mm Hg). The patient was believed to be a marginal candidate for major pulmonary resection.

Bronchoscopic examination and mediastinoscopic staging were considered appropriate before exploration of the thoracic cavity. After induction of general anesthesia, flexible bronchoscopy was performed through the endotracheal tube. No endobronchial lesions were identified. Cervical and left parasternal mediastinoscopic examinations with nodal sampling were then performed. The frozen section evaluation of nodal tissue obtained was negative for malignant infiltration. Thoracoscopic examination of the left chest followed. Our intention was to proceed with Nd : YAG laser excision of the left lung lesion endoscopically or through a small lateral muscle-sparing thoracotomy if malignant pleural involvement could be ruled out. Left lung collapse, achieved with a bronchus blocker, facilitated thoracoscopic inspection of the lung and thoracic cavity. Laparoscopic trocars were used to introduce the operating laparoscope (catalog No. 110730; Weck, Inc, Princeton, NJ) and endoscopic forceps into the left chest. The trocar incisions were located at the fifth intercostal space along the anterior axillary line and the seventh intercostal space at the posterior axillary line. A slightly umbilicated visceral pleural surface was found over the lesion located within the lateral basilar segment of the lower lobe along the interlobar fissure. Thin adhesions along the fissure were lysed sharply with endoscopic scissors and with the aid of the Nd : YAG laser, which was introduced through the suction channel of the laparoscope. The lung was then grasped over the lesion with the forceps and drawn toward the chest wall to demarcate the extent and the base of the lesion from the surrounding collapsed lung (Fig 2).

Thoracoscopic resection was felt to be possible, however, preparations for possible open thoracotomy were made. The Nd : YAG laser system (Model 704, KTP/YA119995; Laserscope Inc, San Jose, CA) was calibrated and placed on a nonpulsed 50 W setting. Nd : YAG laser safety eye wear was used by all persons in the operating room. A 28F chest tube was placed through the eighth intercostal space at the midaxillary line under thoracoscopic guidance and connected to a smoke evacuation system (LASE System II, model SE-11111-BII; LASE Inc, Cincinnati, OH) to remove smoke generated during the anticipated laser resection. The endoscopic forceps was then applied to the perilesional lung tissue, and laser

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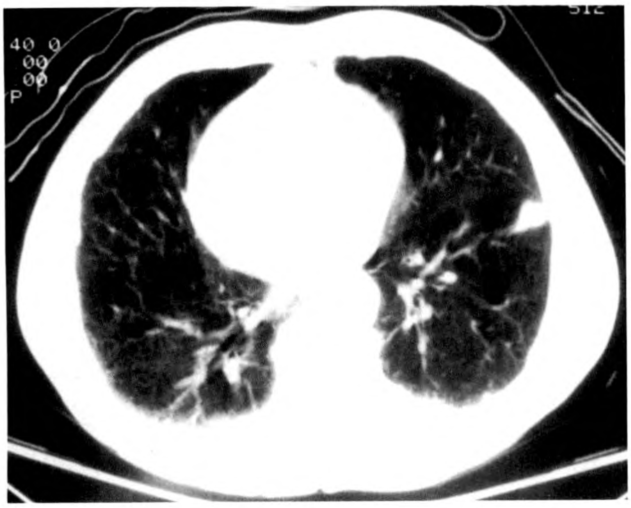

Fig 1. Computed tomography of the chest revealing pulmonary emphysema and a 2.5-cm noncalcified lesion within the periphery of the left lung.

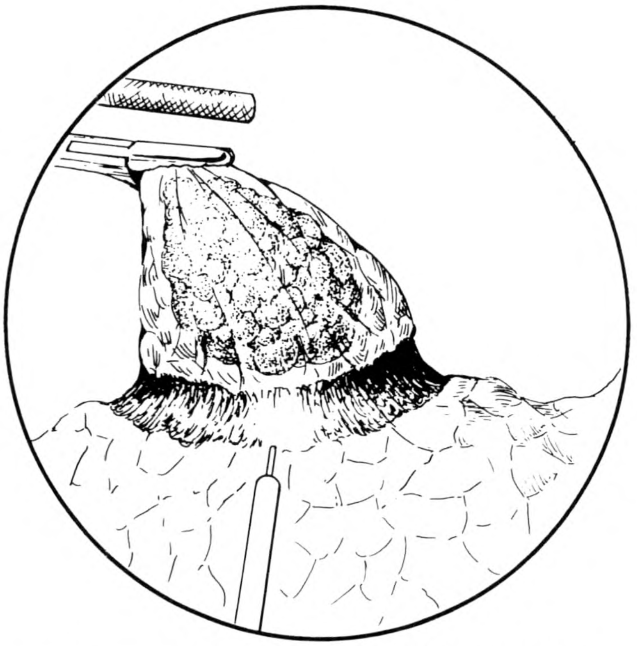

Fig 3. Margin of Nd:YAG laser resection established beneath the lesion. A laser crater is formed as coagulation and vaporization of the surrounding lung tissue occurs.

resection was begun along the base of the lesion so as to obtain a 1.5-cm margin of normal lung tissue (Fig 3).

Careful visualization along the resection margin was facilitated by frequently alternating the position of the laparoscope between the anterior and posterior intercostal trocar sites and applying countertraction with the endoscopic forceps. Hemostasis was assured during the resection by carefully identifying pulmonary parenchymal vessels as vaporization of the surrounding lung tissue occurred. The laser beam was then defocused and trained on the vessel until total coagulation was achieved. After 70 minutes of dissection the lesion was separated from the remaining normal lung tissue (Fig 4). The endoscopic forceps was then used to remove the lesion through the posterior axillary thoracoscopy site. Removal of the lesion was facilitated by extending the skin and fascial incision to a total length of 3 cm. No special measures were needed to close this incisional wound. Frozen section pathologic analysis of the lesion revealed a well-differentiated adenocarcinoma. The surgical margins were free of tumor.

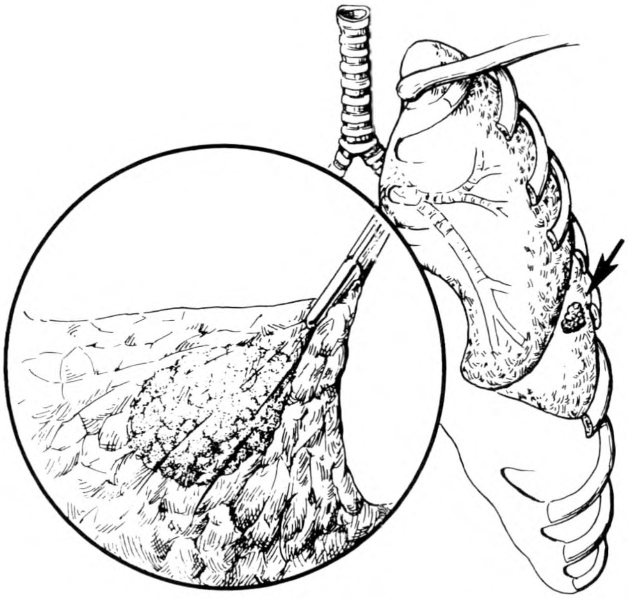

Fig 2. Endoscopic forceps grasping the lung over the lesion to facilitate demarcation of the lesion from the collapsed normal lung.

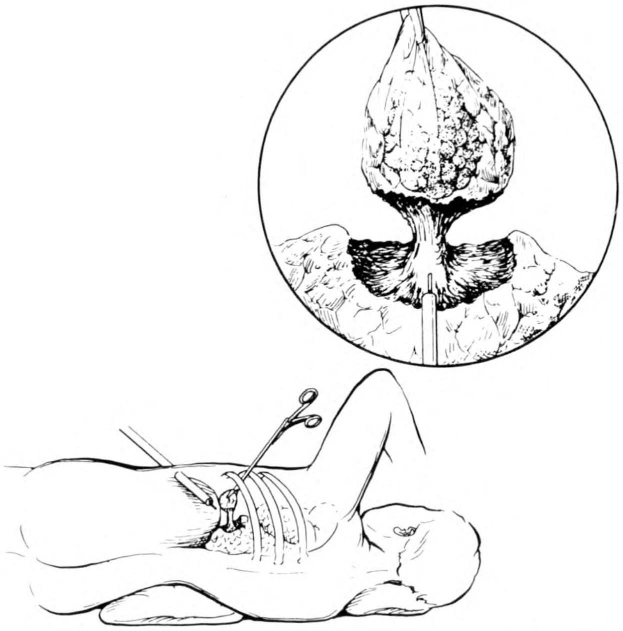

Fig 4. Parenchymal lung lesion and surrounding lung tissue being held to the underlying bed of resection by a pedicle of residual pulmonary tissue.

Hemostasis at the bed of the resection was assured by applying the defocused Nd:YAG laser to oozing and noncoagulated surfaces. The bronchus blocker was temporarily deflated, the lung inflated, and the absence of a serious air leak assured. All trocar sites were closed with absorbable suture, and the procedure was terminated after establishment of underwater seal drainage and 20 cm suction to the previously placed chest tube system.

The patient's postoperative course was uneventful except for the development of supraventricular tachycardia, which was successfully converted to sinus rhythm with digoxin therapy. Minimal postoperative air leak and chest tube drainage occurred in accordance with others' experience with laser lung resection [3, 4]. The chest tubes were removed on the third postoperative day, and the patient was discharged from the hospital on the fifth postoperative day.

Comment

Thoracoscopy is certainly not a new procedure; however, interest in this diagnostic and therapeutic modality has been limited until recently [5]. Reports of thoracoscopic laser-assisted management of spontaneous pneumothorax by Torre and associates [1] and Wakabayashi and co-workers [2] have rekindled interest in therapeutic thoracoscopy. Our group has applied the Nd:YAG laser to perform diagnostic thoracoscopic lung biopsy previously; however, this case represents our first case of transthoracoscopic therapeutic pulmonary resection. Growing experience and success with open Nd:YAG laser pulmonary resection [3, 4] encouraged this attempt at endoscopic removal of this peripheral lung mass. Collapse of the lung by a bronchus blocker or by the use of a double-lumen endotracheal tube is essential for adequate visualization of the lung and effective maneuvering of the lasing thoracoscope, endoscopic forceps, and suction catheters used during the resection.

Other investigators have demonstrated segmental pulmonary resection to have therapeutic benefit similar to lobectomy in the management of small, limited-stage non–small cell carcinoma of the lung [6]. Miller and Hatcher [7] have further shown limited pulmonary resection to be a successful surgical approach for the physiologically impaired patient with lung cancer. We strongly recommend careful prethoracoscopic staging of the patient (including mediastinoscopy when appropriate) to reduce the possibility of missing a more advanced malignancy that may be better treated with a more radical operation. At present, we would reserve thoracoscopic resection for those peripheral, limited-stage primary pulmonary malignancies found in patients with severely impaired pulmonary function or those patients with other confounding physiologic problems that seriously increase the risk of standard thoracic resection. This technique may also be used as a means of diagnosing presumed metastatic or other peripheral lung nodules of uncertain cause.

Although we are encouraged by this successful thoracoscopic resection of a small peripheral adenocarcinoma, we caution that this technique is not applicable for more deeply seated lesions in closer proximity to the pulmonary hilar structures. In fact, we suggest limiting the use of thoracoscopic resection to small (less than 3 cm in diameter) pulmonary lesions located in the outer third of the pulmonary parenchyma. Additionally, the thoracic surgeon should always be prepared to convert to open thoracotomy if there is uncertainty regarding the completeness or safety of the endoscopic resection.

Familiarity with thoracoscopy and Nd:YAG laser pulmonary resective techniques are undeniably important prerequisites to attempting this procedure [3, 4]. Appreciation of the effects of the Nd:YAG laser on pulmonary tissue is also essential to ensure hemostasis and to avoid excessive postoperative pulmonary parenchymal air leak [3, 4, 8]. Despite these stipulations, this limited experience suggests that thoracoscopic Nd:YAG laser–assisted pulmonary resection may become a valuable modality in the armamentarium of the thoracic surgeon approaching selected patients with peripheral pulmonary lesions.

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Modified Robicsek Technique for Complicated Sternal Closure

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A modification of the technique described by Robicsek and associates for treatment of sternum separation after open heart operation is described. The technique consists of placing four interlocking steel wires parasternally on both sides and then including them in the usual transverse peristernal wires.

(Ann Thorac Surg 1991;52:1179–80)

There has been an increase in sternal wound complications since median sternotomy has become the standard approach for most cardiac and mediastinal surgical procedures. The incidence of major wound complications has been reported to range between 0.7% and 1.9% [1–5] with a mortality rate between 10.3% and 39.6% [1, 3–5]. To achieve maximum sternal stability, the use of the figure-of-8 suture technique has been described in various articles with good results [6–8], but this technique is in most cases not sufficient if it has previously failed to provide stable sternal approximation, which is frequently the case if the sternum is fragmented transversely at multiple locations. We describe a technique of sternal approximation that is a modification of the technique described by Robicsek and associates [9].

Technique

Four stainless steel wires are placed parasternally on both sides. The first wire is passed through the manubrium and through the second intercostal space and then twisted, so that a ring is formed. The second suture is placed through the second and third intercostal spaces in an interlocking fashion with the first wire. This series of four interlocking steel wires is continued until the end of the sternum is reached, with the fourth wire through the fifth intercostal space (Fig 1). Placing all four wires creates a chain on both sides of the sternum (Fig 2). Subsequently, two transverse transmanubrial wires are placed laterally to both proximal parasternal wires. From proximal to distal, further transverse sternal wires are placed in the intercostal spaces until the fifth intercostal space has been reached, taking care to avoid injury of the mammary vessels. Finally, the transverse wires are tightened, thus providing lateral reinforcement to the closure (Fig 3).

Comment

We have used this modified Robicsek technique in 3 patients with osteoporotic sterna in whom complete sternal dehiscence with multiple transverse sternal fractures developed postoperatively owing to severe coughing. Chest roentgenograms revealed excellent sternal apposition with no detectable separation in all patients. At 5 months' follow-up there was still no evidence of sternal separation. We believe this technique should not be used routinely but seems to be useful in the stabilization of complicated sternal dehiscence, especially in the presence of multiple transverse fractures of the sternum. The described technique is easy to perform, and its advantage is that it provides good lateral stability. Once the transverse peristernal steel wires have been tightened, a cage is formed in which the tension is equally distributed over the entire sternal surface.

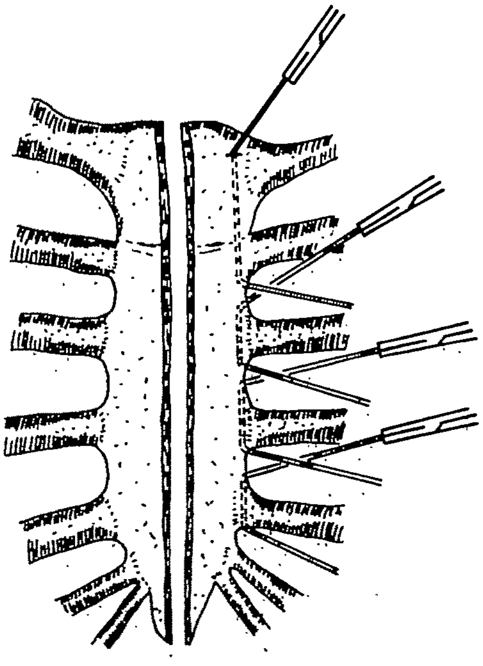

Fig 1. Placement of four interlocking parasternal steel wires.

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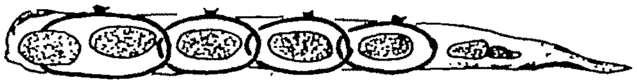

Fig 2. A parasternal chain on both sides of the sternum has been created (sagittal view).

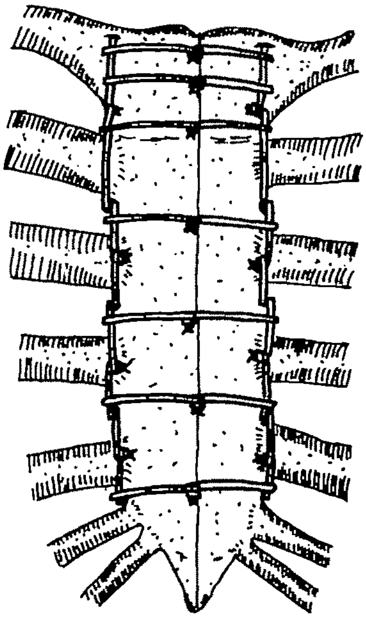

Fig 3. Final aspect: the two parasternal chains are completed with the usual peristernal wires.

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Total Orthotopic Heart Transplantation: An Alternative to the Standard Technique

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An alternative technique of total orthotopic heart transplantation is described. Complete excision of the recipient's atria is undertaken. Ventricular as well as atrial orthotopic transplantation is performed.

(Ann Thorac Surg 1991;52:1181–4)

Heart transplantation is the treatment of choice for patients with end-stage heart failure. Since its first description by Shumway and associates [1], the surgical technique of cardiac transplantation remains unchanged. It is a simple and efficient method that has been used for more than 20 years worldwide. Recent studies using transesophageal Doppler echocardiography [2] have detected anatomic and physiologic imperfections of the transplanted hearts associated with the use of this technique.

Reitz and co-workers [3] have introduced the domino procedure using technical modifications for orthotopic heart transplantation. Also, Yacoub and Banner [4] have introduced modifications to the original description by Shumway and associates. The technique we report here is a total heart transplantation including total excision of both the right and left atria of the recipient and orthotopic transplantation of an intact donor heart with its atria as well as its ventricles.

Surgical Technique

Donor Heart Harvesting

Harvesting of the donor heart is slightly modified to include a segment of the superior vena cava and the inferior vena cava as well as the four pulmonary veins intrapericardially (Fig 1). The tissue bridge between the superior and inferior pulmonary veins is secondarily resected to create a single left and right orifice.

Recipient Heart Explantation

The aorta is cannulated in the standard fashion. The superior vena caval cannula is introduced in the body of the vessel itself. The inferior vena cava is cannulated at its emergence from the diaphragm. The heart is then explanted as usual, leaving the posterior aspect of the left atrium and the posterolateral aspect of the right atrium. Remnants of right and left atria are then resected as follows: the right atrium is totally resected, leaving the origins of the superior vena cava and the inferior vena cava, and the left atrium is also completely resected, leaving only two cuffs of tissue including both ostia of the pulmonary veins (Fig 2).

Heart Transplantation

The heart is then implanted as follows. (1) The left cuff containing the recipient left pulmonary veins is anastomosed to the corresponding orifice on the donor left atrium (Fig 3). (2) The right cuff containing the recipient

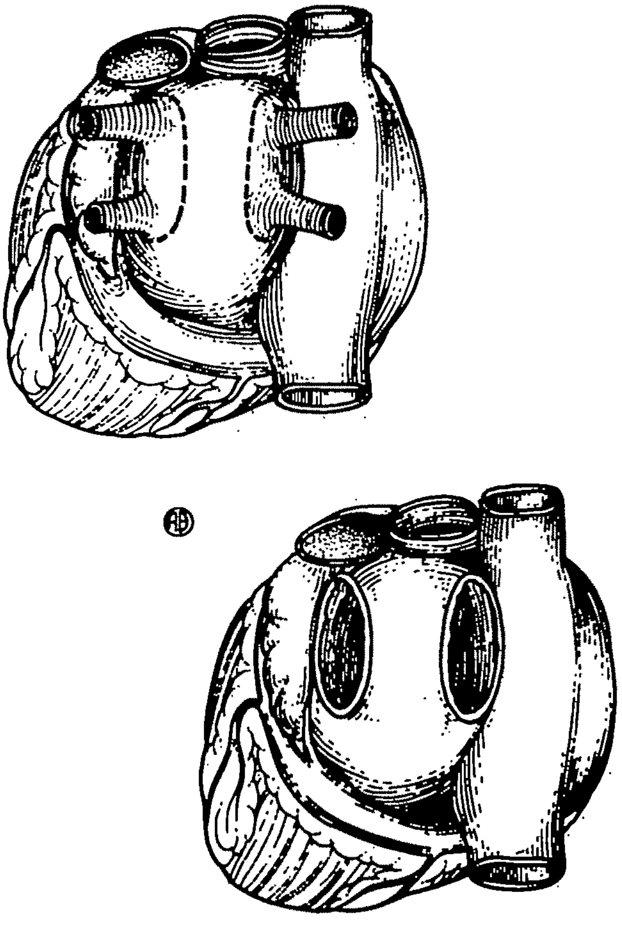

Fig 1. Donor heart harvesting: The four pulmonary veins are kept intact. The superior and inferior venae cavae are cut as long as possible.

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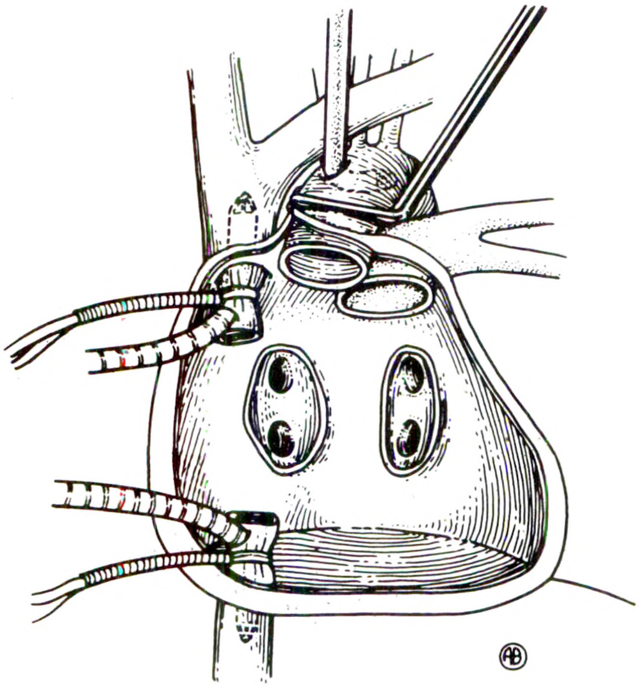

Fig 2. Recipient heart explantation: The posterior aspects of the left and right atria of the recipient are resected, leaving two cuffs with the origin of right and left pulmonary veins. Venae cavae are transected completely.

right pulmonary veins is anastomosed to the corresponding orifice on the donor left atrium (Fig 4). The suture line is started on the posterior aspect of both orifices. (3) The inferior vena cava and the superior vena cava are anastomosed in an end-to-end fashion (Fig 5), starting by the inferior vena caval anastomosis. (4) The pulmonary artery

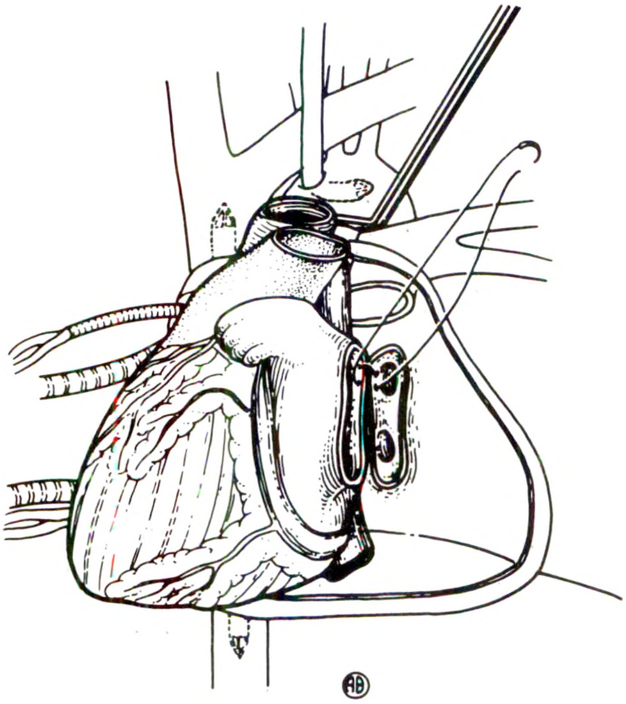

Fig 3. Left atrial anastomosis: the left pulmonary veins are anastomosed first after resection of the tissue bridge between the superior and inferior left pulmonary veins. Sutures begin at the posterior aspect.

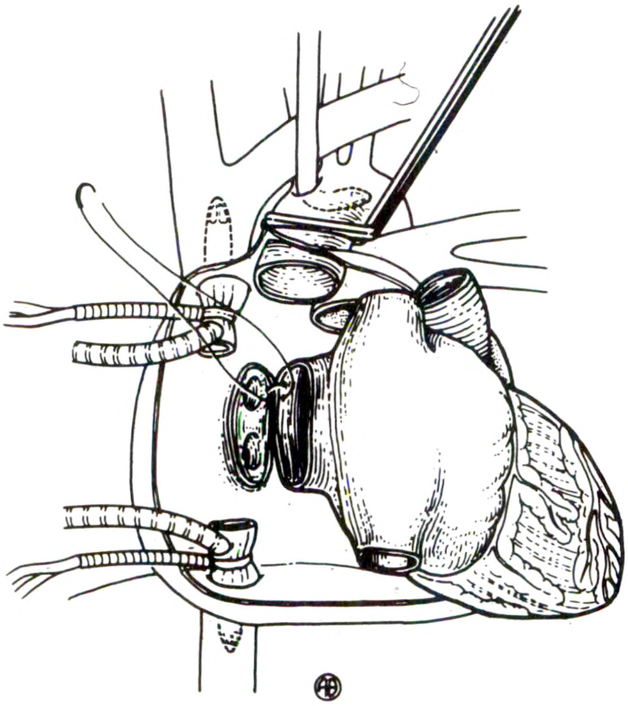

Fig 4. Left atrial anastomosis: the right pulmonary veins are anastomosed as the left one.

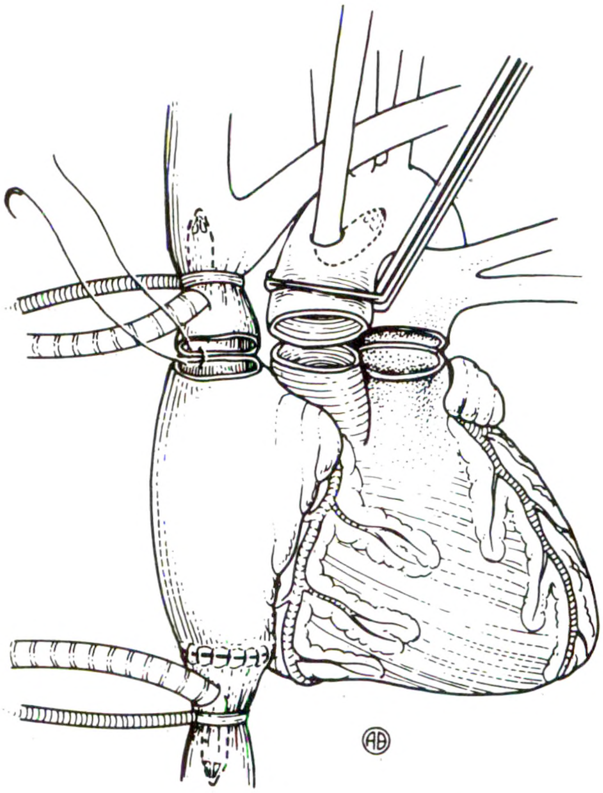

Fig 5. Vena caval sutures: these sutures are performed in an end-to-end fashion.

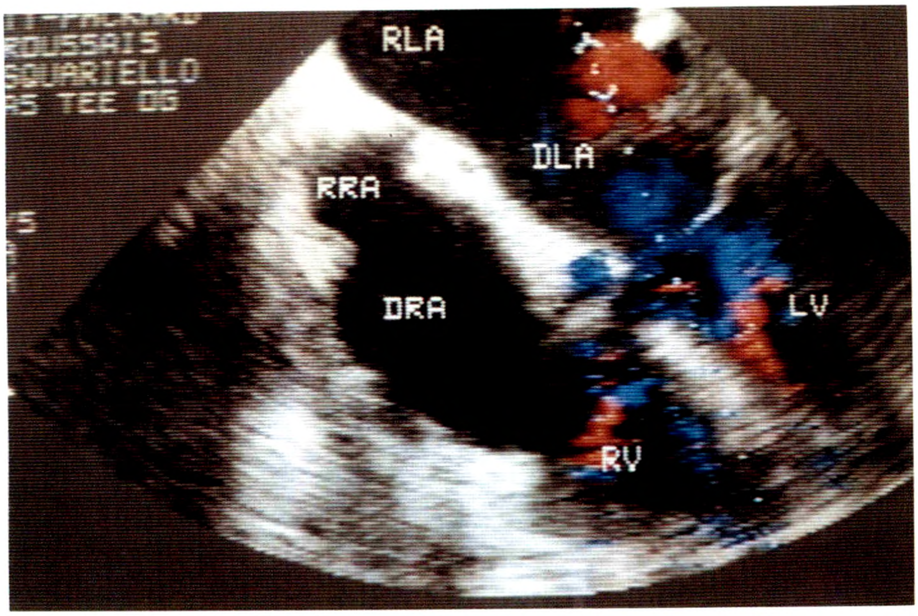

Fig 6. Transesophageal echocardiogram of an orthotopic heart transplanted using the standard technique. Note the increased size of the atria with a protruding suture line between recipient and donor atria. (DLA = *donor left atrium;* DRA = *donor right atrium;* LV = *left ventricle;* RLA = *recipient left atrium;* RRA = *recipient right atrium;* RV = *right ventricle.*)

and the aorta are then anastomosed as in the conventional technique.

Case Report

A 53-year-old patient was admitted for urgent heart transplantation after acute decompensation of a dilated cardiomyopathy. A 35-year-old donor heart became available 48 hours after his admission. Transplantation was undertaken using the method described here. The postoperative course was uneventful. Twenty months later he is alive and doing well under a triple-drug immunosuppressive regimen.

Comment

The technique described by Shumway and associates is the standard manner to perform orthotopic heart transplantation. But this technique, which is efficient, is more a ventricular substitution than a total orthotopic atrioventricular transplantation, as posterior aspects of the right and left atria of the recipient are left in place.

Recent transesophageal Doppler echocardiographic studies [2] have shown anatomic and physiologic abnormalities in hearts transplanted using this technique. The anatomic abnormalities include enlargement of the atrial cavities with protruding suture lines between the recipi-

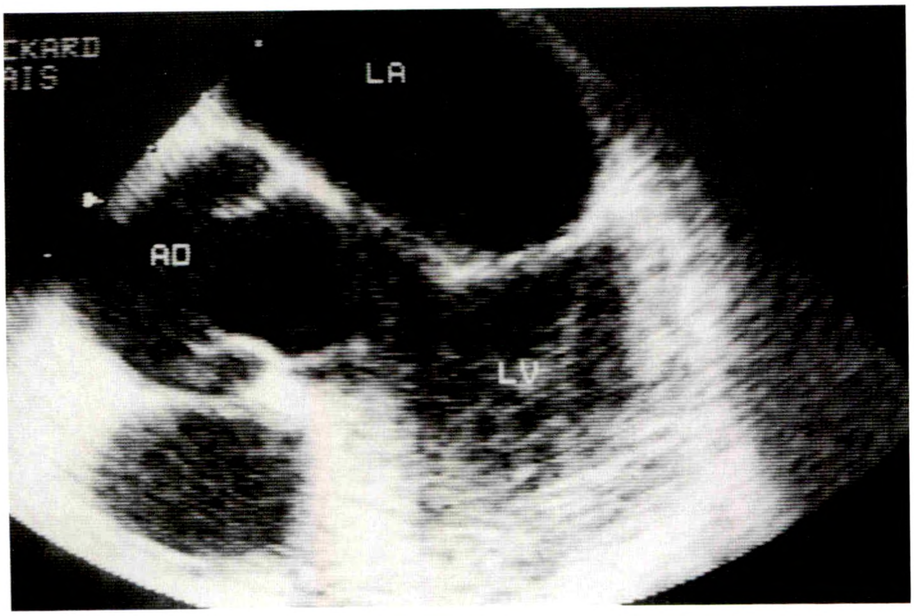

Fig 7. Transesophageal echocardiogram of a total orthotopic heart transplant. Note the physiologic size of the left atrium with no suture line. (AO = *aorta;* LA = *left atrium;* LV = *left ventricle.*)

ent and the donor atria (Fig 6). Angermann and associates [2] have reported some thrombus formation and clearly outlined that it could only be detected by transesophageal Doppler echocardiography, which is not routinely performed in heart transplantation recipients. The physiologic abnormalities include asynchronous contractions of the recipient's and the donor's atria, which lead to turbulent flows with aneurysmal behavior of the recipient's atrium (see Fig 6). Consequently, the atrioventricular valves show asynchronous opening and closure, with mitral regurgitation found in 55% of the patients [2]; and even higher incidence after heart transplantation was found by Stevenson and colleagues [5].

The technique we describe offers at least anatomical advantages, with physiologic size of the atrial cavities and no protruding suture lines (Fig 7), and absence of asynchronous atrial contractions. From the physiological point of view, this technique can avoid asynchronous atrial contraction as seen with the standard technique; it produces a normal pattern of ventricular filling as seen on Doppler echocardiography with normal E and A waves. Seven other patients have undergone transplantation with this technique and have shown no postoperative technical complications. They have had normal follow-up; in particular, there was no superior vena caval stenosis identified. However, further investigations are required to establish the true benefits of this procedure.

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INVITED COMMENTARY

More than 17,000 orthotopic heart transplantations have now been performed, and the results combining all centers reporting to the International Registry show a 1-year survival rate of more than 80%. It is clear that heart transplantation has become a routine and effective method for treating end-stage heart disease.

The authors of this report are to be congratulated for continuing to search for methods to refine the application of this therapy. The technique they present is a modification of that described experimentally by both Cass and Brock [1] and Webb, Howard, and Neely [2] in 1959, although these workers used vascular couplers to speed the anastomoses. The method subsequently described by Lower and Shumway [3], requiring just two atrial anastomoses, was so appealing in its simplicity that all 17,000 clinical orthotopic transplantations have been done in this fashion.

In the method described here, the superior vena cava, inferior vena cava, and the entrances of the pulmonary veins into the pericardium are separately anastomosed. This may theoretically be an advantage, but the proposed technique would take longer and be more technically demanding. Possible complications include stenosis of the inferior or superior vena caval anastomoses and bleeding from the inaccessible medial aspects of the pulmonary venous anastomoses. This technique would, therefore, have to demonstrate real superiority in long-term function to outweigh these disadvantages. However, it would be hard to improve on the results achieved by the conventional method. Postoperative cardiac function is excellent, and the observations described here on valvular and atrial function by echocardiography postoperatively seem to have no clinical relevance. Thromboembolism from the atrial suture lines has not been a problem. It is, therefore, unlikely that the proposed technique will gain wide acceptance.

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Improved Forceps for Pulmonary Embolectomy

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A malleable, nonocclusive forceps for surgical extraction of fresh pulmonary emboli is described. This device allows the performance of pulmonary embolectomy under direct vision with an unobscured peripheral field and reduces the potential for traumatic pulmonary artery laceration.

(Ann Thorac Surg 1991;52:1185–6)

Persistent hypotension refractory to medical treatment in patients with massive, central or paracentral pulmonary artery embolism is currently the primary indication for surgical embolectomy. A number of improvements have been made since the first pulmonary embolectomy, which was performed by Trendelenburg [1] in 1908. The most important step was probably the introduction of cardiopulmonary bypass for this procedure [2]. However, there remain some technical problems that are mainly due to inadequate instruments [3]. In general, the emboli are removed from the right and left pulmonary arteries using traditional stone forceps as in operation on the biliary tract. This may be adequate in some patients. In others, however, the stone forceps will not exhibit the necessary angle, and removal of the emboli may be difficult and induce additional trauma. Furthermore, the design of a traditional forceps with a central pivot point precludes embolectomy under direct vision or digital control because of almost complete obstruction of the pulmonary artery as soon as the hinged device is opened. Finally, the common stone forceps is designed for firm grasping, and this can also injure the friable pulmonary artery. Pulmonary artery laceration was studied in bovine experiments and appeared to be a very serious complication. It may remain hidden until weaning from cardiopulmonary bypass is attempted. At this time, blood foam will drain from the endotracheal tube, and proper ventilation may become impossible.

A new malleable, nonocclusive forceps (Fig 1) has been devised to facilitate operative pulmonary embolectomy (Ulrich AG, St. Gallen, Switzerland). The model depicted here is made from stainless steel and has an effective intrapulmonary artery length of 25 cm. In the closed position, the outer diameter is less than 5 mm despite a closure gap of 0.5 mm, and the mouth opens to 13 mm for an outer diameter of 15 mm. The shaft measures less than 3 mm in diameter. The jaws expose a concentric atraumatic grip and open and close by sliding action of the coaxial mechanism. This feature allows the surgeon to operate the device with much less space (3 mm) than that necessary for a hinged mechanism. The forceps is malleable and can be bent to reach difficult sites, as evaluated in an experimental set-up and demonstrated in Figure 2. To reduce the danger of traumatic pulmonary artery laceration the jaws of the device cannot be closed completely (intended closure gap of 0.5 mm).

Up to now, we have successfully used the malleable, nonocclusive forceps in 2 clinical cases. Pulmonary emboli

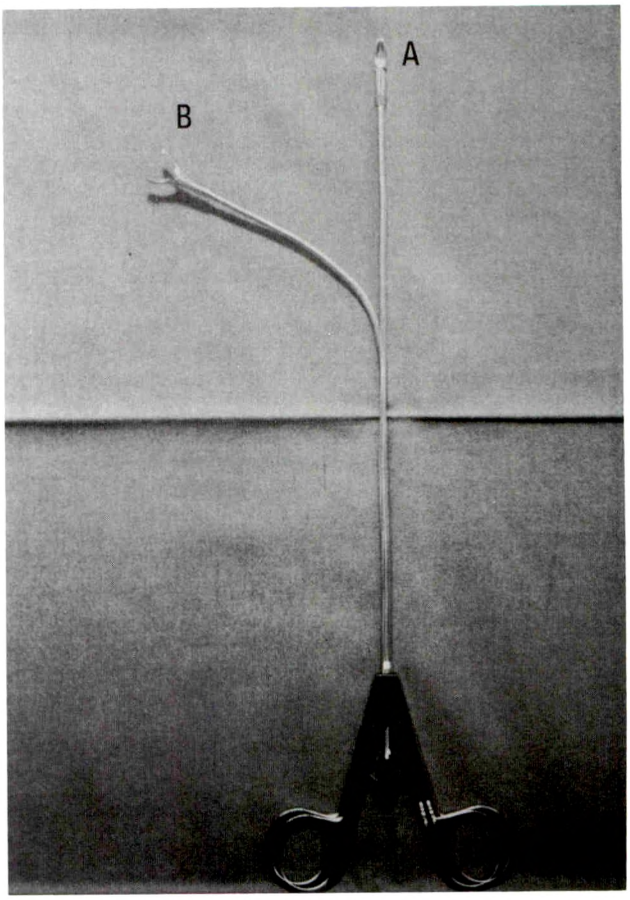

Fig 1. Atraumatic, malleable forceps for pulmonary embolectomy with coaxial mechanism (superimposed pictures): (A) in straight and closed position, (B) In bent and open position.

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were diagnosed by echocardiography; during cardiopulmonary bypass after electrical induction of ventricular fibrillation, the emboli were extracted using the described forceps under direct vision with an unobscured peripheral field. The usual struggle using a stone forceps with improper angle and fumbling to locate and grasp the emboli with the barely opened jaws of a hinged instrument was easily avoided. No complication occurred and both patients are well.

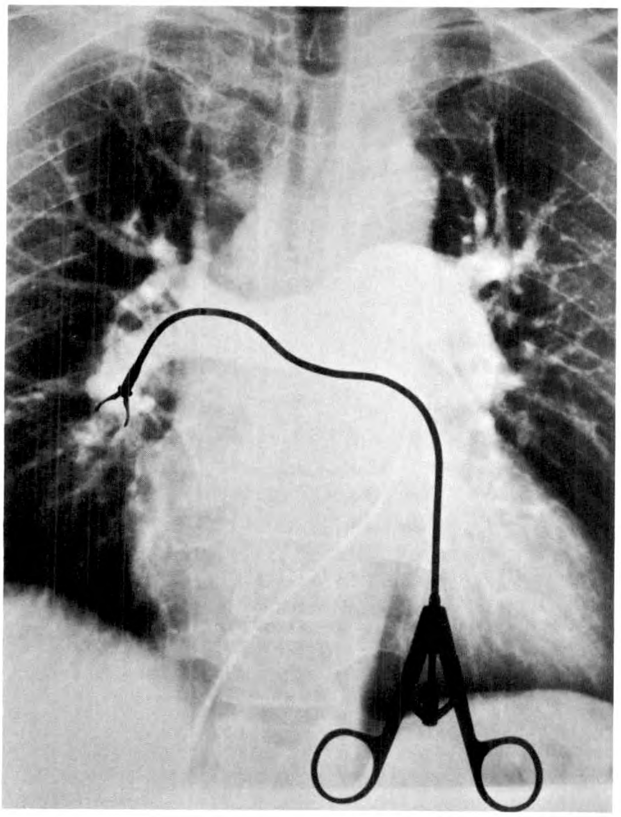

Fig 2. Removal of paracentral pulmonary emboli with the atraumatic, malleable pulmonary embolectomy forceps (schematic backlight view).

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Endarterectomy, Vein Patch, and Mammary Bypass of the Anterior Descending Artery

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Occasionally the left anterior descending (LAD) coronary artery contains such diffuse calcific atherosclerosis that an area suitable for distal anastomosis with the internal mammary artery (IMA) cannot be found. Additionally, the LAD of some patients contains multiple areas of stenosis, which would prevent free outflow from the IMA graft. In these cases the potentially increased operative risk of LAD endarterectomy is justified to avoid leaving poorly revascularized areas of anteroseptal heart. In an effort to provide the long-term patency benefits of IMA grafting for these patients without the technical difficulty of a lengthy IMA to LAD anastomosis, we have combined saphenous vein patch reconstruction with IMA bypass when LAD endarterectomy is required.

(Ann Thorac Surg 1991;52:1187–9)

Since its introduction by Bailey and associates [1], endarterectomy of the coronary arteries has had a controversial role in the reconstruction of diffusely diseased coronary arteries. Endarterectomy has been shown to carry an increased risk relative to routine coronary bypass grafting [2] and to lead to deterioration of postoperative left ventricular function in some cases [3].

When applied to the left coronary system, particularly the left anterior descending coronary artery (LAD), endarterectomy has been shown to significantly increase the risk of mortality and perioperative myocardial infarction [4–7]. Nonetheless, an occasional patient has an LAD that is so diffusely diseased that an acceptable portion of arterial wall with which to construct a distal anastomosis does not exist. Additionally, multiple areas of stenosis may prevent flow from the bypass conduit to important segments of LAD or to important side branches. In such cases, one must weigh the risk of endarterectomy of the LAD against the risk of leaving poorly vascularized segments of left ventricle. Endarterectomy of the LAD with saphenous vein (SV) bypass grafts has led to acceptable results in some hands [8] and has prompted us to proceed with endarterectomy in these cases. Because visualization of the endarterectomized arterial wall allows for a more accurate assurance of adequate tapering of the distal transition points, the resultant LAD arteriotomy can be quite long. The arteriotomy may also extend into multiple side branches in order to complete endarterectomy of the diagonal arteries. Reconstruction of the widely opened LAD can be technically challenging. In an effort to provide at least part of the LAD luminal circumference with a thromboresistant surface without losing the long-term benefits of IMA patency, we have used a technique of SV reconstruction of the LAD and bypass with the internal mammary artery (IMA) when a long endarterectomy is required.

Technique

When the LAD is completely calcified and a suitable area for construction of an anastomosis with the IMA cannot be found, the LAD is opened and a limited area (1.0 to 2.0 cm) is subjected to endarterectomy under 4.5-power magnification. If a 1.5-mm probe cannot be freely passed in the direction(s) of expected blood flow or if an unacceptably "feathered" endarterectomy margin is achieved, a more extensive endarterectomy is performed, including all major septal perforators and diagonals (Fig 1a–c). When necessary to confirm smoothness of the distal endarterectomy edge, we do not hesitate to carry the arteriotomy into the diagonal arteries. Direct visualization of all endarterectomy edges is performed, rather than resorting to "blind" traction on the atheromatous plaque.

After completion of the endarterectomy, a SV patch is opened longitudinally, with orientation appropriate to its expected blood flow. The vein graft is then tailored to accommodate arteriotomy extension into the diagonal arteries, if necessary, and sewn in place using 6-0 or 7-0 continuous polypropylene suture. Trimming the width of the SV patch is desirable to avoid the creation of a patulous LAD lumen. The resultant reconstructed LAD system is depicted in Figure 1e. At this time, additional cardioplegia is administered through either coronary sinus or aortic root, both for myocardial protection and to assess the hemostasis of the reconstruction. Next an anastomosis is created between the tailored outflow end of the IMA and a 3- to 5-mm longitudinal incision in the vein patch using continuous 8-0 polypropylene. The resultant bypass graft is depicted in Figure 1. It should be noted that proximal patency of the LAD is undesirable. In fact, it is preferable to avoid competitive flow for the IMA. Consequently, we usually do not remove the entire prox-

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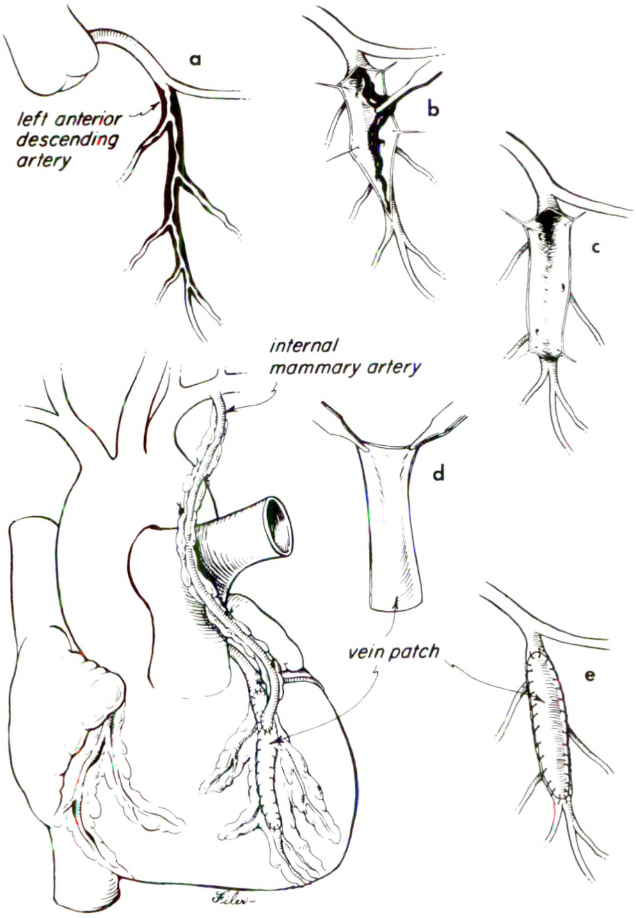

Fig 1. Endarterectomy, reconstruction, and bypass of the left anterior descending coronary artery.

imal plaque unless there is no risk of competitive flow from the left main or circumflex coronary arteries.

Postoperative care is routine, with the exception that we empirically anticoagulate these patients with heparin in the immediate postoperative period. We then replace the heparin with Coumadin (crystalline warfarin sodium), maintaining a prothrombin time of 1.25 to 1.5 times control, for 3 months. At the end of this period, Coumadin administration is discontinued and an aspirin regimen is initiated.

Patients and Results

We have resorted to this technique of endarterectomy, reconstruction, and bypass of the LAD system in 18 patients (0.8%) out of 2,184 coronary artery procedures during the past 4 years. Nine of these patients were in clinical class IV, and 9 had unstable exertional angina. There were 5 female and 12 male patients, with an average age of 60 years. Three patients had suffered preoperative myocardial infarction. Two patients had undergone previous coronary artery bypass procedures. Four of the patients had occluded LADs documented with preoperative angiography. Twelve patients had patent (but stenotic) LADs. Two patients had occluded native LADs with a patent (but stenotic) vein graft supplying the LAD. The average preoperative left ventricular ejection fraction was 0.53 (range, 0.30 to 0.65). All patients had viable left ventricular anterior walls as evidenced by at least hypokinetic motion on ventriculography.

The average length of LAD endarterectomy was 5.5 cm (range, 1.5 to 10 cm), and in 2 patients extension of the arteriotomy into a diagonal artery (with endarterectomy and separate vein patch reconstruction) was performed. An average of 2.5 additional bypass grafts were performed (range, 1 to 4).

Postoperatively, 2 patients suffered myocardial infarctions. One of these patients had an occluded LAD preoperatively, and the myocardial infarction was thought to be in an area other than that supplied by the reconstructed LAD. The second patient had a patent LAD preoperatively and suffered a postoperative myocardial infarction in the anterior heart. This incidence of 11% compares with a postoperative myocardial infarction incidence of 3.6% in 2,166 patients who did not undergo this revascularization technique. The difference in this small series does not attain statistical significance (by χ^2), and the incidence in this series is similar to that of other reported series of LAD endarterectomy [1, 2]. There were no operative deaths.

The 18 patients have now been followed up an average of 12 months (range, 1 to 28 months). One patient returned 8 months postoperatively with angina. Evaluation with coronary angiography revealed a widely patent IMA to LAD reconstruction with severe progression of his circumflex and right coronary artery disease. His anterior wall functioned normally. There have been no late deaths, and all patients except the one just mentioned are free of angina.

Comment

When extensive endarterectomy under direct visualization of the LAD is required, five options exist for reconstruction of the long arteriotomy. Complete proximal endarterectomy with simple closure (or vein patch closure) is generally avoided in favor of simultaneous bypass grafting. A second option consists of creation of a long SV to LAD anastomosis. This technique is simple, but the unquestioned superiority of the IMA is lost. A third technique consists of partial closure of the LAD arteriotomy with bypass grafting to a limited portion of the arteriotomy. In the portion of LAD subjected to primary closure, the entire lumen has undergone endarterectomy and is theoretically "thrombogenic." A fourth option consists of a lengthy anastomosis of the IMA to the LAD. This technique is feasible but difficult and time-consuming [9]. A final technical option consists of reconstruction of the opened LAD with a tailored SV patch (avoiding a patulous lumen and potential IMA to LAD flow discrepancies) and the technically expedient IMA-to-vein-patch anastomosis. We have used this latter technique in a series of 18 patients with acceptable intermediate-term results.

Although our patients have not been subjected to postoperative radionuclide studies or angiography, we are confident that those patients with a patent LAD system preoperatively would have had postoperative electrocardiographic or creatine kinase evidence of myocardial injury if the operative reconstruction of the LAD had occluded. We can therefore recommend this technique as an acceptable method of LAD reconstruction and bypass after extensive endarterectomy.

Addendum

Since submission of this manuscript, we have used this technique in 2 more patients, each with a patent but diffusely diseased LAD. Neither suffered postoperative myocardial infarction. Both are doing well.

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Ross' First Homograft Replacement of the Aortic Valve

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Ross' first homograft replacement of the aortic valve was reported in 1962. The homograft has been in continuous use around the world ever since. Much has been learned about how to handle homografts, both before and during their implantation. Homografts have special advantages, which make them an appropriate choice in a number of settings. This first successful case by Donald Ross set the stage for the growth in homograft valve use and the subsequent development of many ways of using allograft cardiovascular tissue for optimal cardiac reconstructions.

(Ann Thorac Surg 1991;52:1190–3)

Viable cardiovascular allografts were first clinically used by Gross in 1948, and then in 1952, DuBost established aortic continuity with a homograft aorta after resecting an abdominal aortic aneurysm. Direct cardiac applications were conceived in the laboratory preparations of Lam and associates [1]. Valved aortic homografts were first used when placed in the descending aorta to ameliorate native aortic valve insufficiency by Gordon Murray in 1956 [2], but the first orthotopic use of a homograft for replacement of an aortic valve occurred in July 1962 and was reported 6 weeks later in *The Lancet* by Donald Ross [3]. This operation preceded by about 1 week a similar procedure by Barrett-Boyes in New Zealand [4].

To understand the importance of this 1962 contribution, it must be placed in context with the evolution of cardiac surgery. The cardiopulmonary bypass machine had only been available since 1954. The mechanical cardiac valve of Starr (1960) had been reported but was not yet available in the United Kingdom. There were many patients requiring treatment for valvular heart disease, and the limitations of valvuloplastic methods were increasingly appreciated. The current resurgence of interest in homografts makes another look at this initial contribution by Donald Ross more pertinent.

Ross Operation—The Real Story

A fair amount of folklore has arisen about this first case and somewhat apocryphal stories are in circulation. As it turns out, Ross was quite cognizant of Lam's work on dogs in the 1950s and of Duran and Gunning's technical advances placing a homograft aortic valve in the "subcoronary position." In fact, their preliminary communication [5] was reported in the same issue of *The Lancet* on the page next to the publishing of Ross' clinical experience. Ross and his laboratory colleagues had been working on freeze-drying aortic valves and had been collecting valved aortic roots, evaluating them in their pulse duplicators both before and after freeze-drying. At the time of the reported operation, Ross had a small bank of freeze-dried valves and was prepared to use one in the orthotopic position, even though he had not yet sewn one in a dog (Donald Ross: personal communication). Initially Mr Ross attempted a decalcification of the native valve leaflets, which resulted in total destruction during the operation. The freeze-dried homograft was reconstituted and sutured into position with six separate mattress sutures and a distal suture line consisting of continuous 3-0 silk. The patient's atrial septal defect was also closed. He survived approximately 3 years without evident aortic valve dysfunction, and apparently, no autopsy was performed (Donald Ross; personal communication, 1991). The patient's death was due to cardiac failure, consistent with the stage at which aortic valve operation was undertaken in those early days.

As previously demonstrated in this series, "chance favors the prepared mind"; it is clear that this operation was not due to simple, quick thinking. Ross was prepared and had been performing relevant laboratory experiments well before the clinical application. This early surgical success with freeze-dried material later led to the widespread use of this relatively harsh preservation method, which was ultimately associated with poor long-term material durability. Nevertheless, this initial clinical triumph indicated the value of placing a well-functioning aortic valve in the orthotopic position and underscored many of the advantages of the homograft over the early mechanical prostheses (*vide infra*). Two additional advantages specific to this early era were availability and low cost. Once manufactured valves became available in large numbers, the logistics of homograft retrieval and processing made them less attractive. The cost of homografts continued to be favorable in the United Kingdom, Australia, and New Zealand, and economics was one of the reasons for continued use.

Relevance to Current Surgical Practice

The surgical method for aortic valve replacement with a homograft has evolved since the initial Ross operation, to a great part due to Ross himself and the people he has trained. Other major early contributors include Barrett-Boyes, Kirklin, Duran, Angell, O'Brien, Wallace, and

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Yacoub [5–9]. More extensive reconstructions of the aortic outflow involve a spectrum of techniques that capitalize on the material properties of the homograft [10, 11].

Advantages of Homografts

1. Optimal hydraulic function with central nonobstructive flow results in excellent hemodynamic performance, even in small sizes, and thus optimal hemodynamics for both right and left ventricular outflow valve replacement, especially for children and in the adult small aortic root.
2. Thromboembolism and hemolysis rates are extremely low, requiring no major anticoagulation.
3. Surgical implantation is relatively simple: right-sided conduit operations are easier with homografts than with rigid Dacron conduits, and left-sided reconstructions are facilitated by the greater flexibility in the way aortic homografts can be used in either "freehand" implantation or variations of aortic root reconstructions.
4. Calcification rarely affects the leaflets; thus, ultimate homograft failure usually results in gradual valvular insufficiency, rather than obstructive physiology.
5. Rapid "rejection" with disappearance of the leaflets of the homograft has been reported but is extremely rare (specific immunosuppressive therapy is almost never used).
6. The viable allograft appears to have the greatest resistance to prosthetic endocarditis, and therefore it is the valve of choice in the infected heart.

Indications for Use

Current practice by most congenital cardiac surgeons is to always use homografts (when available) for right ventricular outflow tract reconstructions [12]. Homografts can be used even in the smallest of infants. Once a child has reached the 15-kg weight range, adult-sized right ventricular outflow tract reconstructions can be accomplished. The long-term results with wet stored (ie, not cryopreserved) homograft right-sided reconstructions have been excellent when prosthetic extensions have been avoided, and thus the expectation is reasonable that cryopreserved tissues will also perform well in this position.

For left-sided reconstructions, acceptable options include mechanical and xenograft prostheses, each with specific characteristics, advantages, and disadvantages. One clear indication for the use of the homograft in the aortic root is for acute active bacterial endocarditis, either prosthetic or native [6, 13]. Another is in children requiring left ventricular outflow tract reconstruction either as an aortic valve or an aortic root replacement with or without annulus enlargement procedures (eg, Konno). The special qualities of the homograft serve to simplify the technical aspects of complex left ventricular outflow tract reconstructions in both adults and children. In such reconstructions, homografts would be preferred, even if durability were no greater than xenografts, because the other advantages of technical simplicity, hydraulic superiority, and infection resistance are so favorable. The small aortic annulus and the congenital aortic stenosis spectrum can often be repaired more completely and easily with a homograft, encouraging an approach to the left ventricular outflow tract using reconstruction techniques of annuloplasty, aortoplasty, and various grades of root replacement (eg, mini-root, "full" root replacement with coronary ostial transplantation). Many of the difficulties of implanting rigid prostheses into complicated geometric situations are easily resolved with a homograft [10, 11].

The role of freehand homograft aortic valve replacement for routine aortic valve replacements is more controversial. The limitation of the resource counsels against use in elderly patients in whom xenograft valves perform well without anticoagulation. Women of child-bearing age and other patients aged less than 50 years are attractive candidates for the homografts because of the lack of requirement for anticoagulation and the possibility of increased durability (beyond that expected of a xenograft and approaching the expected mechanical prosthetic performance) from the new generation of cryopreserved valves [14]. Patients without contraindications to anticoagulation between age 50 and 65 years are usually well managed with mechanical prostheses.

This original operation by Ross has stimulated other surgeons' ingenuity to expand the applications of such transplanted tissues. Cryopreserved cardiovascular tissues are now used for a variety of nonvalved operations in which their material properties are found to be superior. The hypoplastic left heart operation includes the use of homograft pulmonary artery tissue as a key component for the reconstruction of the aortic arch. Nonvalved pulmonary artery bifurcations and pulmonary artery tissue simplify extensive reconstructions in complicated forms of pulmonary atresia and acquired pulmonary artery stenoses. The Konno procedure has been combined with the aortic root replacement as a total left ventricular outflow reconstruction tunnel aortic stenosis using homograft for all tissue repair.

The first Ross operation gave tremendous impetus to the evolution of homograft surgery. And even more important has been the continuing commitment by Ross and his London colleagues to the further development and refinement of the use of homografts [15]. Their unabated interest has continued since 1962. As with any replacement device, homograft cardiovascular tissue transplants are not perfect and are not always the ideal material.

Possible Disadvantages of Homografts

1. Potential transmission of disease
2. Lack of availability
3. Unknown durability beyond 15 years for currently processed valves
4. Evolving preservation techniques
5. Imperfectly understood role of the immune response
6. Some variability in the performance of these valves
7. Surgical technical challenges

Cardiovascular Tissue Harvesting and Preparation Methodology

Ross' case is a pioneering event that classically illustrates the enormous utility of the surgical case report. The rapidly reported and widely disseminated success in this patient led to life-enhancing operations in hundreds, if not thousands, around the world in the 1960s and after. However, this same case report also illustrates the limitation of this type of communication if extended beyond surgical method, as the preservation methodology was similarly applied elsewhere. Unfortunately, durability was adversely affected by the freeze-drying process (and other harsh preservation methods); this fact was recognized within 5 years [16]. Ross and Barratt-Boyes realized this and settled on a gentle tissue culture–type cold storage method, adopted worldwide during the 1970s. The valves harvested from the donor shortly after death, gently treated and maintained in nontoxic antibiotic-laced tissue culture medium at 4°C (so-called antibiotic wet stored), have been found to have durability matching or slightly exceeding xenografts in the aortic position and tremendously superior in the pulmonary outflow position [17].

The application of cryopreservation to complex tissues (derived from the cryobiology of individual cell preservation—eg, sperm, red blood cells) to capture the durability advantage of the fresher tissue has been a natural and logical progression. The initial work by Angell and O'Brien has led to the Brisbane series of aortic valve replacements with durability approaching or exceeding 90% valve and patient survival at 15 years [14]. Given the other advantages of homograft valves, this is a remarkable achievement that has naturally led to a resurgence of interest by physicians. (An analogous 15-year series for cryopreserved valves in the right ventricular outflow position is not yet available.) Great attention is now focused on the lack of availability of cryopreserved valves, which is the greatest restraint on their use.

The exact role of fibroblast cellular viability is also not yet clear; it is not known how much quantitive cellular viability is needed to confer increased durability. The role of leaflet endothelium is not understood; it may stimulate antibody response and actually decrease the long-term survival, and thus should be specifically removed during processing [18]. Optimal methods are still not strictly defined for managing the intervals between donor demise and processing, the limits of transport conditions, storage, thawing, and insertion. However, certain features are now being teased from the available data. Prolonged warm ischemia (greater than 24 to 36 hours), harsh harvesting, and thermally unstable transportation methods appear to be injurious. High concentrations of antibiotics or prolonged disinfection protocols are particularly damaging to cell survival [19]. Prolonged cold, wet storage before cryopreservation (5 to 7 days) and improper freezing and thawing techniques lead to cell disruption. The enhanced durability of cryopreserved or very fresh valves has been attributed to putative fibroblast viability at the time of implantation. The notion is that these cells can participate in repair and remodeling of the leaflet, thus preserving the material properties, strength, and elasticity of the leaflet matrix—perhaps functioning to normalize it after the stresses of storage and transplantation, and maybe even repairing damage accruing with the 60 to 180 flexions per minute for the months and years following surgical implantations. These attractive hypotheses are not yet proven [20]. It may be that cell viability is simply a marker for well-maintained matrix physical-chemical properties, or that fibroblast viability is present only for the initial restoration of the matrix after transplantation [21–26]. Despite pending proof of mechanism, it is hard to ignore the improving results and increasing "durability" of the "gently" treated homograft.

In essence, the homograft method for aortic valve replacement has almost a continuous 30-year track record—exceeded only by 2 years by the Starr valve. It is an acceptable if not preferable "prosthesis" for many indications and certainly does not qualify as an "experimental" device. The Engineer who designed this valve does not require pulse duplicator experiments to validate His design. The principles of preparation are well understood, and worldwide variations generally fall well within the scope of current tissue banking knowledge and understanding and represent relatively minor modifications of an overall approach. The challenge facing regulatory groups, cryopreservation facilities, cardiovascular scientists, and cardiac surgeons, who wish to establish aspects critical to the enhancement of homograft performance while maintaining the greatest possible safety for the recipient, is to avoid doing worse than has been achieved by Ross and others for the past 28 years.

Ross and the other early advocates of the homograft valve have done a tremendous service by demonstrating the usefulness and in some applications the superiority of the homograft valve. Even the aseptic "nonviable" homografts (fresh wet stored) have great utility and an excellent record that would qualify or even mandate use in patients when other advantages exceed the properties of available mechanical and xenograft prostheses. The homograft is an important therapeutic option. Its availability needs to be greatly amplified by increased donations, more extensive harvesting, and the creation of realistic harvesting, processing, and allocation protocols that maximize availability while achieving, as a minimum, the performance of the "gently treated" "nonviable" valve of the wet storage era. Ultimately, it will be possible to identify and separately categorize valves for which the potential durability exceeds that historical "norm" and thus select these valves for use in younger patients.

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Editor's Note

We asked Mr Ross for his comments on Dr Hopkins' interesting article.

Mr. Ross' Commentary

It is interesting to recall that we had so little confidence in the first homograft that we regarded it as a temporary expedient until we could import a Starr valve and buy time while we did accelerated fatigue testing on our freeze-dried valves. However, as days passed into weeks and finally to months with maintained valve function and with an increasing number of valves in patients, we forgot about mechanical valves and concentrated on refining our homograft insertion techniques. Also, after collateral work by our immunological colleagues at Guy's Hospital, we accepted the decision that although there was an immunological reaction it did not justify the use of steroids and the resulting danger of infection.

Different sterilization and stage methods have not eliminated the problem of tissue failure as far as we are concerned. This feature has been the main stimulus responsible for our development of the pulmonary autograft operation, which we consider to offer a permanent valve uniquely suited to children and young adults.

Encouraged by the impressive function of the autogenous pulmonary valve in the aortic position, we have turned to the pulmonary homograft, which is readily available and usually of better quality than its aortic counterpart. Not only has it performed impeccably in right ventricular reconstruction, but we and others are investigating its use as an aortic valve replacement.

The most rewarding aspect of our homograft experience after years in the surgical wilderness is to enjoy the newfound enthusiasm of colleagues over the world.

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Surfactant Analysis and Replacement Therapy: A Future Tool of the Lung Transplant Surgeon?

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From 1965 to 1974 extensive research was carried out concerning the effects of experimental lung reimplantation and allografting on the surface tension properties of pulmonary surfactant. Since then, surfactant has been more rigorously examined in terms of its composition and function, and the potential roles of three surfactant-associated proteins have been established. Furthermore, surfactant replacement therapy for neonatal respiratory distress syndrome has come of age. The efficacy of surfactant treatment for adult respiratory distress syndrome is currently under clinical scrutiny, and experimental work on alterations in surfactant after lung transplantation has resumed after a 15-year hiatus. This article reviews current knowledge of the pulmonary surfactant system, as well as previous studies of the changes in surfactant after experimental lung transplantation. The experience in surfactant replacement therapy for the neonatal and adult respiratory distress syndromes is briefly described. Suggestions are made concerning the potential experimental and clinical applications of surfactant analysis and replacement therapy in lung transplantation.

(*Ann Thorac Surg 1991;52:1194–200*)

Since the first successful use of surfactant replacement therapy in the neonatal respiratory distress syndrome a decade ago [1], surfactant supplementation has become established in neonatology in the care of premature infants with very low birth weight [2]. Treatment of experimental models resembling the adult respiratory distress syndrome (ARDS) with exogenous surfactant has also proved beneficial [3, 4], and clinical trials of surfactant therapy in ARDS in humans are in progress [3–6].

Since the previous review of experimental lung transplantation and surfactant in 1974 [7], exceptional progress has been made in characterizing the physicochemical properties of surfactant phospholipids and surfactant-associated proteins, and successful lung transplantation has become a clinical reality. Recent experimental work has been directed to documenting the changes in surfactant phospholipids during lung preservation and reperfusion [8] and to the potential value of alterations in the lipid and protein profiles of bronchoalveolar lavage (BAL) in diagnosing lung rejection and infection postoperatively [9]. In addition, the possibility of extending the interval of lung allograft preservation by surfactant therapy is currently under investigation in two Canadian centers. The clinical application of surfactant analysis and replacement will likely become increasingly familiar to thoracic surgeons as new strategies for treating ARDS evolve and as experience with lung transplantation continues to expand.

Function of the Pulmonary Surfactant System

The lung is an extremely complex organ, containing an extensive network of 100 to 300 million alveoli and pulmonary capillaries which are designed to allow for gas exchange between air and blood. This air-liquid interface is subject to surface tension forces, which are an important determinant of pulmonary mechanics [10]. As early as 1929 von Neergaard [11] postulated the existence of pulmonary surfactant as a surface-tension–reducing agent in mammalian lungs. It was not until the 1950s, however, that Pattle [12] observed that lung edema foam contained a substance that stabilized small bubbles, and Clements [13] provided direct evidence for the surface-tension–reducing properties of lung saline extracts.

Pulmonary surfactant can be thought of as an emulsifying agent that stabilizes the critical air-blood interface in the lungs. Given the small diameter of alveoli (100 to 200 μm) and the retractive forces present in the lung, in the absence of surfactant a considerable pressure difference would arise across the alveolus during expiration, and the alveoli would probably collapse or absorb fluid from the interstitial space [10]. By decreasing surface tension, surfactant promotes alveolar stability at low lung volumes and protects against pulmonary edema under physiologic conditions [10]. Surfactant also plays an important secondary role in promoting the function of local airway defense mechanisms against airborne antigens and in

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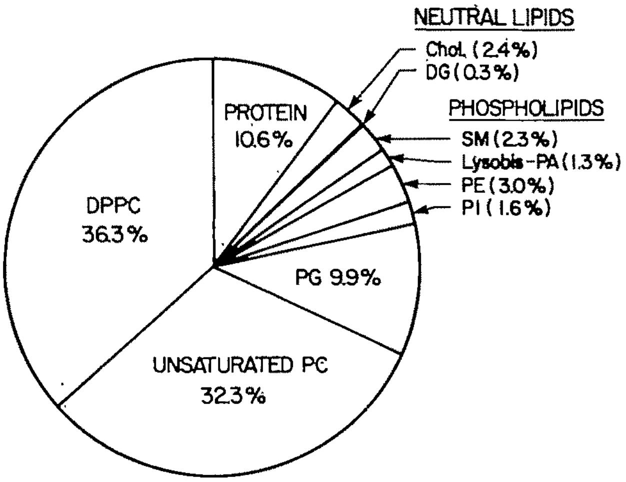

Fig 1. The composition of bovine pulmonary surfactant. (Chol = *cholesterol;* DG = *diacylglycerol;* DPPC = *dipalmitoylphosphatidylcholine;* Lyosbis-PA = *lyso-bis-phosphatidic acid;* PC = *phosphatidylcholine;* PE = *phosphatidylethanolamine;* PG = *phosphatidylglycerol;* PI = *phosphatidylinositol;* SM = *sphingomyelin.) (Reproduced from Possmayer F, Yu SH, Weber JM, Harding PGR. Pulmonary surfactant. Can J Biochem Cell Biol 1984;62:1121–33, with permission.)*

enhancing alveolar and bronchiolar mucous transport [14].

Composition of Pulmonary Surfactant

A comparison of surfactant from a large variety of species shows a striking consistency in its chemical composition [10]. Purified bovine surfactant contains approximately 90% phospholipids, 2% to 3% neutral lipids, and 5% to 10% protein [10]. The composition of bovine pulmonary surfactant is depicted in Figure 1. A majority of the main constituent, phosphatidylcholine, is present in the disaturated form as dipalmitoyl-phosphatidylcholine (DPPC). Dipalmitoyl-phosphatidylcholine is the primary component of surfactant responsible for producing the marked reduction of surface tension necessary to stabilize the alveolus. The anionic phospholipids phosphatidylglycerol and phosphatidylinositol account for slightly more than 10%, and the other phospholipids for 6% to 7% of bovine surfactant by weight.

Synthesis, Storage, and Secretion of Surfactant

The pulmonary alveolar epithelium is composed of two cell types, elongated type I cells, which cover 97% of the alveolar surface, and cuboidal type II cells, which predominate in the alveolar corners [15]. It is the type II alveolar pneumocyte that synthesizes, stores, and secretes pulmonary surfactant [15–18]. The surfactant material is packaged as lamellar bodies, which contain phospholipid bilayers with a high content of disaturated phosphatidylcholines [15, 17–19]. In the lining layer of the alveolus, the lamellar bodies are converted into a unique structure known as tubular myelin [15, 18, 19]. Tubular myelin serves as a reservoir of surfactant molecules which are available for adsorption to the air-liquid interface to form a monolayer of surfactant lipids. Repeated compressions of this surface monolayer during breathing lead to a squeezing out of unsaturated lipids, resulting in a monolayer enriched in DPPC which is capable of producing a very low surface tension (Fig 2).

The release of surfactant from type II cells in the alveolar epithelium is under the control of a number of factors. Surfactant secretion is promoted by β-adrenergic and cholinergic agonists, by various prostaglandins, and by increased ventilation [16, 17]. De novo surfactant synthesis is stimulated by glucocorticoids, and in some species by estrogen and thyroid hormone [17]. An extensive recycling system exists in both neonatal and adult lungs by which surfactant lipids derived from the alveolar surface layer are taken up by type II cells and reutilized for surfactant production [10, 17, 19].

Structure and Function of Surfactant-Associated Proteins

Recent studies have identified three distinct surfactant-associated proteins that appear to play an important role in the biophysical activity of lung surfactant. These proteins include the hydrophilic, large molecular weight surfactant-associated protein A (SP-A) (26,000 to 35,000 daltons), and the smaller, highly hydrophobic proteins SP-B (8,000 daltons) and SP-C (5,000 daltons) [20–22]. Recent work indicates that both SP-B and SP-C facilitate the adsorption and spreading of phospholipids to form

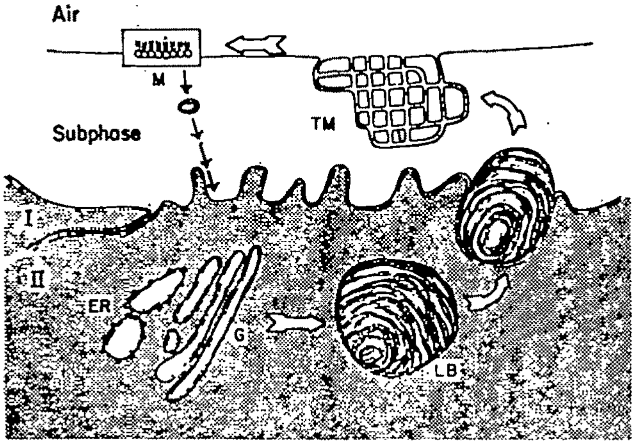

Fig 2. Formation and excretion of pulmonary surfactant. After exocytosis, the lamellar bodies form tubular myelin, which is thought to be the source of the monolayer. Only the monolayer can act in the reduction of surface tension. Surfactant lipids are recycled presumably as small vesicles by type II cells for reutilization in surfactant production. (ER = *endoplasmic reticulum;* G = *Golgi apparatus;* LB = *lamellar bodies;* M = *monolayer;* TM = *tubular myelin.) (Reproduced from Possmayer F, Yu SH, Weber JM, Harding PGR. Pulmonary surfactant. Can J Biochem Cell Biol 1984;62:1121–33, with permission, as modified from Goerke J. Lung surfactant. Biochim Biophys Acta 1974;344:241–61.)*

the surface-active monolayer and thus are important components of pulmonary surfactant activity [20–22]. Surfactant-associated protein A is not essential for surfactant function but acts synergistically with SP-B to enhance lipid adsorption and decrease surface tension [22]. In addition, SP-A promotes calcium-dependent lipid organization and contributes to the formation of tubular myelin in the alveolus [19, 22, 23]. Experiments with the past year have also established that SP-A effectively counteracts the inhibition of surfactant activity in vitro by the serum proteins fibrinogen and albumin [24]. The end result of the concerted action of these surfactant-associated proteins is to generate and maintain the phospholipid monolayer in the alveolus in a condition conducive to alveolar stability and optimal lung function.

Surfactant Inhibitors

The role of surfactant inhibitors has received increasing attention recently in the literature. It has become apparent that the surface activity of surfactant relates not only to its quantity and composition, but also to the presence or absence of intraalveolar surfactant inhibitors. The high permeability pulmonary edema that is present in ARDS results in the accumulation of plasma proteins, cell membrane lipids, and even hemoglobin in the airways, all of which are capable of the biophysical inhibition of surfactant [25]. Of the plasma proteins, fibrin monomers and fibrinogen appear to be more powerful inhibitors of surfactant than albumin [26]. It is likely that instead of aggregating to form a fibrin clot in the alveolus, fibrin monomers form a complex with one or more constituents of surfactant, thus interfering with surfactant function [26]. Recent work has revealed that the plasmin-induced lysis of intraalveolar fibrinogen can result in the prolonged generation of surfactant inhibitors and in the potential impairment of surfactant function for days [27]. Importantly, the inhibition of surfactant by proteins is affected by the surfactant concentration, and the effects of surfactant inhibitors can be mitigated by increasing surfactant concentration to high levels by exogenous supplementation [25, 28, 29]. This finding is of potential clinical relevance and suggests that surfactant replacement therapy might be effective in countering dysfunction of the surfactant system in ARDS or after lung transplantation.

Surfactant Replacement Therapy for the Neonatal Respiratory Distress Syndrome

It has long been suspected that a primary deficiency of surfactant is a major factor in establishing the neonatal respiratory distress syndrome [30, 31]. The first promising results with surfactant replacement therapy for this condition, published more than a decade ago [1], provided the impetus for subsequent randomized clinical trials of surfactant supplementation. More than a dozen such studies in the past 6 years have demonstrated a reduction in oxygen and ventilatory requirements in infants with very low birth weight after endobronchial installation of organic solvent extracts of calf [32–34], bovine [35–39], or porcine [40] surfactant, natural human surfactant [41–43], or artificial surfactants [18, 44]. In a number of these studies, however, the therapeutic effect of exogenous surfactant was transient, and several authors have commented that multiple doses of surfactant may be required for optimal therapy [32, 34, 36], presumably because inhibitory proteins may be present in the airways of infants with the respiratory distress syndrome. Further studies investigating different surfactant regimens in the treatment of this condition are in progress.

In the past 4 years, several "state of the art" reviews of surfactant replacement therapy have been published [2, 15, 18]. Research on the production of artificial surfactant preparations is expanding, and it is hoped that synthetic "natural" surfactants will soon be formulated in vitro using mixtures of phospholipids and surfactant-specific proteins generated using molecular biological techniques [2, 20].

Surfactant Abnormalities in the Adult Respiratory Distress Syndrome

Although the etiology of ARDS is multifactorial and is different than that of the neonatal respiratory distress syndrome, patients with ARDS also exhibit inadequate surfactant activity and qualitative alterations in the phospholipid composition of surfactant [6, 45]. In addition, ventilation with high tidal volumes and with a high inspired oxygen concentration may further impair surfactant synthesis and function, and worsen the preexisting surfactant abnormalities in patients with ARDS.

Several studies have documented alterations in surfactant phospholipid metabolism and composition during ARDS in humans [46–48]. In a heterogeneous group of patients, Hallman and associates [46] found no significant difference between patients with ARDS and normal controls in the amount of phospholipid in surfactant obtained by BAL. The phospholipid profile of patients with ARDS, however, was qualitatively abnormal, exhibiting a decreased lecithin/sphingomyelin ratio and a significantly decreased concentration of phosphatidylglycerol and disaturated phosphatidylcholine [46]. Glycolipids were barely detectable in the BAL of healthy controls, but in patients with ARDS the amount of glycolipid relative to phospholipid was increased more than 20 times [47]. In particular, the glycolipids lactosylceramide and paragloboside were most prevalent in patients with ARDS; these glycolipids were also noted to decrease the surface activity of surfactant in vitro [47]. A more recent prospective study of a relatively homogeneous patient group with posttraumatic ARDS has confirmed that the lavage concentrations of phosphatidylcholine and phosphatidylglycerol decrease during posttraumatic pulmonary dysfunction and that the changes in surfactant composition are correlated with the severity of respiratory failure [48]. The authors of that study also noted that the BAL protein content was markedly increased in patients with the most severe clinical ARDS and commented on the pathophysiologic role of serum protein leakage into the alveolus in causing adverse alterations in surfactant function.

Surfactant Replacement Therapy for the Adult Respiratory Distress Syndrome

Research in the treatment of ARDS has been hampered by the fact that ARDS is a syndrome rather than a disease and exhibits multiple causes as well as a heterogeneous patient population. Surfactant supplementation in experimental animals with hyperoxic lung injury [49] and with various models of acute respiratory failure resembling ARDS in humans [3, 4] has been beneficial and has reliably led to an improvement in gas exchange and lung mechanics.

Published clinical experience with surfactant replacement therapy for ARDS has to date been anecdotal, but some positive results have been reported [3–6]. Prospective, randomized studies of surfactant replacement therapy for ARDS in humans are in progress and should define its role in the treatment of this condition.

Changes in Surfactant in Experimental Lung Transplantation

From 1965 to 1974 extensive laboratory work was performed characterizing alterations in the surface activity of surfactant after lung reimplantation and allografting in dogs. The predominant view was that lung reimplantation did not adversely affect the function of the surfactant system as long as the ischemic time was brief [7]. This conclusion and most of the early research on the changes in surfactant after lung transplantation were based on measurements of surface tension of saline extracts of minced lung tissue rather than on quantitative or qualitative alterations in the composition of surfactant [7].

The results of five studies on surfactant and lung transplantation from this early era merit review. The initial report by Waldhausen and associates [50] found that the "stability index" of the lung decreased progressively in allografts, starting at 48 hours postoperatively. Significant reductions in surfactant activity of lung extracts were demonstrated at 2 and 8 days. This pioneering work therefore documented marked decreases in pulmonary compliance and surfactant activity after lung allotransplantation in the absence of postoperative immunosuppressive therapy.

The next report, by Yeh and associates [51], focused on the pulmonary function of dogs that had received left single-lung transplants and were immunosuppressed with either methotrexate or azathioprine. Biopsies of the allograft were performed at 3 to 4 days and between 20 and 90 days postoperatively, and surfactant activity was monitored. At 3 to 4 days the surface tension of lung extracts was unchanged, but at 20 to 90 days the allografted lungs were consolidated and the surfactant was "grossly abnormal." Yeh and associates noted that the procedure of left lung transplantation per se, with its attendant denervation, interruption of lymphatics, and transient pulmonary ischemia, had no obvious adverse effect on surfactant activity. Only after delayed rejection had occurred did the surfactant become abnormal in their study, as well as in Waldhausen's experiments [50].

In the third study from the early era of experimental lung transplantation, Yakeishi and associates [52, 53] noted a pronounced reduction of surfactant activity during the first week after grafting, with a gradual return to normal by 4 weeks in surviving dogs that were administered azathioprine and prednisone. With the onset of severe allograft infection or congestion, the surface tension and stability index of lung washings again became abnormal. Yakeishi and associates noted an indirect relationship between the duration of graft ischemia and the surface activity of the BAL from transplanted lungs but concluded that surfactant activity determinations did not provide a sufficiently reliable method of evaluating lung function after transplantation.

A further report by Thomas and Jolly [54] investigated the relationship between the intensity of postoperative immunosuppressive therapy and changes in the surface tension activity of lung extracts after left lung transplantation. Dogs that did not receive postoperative immunosuppression and those treated with azathioprine alone exhibited a marked impairment in surfactant activity on postoperative day 7. In animals that were administered both azathioprine and prednisone postoperatively, surfactant function was normal 1 week postoperatively. The dose of azathioprine administered by these investigators (7 mg · kg^{-1} · day^{-1}) was considerably higher than that given by Yakeishi and associates [52, 53].

The final study from the early era was the first attempt at quantitating surfactant turnover in allografted lungs [55, 56]. Drews and associates injected dogs with albumin-bound 1-^{14}C-palmitate before operation. They then performed left lung reimplantation in 10 dogs and left lung allotransplantation in 8 additional dogs. The decrease in radioactivity of the total lipid extract, which was believed to reflect the turnover or loss of surfactant lipids, was identical in control (nonoperated) dogs and in dogs that had undergone left lung reimplantation. After allotransplantation the total lipid radioactivity decreased further and was significantly lower than in controls when measured on postoperative days 2 to 13. Drews and associates concluded that lung transplantation was associated with increased lipid turnover and surfactant loss, which they attributed to a manifestation of rejection despite the postoperative administration of azathioprine and methylprednisolone. In a final experiment, these investigators noted that saline extracts from dog lung allografts markedly inhibited the surface tension of surfactant when mixed with normal dog lung extracts [57]. They hypothesized that this could have been due to the presence of surfactant inhibitors in the transplanted lung extracts, thus anticipating the current interest in the role of surfactant inhibitors in a wide variety of pathological pulmonary conditions.

To our knowledge, from 1974 to 1989 no further studies concerning surfactant and lung or heart-lung transplantation were published. In 1989, Jurmann and associates [9] examined the predictive value of alterations in BAL in diagnosing infection and rejection after heterotopic heart–left lung transplantation. All 11 transplanted dogs received cyclosporine, azathioprine, and prednisone for postoperative immunosuppression. Bronchoalveolar lavage was performed, and the material obtained was analyzed for protein content and for individual phospholipids using thin-layer chromatography. Simultaneous biopsies of the left lung allografts were performed, and the animals were divided into groups with normal histology, pulmonary rejection, bacterial infection, and a mixed group showing both infection and rejection. Jurmann and associates found significant differences in the BAL lipid and protein content depending on whether the allografts exhibited rejection or infection. In the infection group, marked increases in BAL protein were detected, whereas in the rejection group significant increases in the quantity of nonsurfactant lipids were documented. Jurmann and associates concluded that analysis of the BAL fluid after heart-lung transplantation may offer promise in differentiating pulmonary rejection from infection. However, they did not specify the nature of the lipids other than phospholipids that were present in BAL during rejection. Furthermore, the phospholipid composition of the lavages from the control (nontransplanted) dogs exhibited relatively high concentrations of sphingomyelin and lysophosphatidylcholine, indicating that this surfactant was contaminated with cellular membranes. The conclusion of this report must therefore be regarded as tentative, especially in view of the recent finding in heart-lung transplant patients that the profile of BAL proteins did not help to differentiate infection from rejection [58].

The most recent study in the literature concerning pulmonary surfactant and lung transplantation focused on the changes in DPPC levels in surfactant with hypothermic lung ischemia and reperfusion [8]. Klepetko and associates performed left lung transplantation in 12 dogs after flushing the pulmonary artery with 60 mL/kg of modified Euro-Collins solution and storing of the lungs at 4°C for 4 hours. Postoperatively, the oxygen tension was measured after 1, 4, 12, and 24 hours of reperfusion following 20 minutes of right pulmonary artery clamping. Allograft biopsy and BAL specimens were obtained at similar time intervals, and the concentration of DPPC was determined by gas liquid chromatography. Six dogs received a series of L-carnitine infusions preoperatively and postoperatively in an attempt to bolster surfactant lipoprotein content, because carnitine is an essential cofactor for long-chain fatty acid transport through the mitochondrial membrane of alveolar type II cells [8]. Postoperatively, there were no significant differences in oxygen tension between the carnitine and control groups at 1 and 4 hours of reperfusion, but at 12 and 24 hours the carnitine group exhibited significantly higher oxygen tensions and lower alveolar-arterial oxygen differences than controls. Klepetko and associates documented a steady decrease in the DPPC concentration of lungs that were stored at 4°C as well as in the transplanted lungs in the control (noncarnitine) group. In the carnitine group, the DPPC concentration of the transplanted lungs did not change after 4 hours of ischemia and throughout the reperfusion phase. Klepetko and associates concluded that the DPPC portion of total phosphatidylcholine decreases parallel to the extent of the ischemic damage to type II pneumocytes, and that the application of L-carnitine improved pulmonary function and surfactant DPPC levels after transplantation. Unfortunately, they did not measure changes in other surfactant components, such as phosphatidylglycerol, phosphatidylinositol, and the surfactant-associated proteins, and did not document alterations in the surface activity of surfactant after the 4 hours of lung storage and during reperfusion. Furthermore, they employed a relatively short ischemic time for the lung allografts; presumably, the changes in DPPC concentration would have been more pronounced if ischemic times of 8 to 12 hours had been used.

Potential Role of Surfactant Analysis and Replacement in Clinical Lung Transplantation

To our knowledge there are no published reports examining the role of surfactant analysis or replacement therapy in clinical lung or heart-lung transplantation. The distinction of infection from rejection without the need for transbronchial lung biopsy remains an important clinical issue postoperatively, and it is hoped that specific qualitative changes in the phospholipid profile of surfactant may occur in association with lung rejection. In view of the positive experience with surfactant replacement therapy in the neonatal respiratory distress syndrome and possibly ARDS, this treatment modality should be investigated in experimental lung transplantation. If surfactant therapy significantly improves long-term lung preservation, it may be possible to extend the interval of safe lung allograft storage and to accept less than ideal lung donors to ameliorate the severe donor demand-supply imbalance that currently exists in clinical lung and heart-lung transplantation.

Before the clinical application of surfactant therapy in lung transplantation, much work remains to be done in the laboratory. The changes in the composition and function of the surfactant phospholipids and proteins need to be elucidated after various intervals of hypothermic lung storage and reperfusion. In addition, the role of surfactant inhibitors after lung transplantation needs to be examined. Furthermore, the optimal surfactant dosage remains to be established. The action of the different types of surfactant in countering surfactant inhibitors also needs to be addressed in view of the evolving role of the surfactant-associated proteins in this regard [24].

An important issue concerns the optimal method to maximize the distribution of exogenous surfactant in the lungs. A major concern is that the distribution of surfactant used for replacement therapy will be nonuniform, with most of the exogenous surfactant going to better ventilated areas and regions of atelectasis receiving little or no surfactant [25]. Although the delivery of surfactant as a saline suspension to neonates has been well tolerated, the volume of surfactant required for supplementation in adults may exceed 250 mL [59]. Perhaps the direct instillation of exogenous surfactant just before anastomosis of the allograft, combined with an aerosol delivery system for postoperative use, may be optimal.

The possible immunologic sensitization of patients to components of exogenous surfactant is an important concern in surfactant replacement therapy [60, 61]. Experimental work in animals has confirmed the transepithelial flux of exogenous surfactant into the pulmonary interstitium and its clearance by the circulation [60]. This occurrence should expose exogenous surfactant to the immune system, which could result in antibody production and the activation of complement [60]. However, it appears that SP-B and SP-C, which are present in most surfactant lipid extracts used for replacement therapy, are not very antigenic. In a series of 140 premature infants with severe respiratory distress syndrome who received human surfactant supplementation, circulating surfactant immune complexes were detected but no free antisurfactant antibodies [60]. Despite close follow-up for more than 6 months, none of these patients showed any evidence of immune complex–mediated damage. Furthermore, before treating 3 ARDS patients with Curosurf, an organic extract surfactant derived from minced porcine lungs, Richman and associates [6] performed preliminary skin testing and in vitro lymphocyte proliferation assays in 10 adult volunteers. Curosurf caused only a mild nonspecific skin reaction in doses less than 1 mg and was not associated with an in vitro lymphocyte proliferation response or a release of interleukin-2 [6]. There is some evidence in the literature that surfactant enhances bacterial phagocytosis and intracellular killing by alveolar macrophages [61]. The fact that alveolar type II cells can synthesize and secrete proteins of the complement pathways in addition to surfactant provides evidence that surfactant operates in an immunologically complex milieu [61]. The extent to which the immunologic properties of surfactant and the sensitization of the host by heterologous surfactant proteins are mitigated by the concurrent administration of immunosuppressive agents is unknown.

In conclusion, much work remains to be done to elucidate the role of surfactant analysis and replacement therapy in lung transplantation. This area should prove fertile ground for research toward resolving the issues of rejection diagnosis and long-term lung preservation that continue to plague lung transplantation today.

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Bronchial Adenoma

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Diagnosis

Bronchoscopic Appearance

1. Becker HD, Schulz V, Tuenberthal S, Vollhaber H-H. Atlas of bronchoscopy: the bronchi. Philadelphia, B.C. Decker, 1991: 88.

   The premier photographic atlas for fiberoptic bronchoscopy demonstrates the appearance of intraluminal carcinoids and cylindroma. This book is must reading and visualizing for both the endoscopic neophyte and expert.

Bronchoscopic Biopsy Techniques

2. Wang KP. Biopsy techniques in pulmonary disorders. New York: Raven Press, 1989:70, 142–8.

   Commentary on the cytopathologic diagnosis, differential diagnosis, and recommendations for transbronchial needle aspiration are well discussed and illustrated.

Radiologic Fine-Needle Aspiration Biopsy

3. Sinner WN. Triple fine-needle biopsy of Kulchitsky cell carcinomas I, II. Eur J Radiol 1988;8:165–71.

   Thirty-two cases of typical or atypical carcinoids were diagnosed preoperatively by fine-needle biopsy without major complication. Cytohistologic features on fine-needle biopsy correlated directly with clinical outcome.

Computed Tomographic Findings With Bronchial Adenomas

4. Lau LSW, Simpson L, Murphy F. High resolution CT scanning of the bronchial tree. CT bronchography: technique and clinical application. Australas Radiol 1985;29:323–31.

   The technique of high-resolution computed tomographic (CT) scanning allows for more specific staging of endobronchial tumors, characterization of differing endobronchial tissue types, and assessment of abnormal bronchial caliber. The thin-slicing, high-resolution reconstructions enhance anatomical detail and may aid in surgical planning specifically for sleeve-type resection.

5. Aronchick JM, Wexler JA, Christen B, Miller W, Epstein D, Gefter WB. Computed tomography of bronchial carcinoid. J Comput Assist Tomogr 1986;10:71–4.

   Carcinoid tumors may enhance on CT after injection of contrast medium. This enhancement is uniform and can be contrasted with nonenhancement of bronchogenic carcinomas, and may allow greater evaluation of the extent of the bronchial adenoma.

6. Forster BB, Muller NL, Miller RR, Nelems B, Evans KG. Neuroendocrine carcinomas of the lung: clinical, radiologic, and pathologic correlation. Radiology 1989;170:441–5.

   A careful delineation of the differences in the CT findings with Kulchitsky cell carcinomas (KCCs) (see next section) types I through III is reported in 31 cases of these tumors, contrasting differences in endobronchial growth, size, presence of hilar and mediastinal adenopathy, and extrathoracic metastases.

Classification of Adenomas (ie, Carcinoids), With Special Emphasis on Neuroendocrine Features

Kulchitsky Cell Classification

7. DeCaro LF, Paladugu R, Benfield JR, Lovisatti L, Pak H, Teplitz RL. Typical and atypical carcinoids within the pulmonary APUD tumor spectrum. J Thorac Cardiovasc Surg 1983;86:528–36.

8. Paladugu RR, Benfield JR, Pak HY, Ross RK, Teplitz RL. Bronchopulmonary Kulchitzky cell carcinomas. Cancer 1985; 55:1303–11.

9. Yellin A, Benfield JR. The pulmonary Kulcitsky cell (neuroendocrine) cancers: from carcinoid to small cell carcinomas. Vol 9. Chicago: Year Book Medical, 1985.

   These three reports summarize the classification of typical carcinoids as KCC I, atypical carcinoids as KCC II, and small cell lung cancer as KCC III, representing a spectrum of tumor originating from the same stem cell but with very differing clinical courses. The use of DNA content to classify these neoplasms (which correlated with aggressiveness of the cancers) represented a novel approach to their diagnosis and definition of prognosis. Reference 9 is a superb summary of the classification, clinical manifestations, staging, and surgical and adjunctive approaches for KCC I through III.

Gould's Neuroendocrine Differentiation Classification

10. Gould VE, Llinoila I, Memoli VA, Warren WH. Biology of disease. Neuroendocrine components of the bronchopulmonary tract: hyperplasias, dysplasias, and neoplasms. Lab Invest 1983;49:519–37.

11. Warren WH, Gould VE, Faber LP, Kittle CF, Memoli VA. Neuroendocrine neoplasms of the bronchopulmonary tract. J Thorac Cardiovasc Surg 1985;89:819–25.

12. Warren WH, Faber LP, Gould VE. Neuroendocrine neoplasms of the lung. A clinicopathologic update. J Thorac Cardiovasc Surg 1989;98:321–32.

13. Warren WH, Memoli VA, Jordan AG, Gould VE. Reevaluation of pulmonary neoplasms resected as small cell carcinomas. Significance of distinguishing between well-differentiated and small cell neuroendocrine carcinomas. Cancer 1990; 65:1003–10.

    This classification uses electron microscopy, immunohistochemistry, and molecular markers, as well as light microscopy, to classify neuroendocrine tumors as well-differentiated (atypical or malignant carcinoids and some peripheral stage I small cell lung cancer), more aggressive intermediate cell neuroendocrine carcinomas, and small cell neuroendocrine carcinomas. These tumors, representing a spectrum of low-grade to high-grade carcinomas, are to be contrasted with the true benign bronchial carcinoids. Reference 13 retrospectively comments on the behavior of tumors that, though originally classified as small cell lung cancers, prove to be well-differentiated neuroendocrine carcinomas and have a significantly better prognosis.

14. Benfield JR. Neuroendocrine neoplasms of the lung. J Thorac Cardiovasc Surg 1990;100:628–9.

    The shortcomings of each classification of neuroendocrine tumors are debated by their chief proponents.

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Bronchial Carcinoids and the Ectopic ACTH Syndrome

15. Findling JW, Aron DC, Tyrrell JB, Shinsako JH. Selective venous sampling for ACTH in Cushing's syndrome: differentiation between Cushing disease and the ectopic ACTH syndrome. Ann Intern Med 1981;94:6647–52.

    Selective venous sampling of the petrosal sinus effluents for adrenocorticotropic hormone (ACTH) could define whether the source of Cushing's syndrome was from a pituitary adenoma or from an ectopic source. Failure for the elevated ACTH level to lateralize would prompt a search for an ectopic source, chiefly in the thorax.

16. Doppman JL, Loughlin T, Miller DL, et al. Identification of ACTH-producing intrathoracic tumors by measuring ACTH levels in aspirated specimens. Radiology 1987;1663:501–3.

    Percutaneous aspiration of bronchial carcinoid tumors in 3 patients with Cushing's syndrome from an ectopic source revealed high levels of ACTH in the tumor, permitting absolute identification of the ectopic source.

17. Doppman JL, Pass HI, Nieman LK, et al. Detection of ACTH-producing bronchial carcinoid tumors: MR imaging vs CT. AJR 1991;156:39–43.

    Magnetic resonance imaging confirmed the location of bronchial carcinoid tumors that were detected by CT. Moreover, the tumors appeared "bright" on certain magnetic resonance imaging sequences, and in 2 cases magnetic resonance imaging detected small tumors that were missed by CT.

18. Pass HI, Doppman JL, Nieman L, et al. Management of the ectopic ACTH syndrome due to thoracic carcinoids. Ann Thorac Surg 1990;50:52–7.

    Fifteen patients with thoracic carcinoids had thoracotomy for ectopic ACTH syndrome. There was a 50% incidence of lymph node involvement, confirming the malignant potential of these tumors. Despite this, 71% of the patients were cured with normal ACTH levels 5 to 57 months after anatomic (lobar) resection and lymph node dissection. A review of the world's literature for this phenomenon is included.

19. Scully RE, Mark EJ, McNeely WF, McNeely BU, eds. Case records of the Massachusetts General Hospital. N Engl J Med 1987;317:1648–58.

    A case of ectopic ACTH syndrome from a bronchial carcinoid secreting corticotrophin-releasing factor is discussed and serves as a nice review of the topic.

Pathologic Features

Biochemical and Ultrastructural Markers

20. Feldman JM, Benning TL, Saltzman H. Biochemical and ultrastructural differences between mucoepidermoid and carcinoid tumors of the bronchus. J Surg Oncol 1988;37:227–31.

    Bronchial carcinoids differ from mucoepidermoid adenomas with respect to measurement of monoamine oxidase and the presence of mucin and tonofilaments on electron microscopy.

21. Al-Saffar N, White A, Moore M, Hasleton PS. Immunoreactivity of various peptides in typical and atypical bronchopulmonary carcinoid tumours. Br J Cancer 1988;58:762–6.

    Well-differentiated neuroendocrine carcinomas differed from typical carcinoids in immunostaining for bombesin and gastrin, and in 30 of 57 cases, immunoreactivity for more than one peptide (bombesin, gastrin, glucagon, somatostatin, calcitonin, and ACTH) was detected.

DNA Analysis of Carcinoid Tumors

22. Jones DJ, Hasleton PS, Moore M. DNA ploidy in bronchopulmonary carcinoid tumours. Thorax 1988;43:195–9.

    Fifty-three (28 typical carcinoids and 25 well-differentiated neuroendocrine tumors) bronchopulmonary carcinoids were analyzed by flow cytometry for DNA content, and aneuploidy was significantly associated with increased malignant potential.

Clinicopathologic Prognostic Factors

23. Hasleton PS, Gomm S, Blair V, Thatcher N. Pulmonary carcinoid tumours: a clinico-pathological study of 35 cases. Br J Cancer 1986;54:963–7.

    In an analysis of 35 bronchial carcinoid tumors, mitotic count, necrosis, nuclear pleomorphism, vascular and lymphatic permeation, and diffentiation were histologic prognostic variables. Immunocytochemical staining did not help in assessing prognosis.

Pathologic Review

24. Travis WD, Linnoila RI, Tsokos MG, et al. Neuroendocrine tumors of the lung with proposed criteria for large-cell neuroendocrine carcinoma. Am J Surg Pathol 1991;15:529–53.

    Thirty-five cases of pulmonary endocrine tumors from the National Institutes of Health were characterized, with specific attention to the large-cell neuroendocrine carcinomas. All of the presently accepted classifications for atypical carcinoids are reviewed, as well as an excellent discussion of immunohistochemical and electron and light microscopic features.

Review Series

Specific Terminology—"Bronchial Adenomas"

25. Burcharth F, Axelsson C. Bronchial adenomas. Thorax 1972;27:442–9.

    Twenty-six patients from 1956 to 1971 (23 with carcinoids, 2 mucoepidermoid, 1 cylindroma) are reported, with 7 of 26 having metastases. Five-year survival for the patients with carcinoids was a disappointing 56%, and this was one of the first articles to point out the malignant nature of these tumors.

26. Goldstraw P, Lamb D, McCormack RJ, Walbaum PR. The malignancy of bronchial adenoma. J Thorac Cardiovasc Surg 1976;72:309–14.

    Seventy-nine cases (71 carcinoid, 7 adenoid cystic, 1 mucoepidermoid) between 1946 and 1976 were reviewed. Carcinoids exhibited the least malignant behavior, whereas the adenoid cystic tumors were of intermediate position in frequency and malignancy.

27. Lawson RM, Ramanathan L, Hurley G, Hinson KW, Lennox SC. Bronchial adenoma: review of 18-year experience at the Brompton Hospital. Thorax 1976;31:245–53.

    This is a classic British review from 1955 to 1972 of 72 patients with excellent description of symptoms, plain radiographic findings, and conservative resection, advocating lobectomy and lymph node dissection. The actuarial survival for patients who were operated on (71) was 75% at 15 years.

28. Attar S, Miller JE, Hankins J, et al. Bronchial adenoma: a review of 51 patients. Ann Thorac Surg 1985;40:126–32.

    Forty-three carcinoids, 5 adenoid cystic carcinomas, 2 mixed, and 1 mucoepidermoid carcinoma are reported, with subclassification of the carcinoids into typical and atypical. Surgical resection resulted in 88% 10-year survival for patients with typical carcinoids and 59% for those with atypical carcinoids.

29. Alp M, Ucanok K, Dogan R, et al. Surgical treatment of bronchial adenomas: results of 29 cases and review of the literature. Thorac Cardiovasc Surg 1987;35:290–4.

    This Turkish series of 29 resected bronchial adenomas emphasizes conservative (bronchotomy excision, 7; sleeve resection, 4; lobectomy, 12; segment and lobe, 1; pneumonectomy, 5) with a reported disease-free and overall survival of 96% at 5 years. Only 1 patient had lymph node metastases.

30. Su WJ, Lee YC, Perng RP. Bronchial adenoma—a heterogenous group of tumors: report of 38 patients. Chung Hua I Huseh Tsa Chih 1990;45:174–80.

    From 1976 to 1988, 38 bronchial adenomas (9 carcinoid, 14 mucoepidermoid, 15 cylindromas) all showed malignant potential, with 5-year survivals of 56%, 11%, and 83%, respectively. This is a good series for discussion of noncarcinoid bronchial adenomas.

Specific Terminology—"Bronchial Carcinoid Tumors"

31. Todd TR, Cooper JD, Weissberg D, Delarue NC, Pearson FG. Bronchial carcinoid tumors. Twenty years' experience. J Thorac Cardiovasc Surg 1980;79:532–6.

    Sixty-seven carcinoids, seven with regional metastases, were resected, chiefly by lobectomy or sleeve resection, with less than 1% recurrence at the time of publication. Indications for biopsy and risks are discussed.

32. Brenez D, Gonette L, Francis C, et al. The bronchial carcinoid adenoma. A clinical, radiological and anatomopathological study of 25 operated patients. Acta Clinica Belgica 1984;39: 18–26.

    This is an excellent discussion of endoscopic biopsy methods.

33. Wilkins JR Jr, Grillo HC, Moncure AC, Scannell JG. Changing times in surgical management of bronchopulmonary carcinoid tumor. Ann Thorac Surg 1984;38:339–44.

    This is one of the largest surgical series (111 with 11 atypical carcinoids), emphasizing a change over time toward more conservative resections and maintaining an 82% 10-year survival. There is stimulating discussion at the end of the article.

34. Brandt B III, Heintz SE, Rose EF, Ehrenhaft JL. Bronchial carcinoid tumors. Ann Thorac Surg 1984;38:64–5.

    Six of 46 resected carcinoids had lymph node metastases, but this did not influence long-term survival. There is no attempt at subclassification of carcinoids and an emphasis on "standard surgical resection" to accomplish a 90% "cure" rate.

35. Hurt R, Bates M. Carcinoid tumours of the bronchus: a 33 year experience. Thorax 1984;39:617–23.

    A 33-year experience with 79 cases emphasized use of bronchotomy and sleeve resection (n = 9, all without recurrence), but there was only one atypical carcinoid. The survival rates were 94% at 10 years, 80% at 15 years, and 64% at 25 years.

36. Bertelsen S, Aasted A, Lund C, et al. Bronchial carcinoid tumours. A clinicopathologic study of 82 cases. Scand J Thorac Cardiovasc Surg 1985;19:105–11.

    A retrospective clinicopathologic review of 82 cases found 65 to be typical carcinoids of which two had metastases. Among the 17 others, ten had regional nodal metastases. Conservative resection, including lymph node removal, was recommended for the typical carcinoids.

37. Mårtensson H, Böttcher G, Hambraeus G, Sundler F, Willen H, Nobin A. Bronchial carcinoids: an analysis of 91 cases. World J Surg 1987;11:356–64.

    This 30-year review of 91 cases had an emphasis on immunocytochemical analysis of the specimens, despite only 1 patient with endocrine symptoms. Only one tumor had lymph node metastases.

38. Rea F, Binda R, Spreafico G, et al. Bronchial carcinoids: a review of 60 patients. Ann Thorac Surg 1989;47:412–4.

    Sixty patients had a 10-year survival of 90% for those with typical carcinoids, whereas that for patients with atypical carcinoids was 60%. All patients with lymph node involvement died within 5 years. A more aggressive approach to the atypical carcinoids is suggested.

39. Toledo J, Roca R, Anton JA, et al. Conservative and bronchoplastic resection for bronchial carcinoid tumours. Eur J Cardiothorac Surg 1989;3:288–91.

    This series emphasizes local, conservative measures including bronchotomy, segmentectomy, enucleation, and wedge resection for carcinoid management in 50% of the 45 cases without local recurrence on follow-up.

40. Stamatis G, Freitag L, Greschuchna D. Limited and radical resection for tracheal and bronchopulmonary carcinoid tumour. Report on 227 cases. Eur J Cardiothorac Surg 1990;4: 527–32.

    This is a gigantic series of 227 patients over a 20-year period with an astounding 0.44% operative mortality. Patients with typical carcinoids had a 98% 5-year survival compared with 41% for those with atypical. Surgical management depending on the histologic aspects of the tumor is emphasized.

41. Chapleau D, Page A, Verdant A, Beauchamp G, Kakhle G. Bronchial carcinoids: long-term prognostic factors. Can J Surg 1991;34:111–4.

    Prognostic factors for enhanced survival included peripheral location, and there was an association between transbronchial invasion and lymph node metastases in 41 resected patients.

Nd:YAG Laser Management

42. Diaz-Jimenez JP, Canela-Cardona M, Maestre-Alcacer J. Nd: YAG laser photoresection of low-grade malignant tumors of the tracheobronchial tree. Chest 1990;97:920–2.

    Nineteen patients (14 with carcinoid, 5 with adenocystic carcinoma) had laser ablation; two carcinoids had massive hemoptysis.

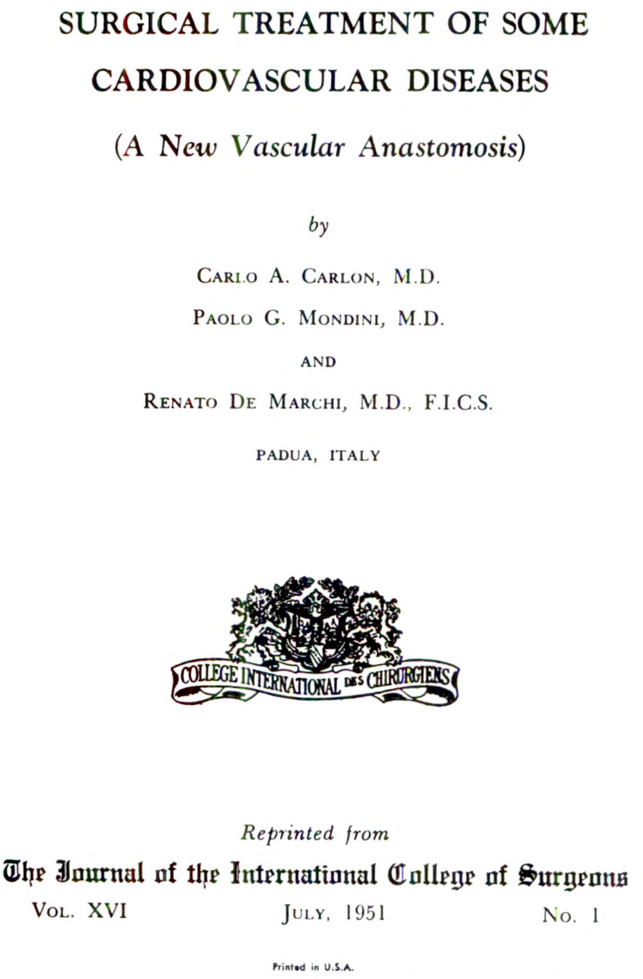
CORRESPONDENCE

Carlo A. Carlon and the Cavopulmonary Anastomosis

To the Editor:

Anastomosis between the superior vena cava and the right pulmonary artery has been devised to increase the pulmonary blood flow in congenital heart malformations characterized by pulmonary hypoperfusion. The problem of oxygenation of the pulmonary blood under venous pressure and without the contribution of the right cardiac output was first studied and applied experimentally by Carlon, Mondini, and de Marchi, at the University of Padova, by creating a cavopulmonary anastomosis as a means to increase pulmonary blood flow in certain congenital cardiac anomalies. The results of these investigations were published in 1950 [1] and 1951 [2] (Fig 1). The concept was based on the consideration that venous blood returned "to the right side of the heart because of vis a tergo and thoracic suction during respiratory movements," being not significantly influenced by the suction of the right atrium. Thus, diversion of blood from the superior vena cava to the right pulmonary artery was obtained by a new vascular anastomosis that was proposed by these authors as an alternative to the classic Blalock-Taussig shunt [1].

Direct end-to-end anastomosis between the superior vena cava and right pulmonary artery was initially unsuccessful because dogs could not tolerate the sudden decrease in cardiac output due to acute closure of the superior vena cava. Therefore, the surgical technique was slightly modified allowing a successful outcome. Essential surgical steps included (1) a right thoracotomy in the fourth intercostal space; (2) identification, isolation, and mobilization of the superior vena cava, azygos vein, and right pulmonary artery; (3) ligation and division of the azygos vein and right pulmonary artery; and (4) end-to-end anastomosis between the distal right pulmonary artery and proximal end of the azygos vein (Fig 2).



SURGICAL TREATMENT OF SOME CARDIOVASCULAR DISEASES

(*A New Vascular Anastomosis*)

by

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AND

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Fig 1. Title page of the report by Carlon and associates [2], describing for the first time in the English-language literature the cavopulmonary anastomosis. (Reproduced with permission.)

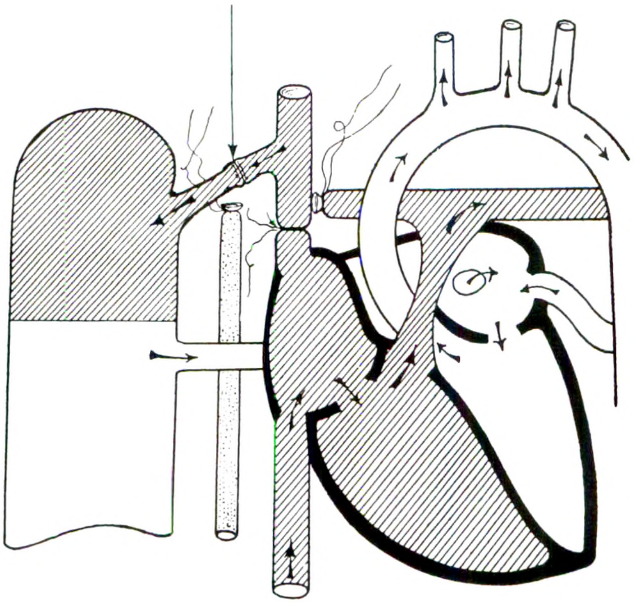

Fig 2. Final step of the cavopulmonary anastomosis as originally described by Carlon, Mondini, and de Marchi. (Reproduced with permission from Carlon CA, Mondini PG, de Marchi R. Su una nuova anastomosi vasale per la terapia chirurgica di alcuni vizi cardiovascolari. Giorn It Chir 1950;6:760–74.)

The cavopulmonary anastomosis, developed in the early 1950s, was subsequently popularized by others [3]. As pointed out by Robicsek, in his excellent and comprehensive review on this subject [4], the pioneering work of Carlo A. Carlon and his group has seldom received adequate credit, probably because the results of their work were mostly published in Italian journals with limited circulation and are rarely available to the foreign surgical population [1, 5].

Carlo A. Carlon died on March 7, 1991, at the age of 71 years. Today, now that the cavopulmonary anastomosis has partly regained its popularity and has acquired a definite place in the surgical correction of complex cardiac anomalies [6], it seems appropriate to recognize the fundamental contribution to cardiac surgery of the surgeon who first demonstrated experimentally the feasibility and effectiveness of this procedure.

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2. Carlon CA, Mondini PG, De Marchi R. Surgical treatment of some cardiovascular diseases (a new vascular anastomosis). J Int Coll Surg 1951;15:1–11.
3. Glenn WWL. Circulatory bypass of the right side of the heart. IV. Shunt between the superior vena cava and distal right pulmonary artery: report of clinical application. N Engl J Med 1958;259:117–20.
4. Robicsek F. An epitaph for cavopulmonary anastomosis. Ann Thorac Surg 1981;34:200–20.
5. Carlon CA, Albertini B. Su un accorgimento di tecnica nella anastomosi fra vena cava superiore e ramo destro dell'arteria polmonare per la terapia della trasposizione dei grandi vasi. Boll Soc Triveneta Chir 1952;6:1–8.
6. de Leval M, Kilner P, Gewillig M, Bull C. Total cavopulmonary connection: a logical alternative to atriopulmonary connection for complex Fontan operations. Experimental studies and early clinical experience. J Thorac Cardiovasc Surg 1988; 96:682–95.

Penetrating Cardiac Injuries

To the Editor:

Attar and colleagues [1] present an elegant series of patients with penetrating cardiac injuries. I have only one problem with their presentation, and that is their mention that "pericardial window was performed in 10 patients." Do the authors really mean a "window"? Too often, tube drainage of the pericardium is reported as a "window"—an operation that creates an opening between the pericardium and the left (rarely the right) pleura to drain resistant pericardial effusions [2, 3] and is not always ultimately successful [4].

Attar and colleagues are not alone in their terminology; I do not know why tube drainage of the pericardium is so often referred to as a "window." Surely, no other major cavity is drained by a window. Somehow, for the pericardium, the terms became confused in many physicians' minds by slipshod extension. (Of course, if I inferred wrongly and Attar and colleagues did produce a true window, that would have been unusual under the conditions of acute trauma.)

Terminology is only a quibble unless it serves to enlighten or to obscure facts and concepts [5]. In this case, the reader should be apprised of exactly what was done. We should reserve the term *pericardial window* for its original, precise meaning. If pericardial tube drainage (as it has often been described) has become a "window," how should we rename the fenestration of pericardium with pleura?

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Reply

To the Editor:

Dr Spodick questioned the meaning of the term *pericardial window* performed in 10 patients with penetrating cardiac injuries. We used this term to denote opening the pericardium through the subxiphoid approach to detect blood in the pericardial sac. If blood was detected, sternotomy was performed and the bleeding site controlled. If the exploration was negative, the implication was that penetration of the cardiac chambers had not occurred and further surgical intervention was not necessary; the pericardium was not drained in this situation.

In more recent times we have rarely used subxiphoid windows in acute trauma. However, we still find the procedure useful in cases of multiple trauma when the patient becomes hemodynamically unstable during operation in the abdomen or on the extremities.

We continue to use this procedure in cases of pericardial effusion of unknown etiology, in uremia, and in cases of malignant pericardial effusion with pericardial tamponade. In these situations, the pericardial window can be diagnostic as well as therapeutic.

Finally, we occasionally perform a pericardial window through a thoracotomy incision to drain the pericardium into the pleural cavity. Our experience with this has not been as rewarding as that with the more extensive pericardiectomy.

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Mammary Artery to Saphenous Vein Bypass Graft: An Alternative Method of Redo Operation

To the Editor:

A patient underwent redo coronary artery revascularization that included grafting of the left internal mammary artery to the stump of a saphenous vein graft to the left anterior descending artery, which was occluded. The procedure was done without cardiopulmonary bypass. Because of the simplicity of the procedure we describe its details.

A 49-year-old man underwent double aortocoronary bypass grafting and radical pericardiectomy in 1983. He did well initially, but returned in 1990 (aged 56 years) with incapacitating angina. Thallium scanning showed reversible changes in the distribution of the anterior wall. Angiography revealed a grade III ventricle with anterolateral and inferobasal hypokinesis. The vein graft to the obtuse marginal artery of the circumflex was patent. The native left anterior descending artery was occluded and filled the stump of a vein graft through collateral circulation. The artery was small and diseased.

At operation, dense adhesions were found. The left internal mammary artery was dissected and an end-to-side anastomosis was performed to the remnant of the saphenous vein without

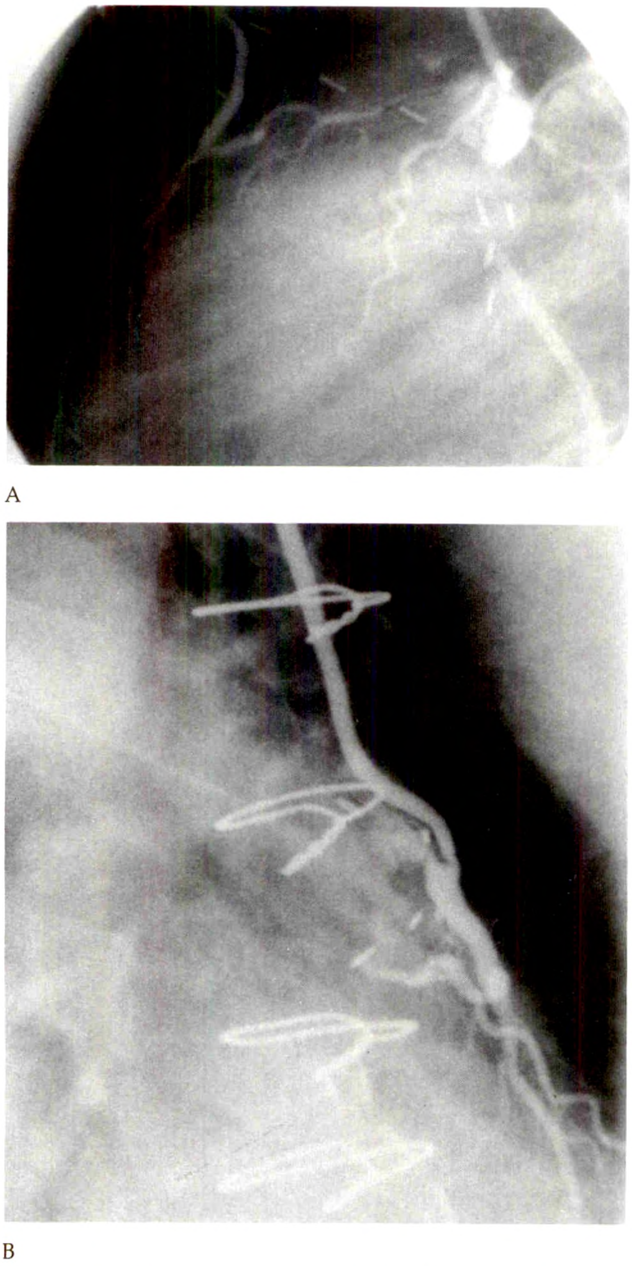

Fig 1. Follow-up angiography 6 weeks postoperatively showed a patent left internal mammary artery graft to the remnant of the saphenous vein bypass graft.

cardiopulmonary bypass. The patient did well postoperatively, and he was discharged home 5 days postoperatively. Follow-up angiography 6 weeks later (Fig 1) showed a patent left internal mammary artery. This technique has been used successfully in 2 additional patients, but the angiograms are pending.

Dense adhesions in patients with calcific pericarditis make redo revascularization operations even more difficult. Patients with advanced coronary artery disease, whose grafts may have occluded, may not be suitable candidates for conventional redo operation.

In this case report, the vein graft to the obtuse marginal artery was patent but the left anterior descending artery graft had occluded proximally. Previous radical pericardiectomy posed an additional challenge. Anastomosis of the left internal mammary artery to the side of the vein stump provided good revascularization without the need for dissection of the heart, heparinization, and cardiopulmonary bypass.

Although we cannot recomend this technique for routine redo operation, it may provide an alternative method in select cases. The long-term result of this type of revascularization remains to be determined.

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Surgical Treatment of Ebstein's Malformation

To the Editor:

I greatly enjoyed the paper entitled "Christiaan Barnard's Contribution to the Surgical Treatment of Ebstein's Malformation" and the accompanying short commentary by Professor Barnard published in the January 1991 issue of *The Annals of Thoracic Surgery* [1]. I congratulate the author and thank you for publishing this enjoyable and stimulating article, which gave this rather rare congenital anomaly an important airing. The controversy regarding the optimal treatment of Ebstein's anomaly will perhaps continue until long-term results of reconstructive operations for the tricuspid valve become established. Our views on the topic were expressed in 1984 when A. Blesovsky and I published our 16 years' experience of tricuspid valve replacement without plication of the atrialized portion of right ventricle for Ebstein's anomaly [2].

The purpose of this letter, however, is not to get involved in or to revive the existing controversy but to highlight the events in our first patient, which strangely coincided with the publication of Dr Kirklin's paper, and to credit Professor Barnard for his original concept, which has given us excellent long-term results in our patients. Just about the time when Professor Barnard was visiting his Cardiac Clinic to find out what happened to his first patient, I happened to have an exciting experience with our first patient. This patient was operated on in 1967; under normothermic perfusion with a beating heart she underwent closure of interatrial communication and tricuspid valve replacement with a Hammersmith valve prosthesis in accordance with the method described by Barnard and Schrire [3]. The patient at that time was 17 years old and had cyanosis, dyspnea, and congestive cardiac failure due to Ebstein's malformation. She was in sinus rhythm and had a grade IV disability according to the New York Heart Association classification. The operation was performed by my mentor and predecessor Mr A. Blesovsky, interestingly, another South African.

The patient had no postoperative complication. She remained in sinus rhythm, her cardiothoracic ratio came down gradually from 0.57 to 0.50, and her clinical status gradually improved to New York Heart Association class I. She was anticoagulated with warfarin and later on had early abortions on two occasions. The third pregnancy, however, was managed by replacing warfarin with subcutaneous heparin, and it ended successfully with the birth of a healthy child at full term. The patient was followed up regularly at yearly intervals.

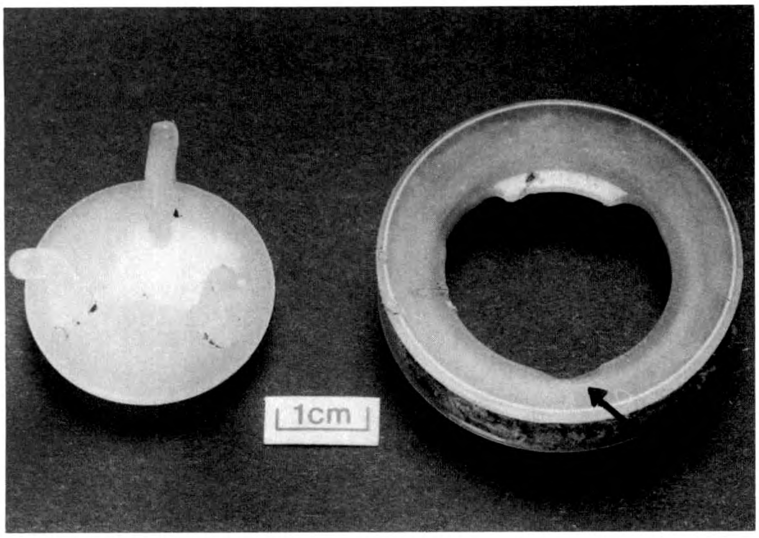

Fig 1. Hammersmith prosthesis: ring and disc. The arrow indicates the site of wear.

In December 1990 her condition suddenly deteriorated, she became dyspneic and listless, and she also started having palpitations. She volunteered an interesting observation of not being able to hear the clicking of the prosthetic valve. Clinically she had signs of mild tricuspid stenosis with gross regurgitation, and prosthetic clicks were not audible. Echocardiographic examination suggested a thrombosed prosthesis causing stenosis with regurgitation. Chest roentgenogram showed mild cardiomegaly.

On January 17, 1991, she underwent reoperation. After institution of routine cardiopulmonary bypass, a vent was placed in the left ventricle through the right superior pulmonary vein. At normothermia and with the heart beating, the right atrium was opened. To my amazement I could not see the disc in the prosthetic ring. There was no clot or thrombus in the right atrial cavity. I removed the prosthetic ring but could not find the disc in the right ventricular cavity. Palpation of the main pulmonary artery revealed the presence of a hard object which, when removed after pulmonary arteriotomy, was found to be the disc of the Hammersmith valve. The disc was embedded in the main pulmonary artery with mild fibrous reaction around it. It is unlikely that it was dislodged during initial manipulation and cannulation of the heart. Pulmonary arteriotomy was closed with 6-0 continuous Prolene (Ethicon, Somerville, NJ). A size 33 Carbomedics mechanical prosthesis was sutured in place with sixteen interrupted 2-0 Ethibond (Ethicon) sutures. The patient made an uneventful postoperative recovery and was discharged from the hospital on January 28, 1991. She was reviewed 6 weeks later when her condition was satisfactory. She was in sinus rhythm with a substantial improvement in her symptoms at this early stage.

The preoperative echocardiograms were reviewed without revealing operative details. The issue of when the disc actually slipped out of the ring, although now of academic interest only, could not be resolved. However, inspection of the prosthesis (Fig 1) shows, as observed previously [4], wear of the polypropylene material at the site of the contact between the short leg of the disc and the valve ring from where the disc escapes very easily.

Clearly, the long-term surgical results of tricuspid valve replacement in Ebstein's anomaly in careful hands are most gratifying. It is equally interesting that the Hammersmith valve, which is now obsolete, has given 24 years of good life to this patient. Perhaps the choice of the prosthesis, when replacing the tricuspid valve, does not make much difference in achieving good long-term results.

P. Raj Behl, MS, FRCS

Regional Cardiothoracic Centre
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Freeman Rd
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Gastric Emptying After Gastric Pull-up

To the Editor:

We read with interest the paper by Morton and associates [1], who used radionuclide transit studies to evaluate function of the interposed stomach after total esophagectomy in 14 patients. Although we concur with their results, that gastric emptying is dependent on position, studies performed at our institution on 35 patients reconstructed in a similar manner [2] have indicated the following additional findings: (1) patterns of spontaneous emptying of the vagotomized intrathoracic stomach are complex, and mean percentage radionuclide clearances at 30 minutes are comparable with emptying of the normal intraabdominal stomach; (2) there appears to be very little correlation between subjective symptoms (dysphagia, early satiety, reflux) and objective findings as measured by radionuclide transit studies; and (3) attempts at modifying function of the interposed stomach using the gastric prokinetic domperidone (Motilium; Janssen Pharmaceutica) appeared to have little objective efficacy, although symptomatic improvement was reported [3]. Our data suggest that the interposed stomach appears to retain its gastric identity rather than acting as an inert conduit and remains a satisfactory method of reconstruction after esophagectomy for both benign and malignant esophageal disease [4].

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Richard Inculet, MD
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2. Powe JE, Casson AG, Inculet RI, Laurin N, Finley RJ. Functional evaluation of gastric interposition following total esophagectomy. J Nucl Med 1990;31(Suppl):775.
3. Casson AG, Powe JE, Inculet RI, Finley R. Efficacy of domperidone (Motilium) on modifying function of the interposed stomach following total esophagectomy. Gastroenterology 1989;98(Suppl):A335.
4. Casson AG, Powe J, Inculet R, Finley R. Functional results of gastric interposition following total esophagectomy. Clin Nucl Med (in press).

Surgical Management of Carcinoid Heart Disease

To the Editor:

In their recent report on the surgical management of carcinoid heart disease, Fetherston and Davis [1] suggest that mechanical tricuspid valve replacement be performed in patients with the carcinoid syndrome because of the "theoretical potential" for carcinoid heart disease to recur on bioprosthetic valve leaflets. Recently, we have reported just such a case [2], in which recurrent carcinoid plaque on a Hancock porcine prosthesis contributed to failure of the valve and eventually led to reoperation. We also are aware of at least one postmortem study demonstrating that carcinoid plaque can develop on bioprosthetic valves [3]. We therefore agree with the recommendations of Fetherston and Davis that mechanical tricuspid valve replacement be considered in patients with carcinoid heart disease, particularly for those individuals at low risk for anticoagulation failure.

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References

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3. Schoen FJ, Hausner RJ, Howell JF, Beazly HL, Titus JL. Porcine heterograft valve replacement in carcinoid heart disease. J Thorac Cardiovasc Surg 1981;81:100–5.

Thermodilution Catheter-Induced Endobronchial Hemorrhage With Pulmonary Hypertension

To the Editor:

Various techniques have been described to control severe endobronchial hemorrhage caused by Swan-Ganz catheter injury to the distal pulmonary artery. The method of endobronchial blockade under direct vision described by Dr Purut and his colleagues [1] is a precise and reliable technique in patients with normal pulmonary artery pressure undergoing coronary artery operation. However, in patients with pulmonary hypertension this technique should be employed with great caution.

We recently performed a reoperation of mitral valve replacement and tricuspid valve repair on a 78-year-old woman with preoperative pulmonary artery pressure of 60/30 mm Hg. Blood issuing through the endotracheal tube was noticed immediately after operation. A chest roentgenogram showed a triangular shadow of segmental consolidation on the left side. Bronchoscopy was performed, and the bleeding from the lingular segment of the left upper lobe was arrested using a Fogarty balloon catheter. Postoperatively, systolic pulmonary artery pressure was maintained less than 40 mm Hg. The patient's hemodynamic status deteriorated in the first 24 hours after operation, and a chest roentgenogram showed increasing opacity on the left side. A chest drain was inserted, and about 2,000 mL of blood was drained. The patient was reoperated on for left upper lobectomy as the bleeding continued. The patient died 48 hours postoperation due to severely compromised hemodynamic status.

In patients with pulmonary hypertension, endobronchial blockade can lead to increased intralobar pressure due to continued bleeding and eventual rupture of the lobe. Alternate methods such as selective embolization of the bleeding artery or local injection of vasoconstrictors through the bronchoscope may be of value in this situation to avoid this fatal complication.

Pasala S. Ravichandran, MD
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Jeffrey S. Swanson, MD
Cindy L. Fessler, BS
Albert Starr, MD

The Heart Institute at St. Vincent Hospital and Medical Center
9155 SW Barnes Rd, Suite 236
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Reference

1. Purut CM, Scott SM, Parham JV, Smith PK. Intraoperative management of severe endobronchial hemorrhage. Ann Thorac Surg 1991;51:304–7.

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

■ Fifty-seventh Annual Scientific Assembly, American College of Chest Physicians, San Francisco, California—November 4–8, 1991

For information on this meeting, contact the American College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068; or telephone (708) 698-2200.

■ Thirty-eighth Annual Meeting of the Southern Thoracic Surgical Association, Orlando, Florida—November 7–9, 1991

For information on this meeting, contact Hendrick B. Barner, MD, Southern Thoracic Surgical Association, 401 N Michigan Ave, Chicago, IL 60611-4267; or telephone (312) 644-6610 (facsimile: (312) 321-6869).

■ Third Combined Meeting of the Nederlandse Vereniging Voor Thoraxchirurgie and the Deutsche Gesellschaft für Thorax-, Herz- und Gefäßchirurgie, Maastricht, the Netherlands—November 7–9, 1991

For information on this meeting, contact Organizing Secretariat, Conference Agency Limburg, Mrs Brigitte Bessems, c/o University Hospital Maastricht, PO Box 1918, 6201 BX Maastricht, the Netherlands; or telephone 31 (0)43 258344 (facsimile: 31 (0)43 258355).

■ Sixth World Conference on Lung Cancer, Melbourne, Australia—November 10–14, 1991

For information on this meeting, contact Ms Jane Willis, MCS Convention Services, PO Box 335, Heidelberg, Vic 3079, Australia; or telephone (613) 499-6722 (facsimile: (613) 499-7137).

■ Sixty-fourth Scientific Session of the American Heart Association, Anaheim, California—November 11–14, 1991

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 706-1253.

■ Circulatory Support 1991, San Francisco, California—November 15–17, 1991

For information on this meeting, contact The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

■ Twenty-eighth Annual Meeting of The Society of Thoracic Surgeons, Orlando, Florida—February 3–5, 1992

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

■ Second International Lung Transplant Symposium—Update 1991, Laguna Niguel, California—February 12–14, 1992

This symposium is sponsored by Cedars-Sinai Medical Center. For information on this meeting, contact Department of Continuing Medical Education, Cedars-Sinai Medical Center, at (213) 855-5547.

■ First International Symposium on Diseases of the Aorta, Tokyo, Japan—February 24–26, 1992

For information on this meeting, contact Japan Convention Services, Inc, Nippon Press Center Building, 2-1, 2-chome, Uchisaiwai-cho, Chiyoda-ku, Tokyo 100, Japan.

■ American College of Surgeons, Orlando, Florida—March 22–25, 1992

For information on this meeting, contact American College of Surgeons, 55 Erie St, Chicago, IL 60611; or telephone (312) 664-4050.

■ Twelfth Annual Meeting of the International Society for Heart Transplantation, San Diego, California—April 2–4, 1992

For information on this meeting, contact ISHT, 435 N Michigan Ave, Suite 1717, Chicago, IL 60611; or telephone (312) 644-0826.

■ Forty-first Annual Meeting of the American College of Cardiology, Dallas, Texas—April 12–16, 1992

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

■ Seventy-second Annual Meeting of the American Association for Thoracic Surgery, Los Angeles, California—April 27–29, 1992

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Fourteenth World Congress of The International Society for Heart Research, Kobe, Japan—May 10–14, 1992

For information on this meeting, contact Makoto Nagano, MD, Secretariat, XIV World Congress of ISHR, Department of Internal Medicine, Jikel University Medical School, Aoto Hospital, 6-41-2 Aoto, Katsushika-ku, Tokyo 125, Japan.

■ The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Chicago, Illinois—June 8–10, 1992

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Eighteenth Annual Meeting of The Western Thoracic Surgical Association, Kauai, Hawaii—June 25–27, 1992

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Sixth Annual Meeting of The European Association for Cardio-Thoracic Surgery, Geneva, Switzerland—September 14–16, 1992

For information on this meeting, contact Symporg S.A., 108, route de-Frontenex, CH-1208 Geneva, Switzerland; or telephone 41-22 786 37 44 (facsimile: 41-22 786 40 80).

■ Thirty-ninth Annual Meeting of the Southern Thoracic Surgical Association, Wesley Chapel (near Tampa), Florida—November 5–7, 1992

For information on this meeting, contact Southern Thoracic Surgical Association, 401 N Michigan Ave, Chicago, IL 60611-4267; or telephone (312) 644-6610 (facsimile: (312) 321-6869).

CLASSIFIED ADVERTISEMENTS

Classified advertisements for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Art Carlucci, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010 (telephone: (212) 633-3806; facsimile: (212) 633-3820). All advertisements are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.50 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $18.00 per insertion for box number advertisements.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Advertisement orders should be for a maximum of 6 months; advertisements may be renewed for up to 6 months at a time. Advertisement copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Advertisements, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3853). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the advertisement. To respond to an anonymous advertisement, write to Box Number, *The Annals of Thoracic Surgery*, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Possible academic affiliation. Please respond with CV.

Please respond to Box 404. 404I/C

Cardiovascular and thoracic surgeon experienced in adult and pediatric cardiac surgery as well as general thoracic surgery needed to join the practice of an established cardiovascular and thoracic surgeon. Send curriculum vitae and details of experience.

Please respond to Box 541. 541F/K

Cardiothoracic and vascular surgeon, board eligible or certified, to join 2-man private practice in Ft. Lauderdale, Florida. Send CV.

Please respond to Box 542. 542F/K

Cardiothoracic surgeon, BC/BE, to join rapidly expanding single-hospital practice in Chicago area. Practice is 90% adult cardiac, 10% vascular with potential for general thoracic procedures. Generous salary leading to partnership. Send curriculum vitae.

Please respond to Box 545. 545F/K

Cardiovascular and thoracic surgeon, BE/BC, wanted to join an established expanding private practice in the Northeast with an academic affiliation. Must have experience in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership.

Please respond to Box 554. 554G/L

Cardiovascular surgeon, board certified or board eligible in thoracic surgery. Large surgical practice in western Pennsylvania. Excellent compensation and benefits. Please send CV and references.

Please respond to Box 555. 555G/L

University of Arizona. Expanding university program creates exciting opportunities for new faculty, both academic (tenure) and clinical tracks. Applicants must be interested in academic career. Interested particularly in cardiac, obstetric, ICU, and pediatric subspecialties. An AA/EOE.

Contact Burnell R. Brown, Jr, MD, PhD, Department of Anesthesiology, Arizona Health Sciences Center, Tucson, AZ 85724. Closing date: 03/31/92 or until position is filled. 560G/L

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join 2-man surgical group in the Southeast. 1–2 years of experience preferred. Please respond with curriculum vitae.

Please respond to Box 562. 562G/L

Cardiothoracic surgeon, recent graduate or no more than 2 years out of training, to join established group in a major Texas city. Practice includes adult and some pediatric cardiac, electrophysiologic, thoracic, and peripheral vascular surgery. Please respond with CV.

Please respond to Box 564. 564G/L

Adjunct staff surgeon—the Department of Surgery of the Deborah Health and Lung Center invites applications for the position of adjunct staff surgeon for the Division of Cardiac Surgery. This opportunity is appropriate for a surgeon having completed his/her cardiac surgery training and desiring further operative experience in the setting of a busy and varied clinical practice.

Please send curriculum vitae to Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart and Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015. 567G/K

BC/BE cardiovascular and thoracic surgeon wanted to join busy two-member group in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308 [(305) 561-9781 after 9:00 PM]. 570H/A

Cardiac surgeon needed immediately for position on East Coast. Prefer 1–3 years' experience in all aspects of adult cardiac surgery. Salary with early partnership. Reply with curriculum vitae.

Please respond to Box 572. 572H/K

Cardiovascular and thoracic surgeon to join established 3-man private practice in mid-South. Practice includes thoracic, cardiac, and vascular surgery. Must be board certified or eligible in thoracic and cardiovascular surgery. Prefer candidate who has recently completed his/her training. Please respond with curriculum vitae.

Please respond to Box 580. 580H/A

Wanted—thoracic surgeon, BE/BC, to join a board-certified surgeon for noncardiac thoracic surgery and peripheral vascular surgery in a growing medical center in upstate New York.

Please respond to Box 588. 588I/K

Cardiothoracic surgeon—metropolitan Kansas City area: An exceptional opportunity exists for BC/BE cardiothoracic surgeon in a large, expanding, independent multispecialty group. The practice is conveniently located close to hospitals, affordable luxury housing, and nationally recognized school systems. There is a strong, diversified mix of patients with an excellent referral base. A very attractive incentive package is available including a guaranteed income. Malpractice, marketing, and relocation assistance is also available.

Please send curriculum vitae to John Storm, 51 N 12th St, Kansas City, KS 66102; (913) 281-7775, FAX (913) 281-8494. 591I/L

Cardiovascular/thoracic surgeon: A progressive major regional multispecialty group in Texas Gulf Coast area is seeking a BC/BE cardiovascular /thoracic surgeon. Applicant should be technically strong in all phases of adult cardiac surgery. Cardiology department has three cardiologists. Practice is located in subtropical climate in an upward bound economic environment. Guaranteed salary with bonus plan. Shareholder status available 12–18 months. Malpractice insurance, interview, and relocation expenses included in benefits package. Location is attractive to those who enjoy outdoor sports such as fishing, golfing, and sailing. Look into our great team!

Contact Amanda Fuhro, Valley Diagnostic Medical and Surgical Clinic, PA, 2200 Haine Dr, Harlingen, TX 78550; (512) 421-5199. 592I/L

Cardiothoracic surgeon wanted for January 1992: Expanding private practice in Philadelphia. Adult cardiac and thoracic surgery. Academic appointment. Eventual partnership.

Send CV and References to PO Box 1295, Philadelphia, PA 19105. 594I/K

Thoracic-peripheral vascular surgeon: BC/BE to join busy 32-physician multispecialty group practice in central Florida. Competitive salary incentive and benefit package with early partnership. Five-hundred–bed hospital located less than ½ mile from office. Attractive central Florida lake country community is abundant in water sports, fishing, hunting, and outdoor sports. One and one-half hours to either coast.

Please respond to Box 600. 600J/A

Cardiothoracic surgeon, BE/BC, to join busy 1-man private practice in mid-Michigan. Excellent opportunity. Salary leading to partnership. Please send CV and references.

Please respond to Box 601. 601J/L

Thoracic and vascular surgeon, BE/BC, to join active (noncardiac) thoracic and vascular practice in Pittsburgh, PA. Please respond with curriculum vitae and details of experience.

Please respond to Box 602. 602J/L

Chairman, Cardiothoracic Surgery—excellent opportunity to lead cardiothoracic department for 400+-bed tertiary care teaching center located in western New York. Excellent program affiliated with prestigious medical school. Qualified candidate should possess academic credentials and should have interest in developing clinical research.

Please contact Jeanne Dowrick, Fulton, Longshore & Associates, Inc, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 604J/L

Cardiovascular surgeon, board certified/eligible, wanted to join an established cardiovascular program in Southeast performing over 600 adult open heart operations per year. Excellent salary and benefits leading to full partnership. Please send detailed CV.

Please respond to Box 605. 605J/L

Cardiovascular and thoracic surgeon, BC/BE in thoracic surgery, to join active cardiac, thoracic, and vascular practice with academic affiliation in large Midwest city. Transplant experience desired. Reply with CV.

Please respond to Box 608. 608J/A

Cardiovascular and thoracic surgeon, board certified or board eligible, to join a rapidly expanding single hospital practice in the upper Midwest. Salary leading to partnership. Please respond with curriculum vitae.

Please respond to Box 609. 609J/C

Cardiothoracic surgeon—Northern California hospital is seeking an experienced full time cardiothoracic surgeon to direct and develop its new cardiac surgery program. Requirements include board eligibility or board certification and a minimum of 3 years' experience. This 250-bed, full-service facility anticipates 150 to 200 cases the first year.

Please respond to Box 610. 610J/C

Private practice opportunity in New York. Adult and pediatric cardiac surgery only. Postgraduate training in pediatrics a plus but will seriously consider all qualified applicants. Please respond with CV.

Please respond to Box 611. 611J/C

Cardiovascular/thoracic surgeon: Opportunity available for young surgeon to join two-man private practice doing cardiac, thoracic, pacemaker, and major vascular cases in Michigan. Early partnership. Excellent community and working conditions. Send CV and operative experience.

Please respond to Box 612. 612J/L

Cardiothoracic surgeon, BC/BE, for busy Palm Beach County CVT practice.

Send CV and references to Kenneth Begelman, MD, 1599 NW 9th Ave, Boca Raton, FL 33486. 614J/K

Cardiovascular thoracic surgeon—Board certified/board eligible and recently trained to join an established, busy, dynamic, and expanding private practice in northwest Indiana. The sphere of practice is approximately 30 minutes from downtown Chicago. Guaranteed excellent starting salary with progressive future growth and comprehensive corporate fringe benefit program.

Write including CV and details of experience and capabilities to PO Box 3066, Munster, IN 46321. 615J/L

Thoracic/vascular surgeon—Needed to join and expand active four-man group in desirable community in Ohio. (Emphasis on peripheral vascular, pulmonary, esophageal surgery and pacemakers.) Excellent opportunity for well-trained, highly motivated individual. Medical school affiliation possible. Please send curriculum vitae.

Please respond to Box 617. 617J/L

Assistant surgeon: Cardiac, thoracic, or general surgeon with cardiac experience to join well-established four-man group in the midwest. Ideal for physician who desires low-stress practice. Generous salary and fringe benefits. Please send curriculum vitae.

Please respond to Box 618. 618J/L

Cardiothoracic surgeon: Wanted to join a busy, well-established practice located in the midwest. Prefer recent graduate, must be board certified or eligible, medical school affiliation possible. Respond with curriculum vitae.

Please respond to Box 619. 619J/L

Southeast Florida! Cardiovascular surgeon needed to join an established surgeon. Do vascular, thoracic, and all of the cardiovascular surgery. For details, call Eloise Gusman, 1-800-535-7698 or send CV to PO Box 101656, Fort Worth, TX 76185. 620J/K

Excellent practice opportunity for well-mannered, technically accomplished CV surgeon in California wine country. Growing 2-surgeon practice in beautiful semirural area close to San Francisco—great family environment. Prefer young surgeon with 2–5 years' experience, ABTS board certified.

Please respond to Box 621. 621J/C

The University of South Florida seeks an Assistant/Associate Professor in thoracic surgery. Suitable candidates should have a major interest in esophageal and pulmonary surgery with dedication to research and teaching. This is an expanding program with excellent facilities including the H. Lee Moffitt Cancer Center. AA/EOE.

Please reply with curriculum vitae to Dr Peter McKeown, Director of Cardiovascular and Thoracic Surgery, The University of South Florida, Suite 730, 4 Columbia Dr, Tampa, FL 33606. 622J/K

The University of South Florida seeks an Assistant/Associate Professor in adult cardiothoracic surgery. Experience in transplantation and/or electrophysiology advantageous. Commitment to teaching and research essential. Suitable opportunity for a well-trained, highly motivated, technically skilled individual. AA/EOE.

Please reply with curriculum vitae to Dr Peter McKeown, Director of Cardiovascular and Thoracic Surgery, The University of South Florida, Suite 730, 4 Columbia Dr, Tampa, FL 33606. 623J/K

Academic cardiothoracic surgeon: The Division of Cardiothoracic Surgery at the University of Massachusetts is expanding its academic faculty. Candidates will be expected to have an interest in developing an active cardiothoracic practice, to participate in thoracic and general surgical residency training, and to take part in clinical and/or laboratory research. The University of Massachusetts is an equal opportunity employer.

Please respond with curriculum vitae to Thomas J. Vander Salm, MD, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655. 627J/K

Thoracic surgeon needed to participate in growth of thriving cardiothoracic surgical practice located in greater New York metropolitan area. Excellent opportunity for well-trained, highly motivated surgeon who is board certified or eligible. Please submit CV.

Please respond to Box 629. 629J/L

Looking for a cardiac surgeon with good thoracic and vascular experience. Ideal candidate would have training in electrophysiologic surgery; however, not a prerequisite. MD or DO acceptable. Southeastern Pennsylvania area. Early partnership offered.

Please respond to Box 630. 630K

General thoracic surgeon: The University of Rochester Division of Cardiovascular Surgery is seeking applicants for a full-time academic position in general thoracic surgery. Applicants must be BC/BE in cardiothoracic surgery. He or she will join the thoracic surgery section and must be devoted to the ongoing advancement of thoracic surgical clinical practice and academic scholarship. This position offers the individual committed to general thoracic surgery the opportunity to participate in the continued growth of academic thoracic surgical section in a comfortable and genuinely supportive environment. UR is an EEO/AA employer and represents an excellent environment for academic development.

Please respond and send curriculum vitae to Richard H. Feins, MD, Strong Memorial Hospital, 601 Elmwood Ave, Box Surgery, Rochester, NY 14642. 631K/A

Cardiovascular and thoracic surgeon: Position available for a cardiovascular and thoracic surgeon to join a 3-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association features competitive salary, excellent fringe benefits, and a goal of early partnership. Please send curriculum vitae.

Please respond to Box 634. 634K/A

Academic appointment—thoracic surgery: Queen's University, Department of Surgery, geographic full-time position for a thoracic surgeon/intensivist commencing July 1, 1992. Applicants should be licensed to practice in Ontario and be Fellows of the Royal College of Physicians and Surgeons of Canada. The successful candidate will be expected to teach at undergraduate and postgraduate levels and engage in research. He/she will be expected to have specialized training and interests in thoracic surgery and intensive care. He/she will be expected to take a proportion of emergency calls. Salary, ceiling, and academic rank will be by negotiation according to qualifications and experience. Closing date for applications is January 31, 1992. In accordance with Canadian immigration requirements this advertisement is directed to Canadian citizens and permanent residents. Queen's University has an employment equity program and encourages applications from all qualified candidates including women, aboriginal peoples, people with disabilities, and visible minorities.

Applications with a curriculum vitae and the names of three referees should be sent to Dr Charles Sorbie, Head, Department of Surgery, Queen's University, Kingston, Ontario K7L 3N6, Canada. 635K

Cardiothoracic surgeon: Immediate opening for staff surgeon at a university-affiliated VA Medical Center. Will perform cardiothoracic operations including cardiac transplantations. Will engage in cardiac-related research and will provide clinical instruction to medical students and residents in cardiothoracic surgery. Must have completed a cardiac surgery residency and have a minimum 2 years postresidency fellowship in adult cardiac surgery. Must have extensive experience in all types of open heart procedures including redos, valvular surgery, and use of IMA. Must also have research experience in cardiac surgery. Salary commensurate with education and experience.

Please contact by phone or send CV to Personnel Service, VA Medical Center, 3495 Bailey Ave, Buffalo, NY 14215. (716) 862-3643. Equal opportunity employer. Applicants are subject to random drug testing. 636K

Assistant Professor, Cardiac Surgery: The Section of Cardiac Surgery of UMDNJ-Robert Wood Johnson Medical School is seeking a highly qualified individual as an addition to the faculty at the level of Assistant Professor. This individual must have a keen interest in pursuing an academic career and must have demonstrated expertise in research.

Please send CV to Alan J. Spotnitz, MD, Department of Surgery, University of Medicine & Dentistry of New Jersey-Robert Wood Johnson Medical School, One Robert Wood Johnson Place, CN 19, New Brunswick, NJ 08903-0019. The UMDNJ is an affirmative action/equal employment opportunity employer, m/f/h/v, and a member of the University Health System of New Jersey. 637K/L

SITUATIONS WANTED

Board-certified cardiovascular surgeon, 48, university trained, wishes to relocate. Interested in group practice. CV and references upon request.

Please respond to Box 573. 573H/A

Thoracic and vascular surgeon, university trained, 37, ABS, ABTS wishes to relocate. Prefer southeastern US. CV and references upon request.

Please respond to Box 590. 590I/K

Cardiothoracic surgeon, 37, US graduate, university residencies, cardiopulmonary transplantation fellowship. Three years of busy academic adult cardiothoracic surgery practice including transplantation and arrhythmia surgery. ABS, ABTS certified. Fellow ACS, ACCP. Member STS, ISHT. Publications. Wishes to relocate, interested in private or academic group practice.

Please respond to Box 599. 599J/A

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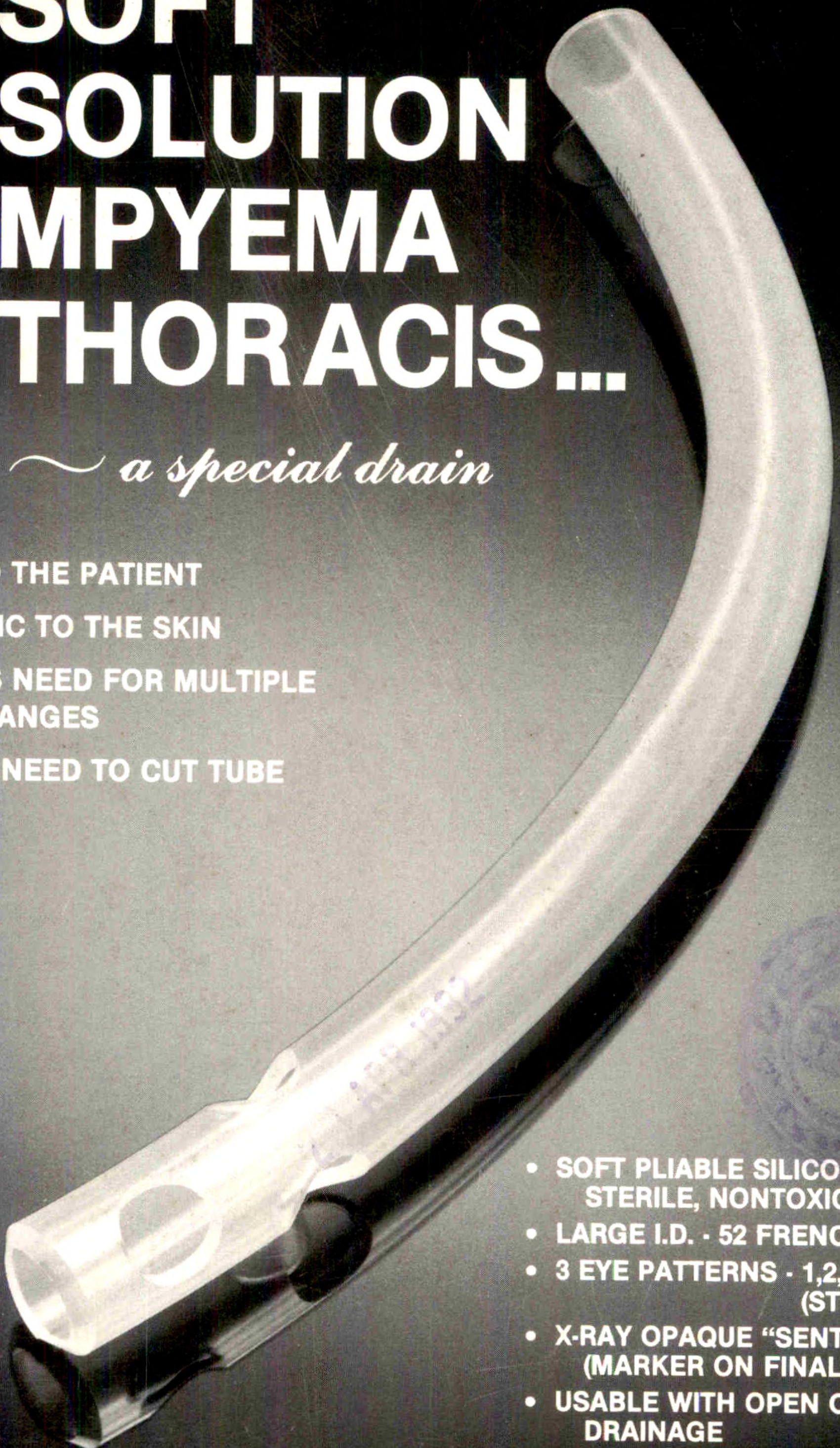

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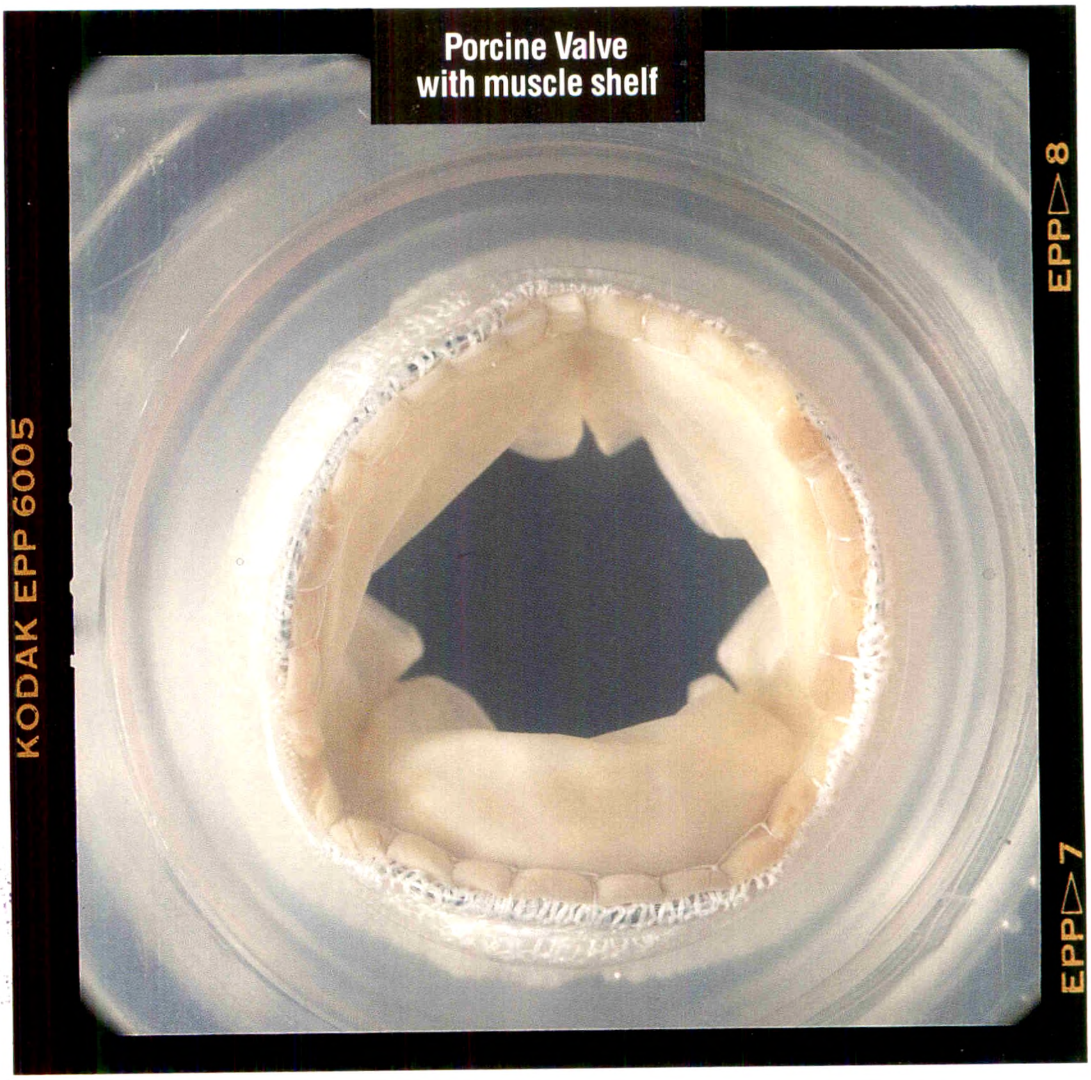

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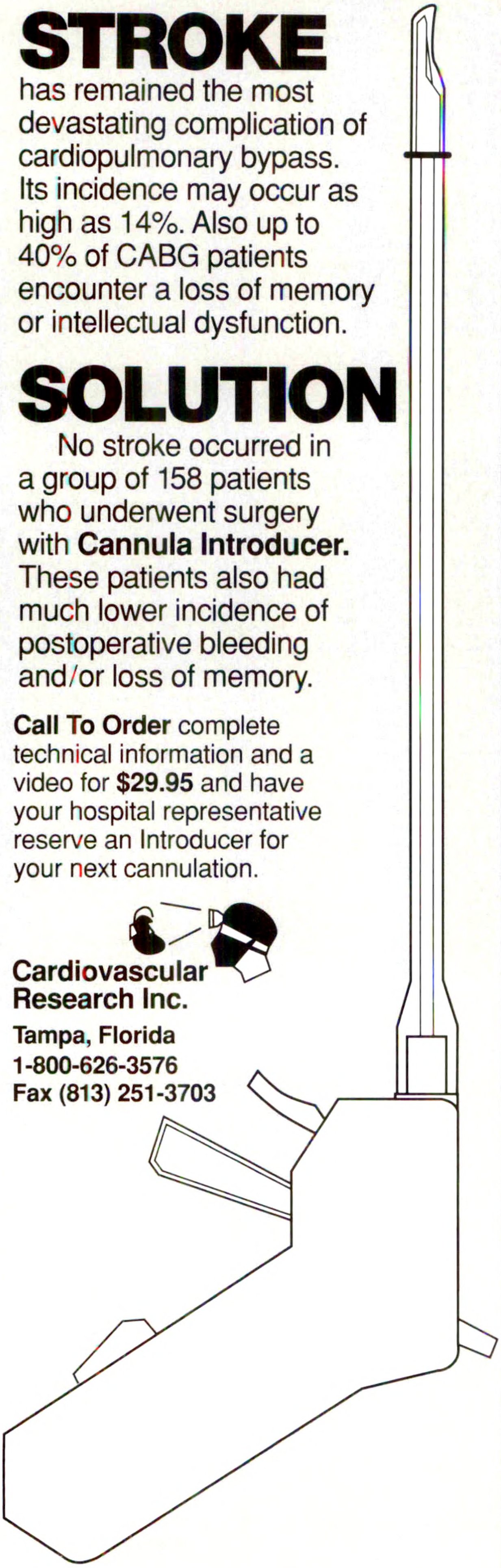

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The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient and consideration of alternative methods of treatment. Overall durability, especially long-term, has not been established for bioprostheses.

Serious complications, sometimes leading to the replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery.

Note: Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis.

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Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy (except where contraindicated) during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall, or history of previous atrial thrombus. However, the appropriate anticoagulation therapy must be determined by the physician on an individual basis.

Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc., should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result.

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Storage between 10°C and 25°C (50-77°F) is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue.

Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with the eyes, seek medical attention. For more information about glutaraldehyde exposure refer to MSDS BAX 10424, available from Edwards CVS Division.

Complications

As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days), necessitating reoperation and replacement of the prosthetic device.

Complications associated with the use of Carpentier-Edwards® bioprostheses compiled from the literature and from reports received through the complaint handling system in accordance with the United States (Federal) regulations establishing Good Manufacturing Practices, Section 820.198, include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, and malfunctions of the valve due to distortion at implant, fracture of the Elgiloy wireform, or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, degeneration, suture abrasion, instrument trauma, and leaflet detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrhythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct.

Note: Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes a potential relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier Edwards® pericardial bioprostheses to calcification as compared to commercially available bioprostheses.

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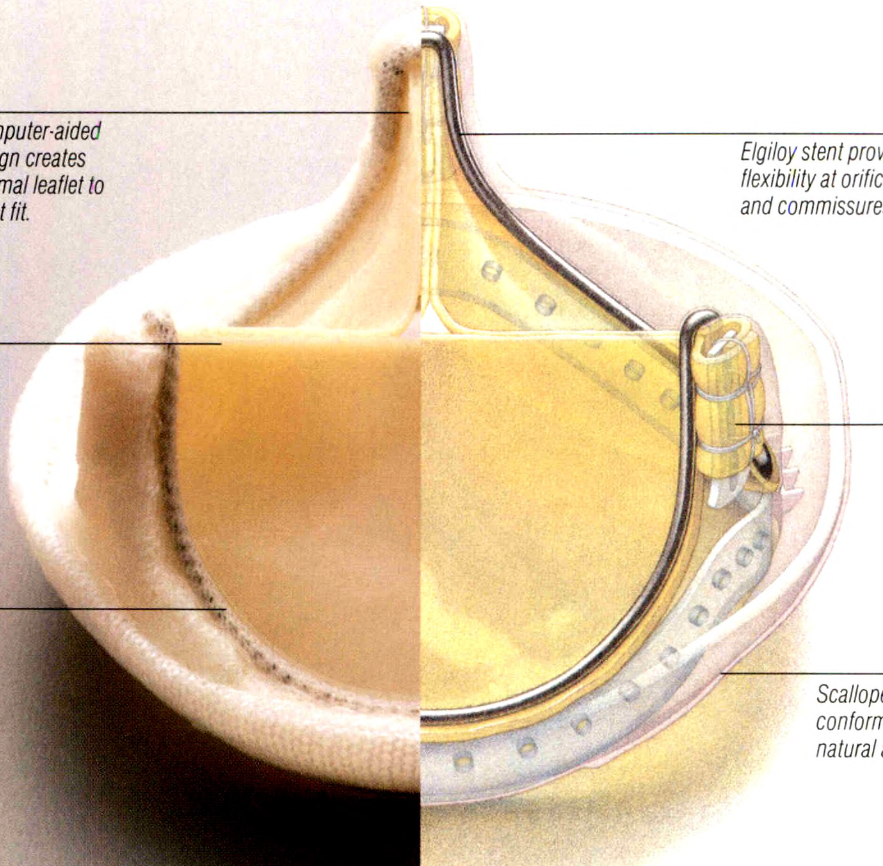
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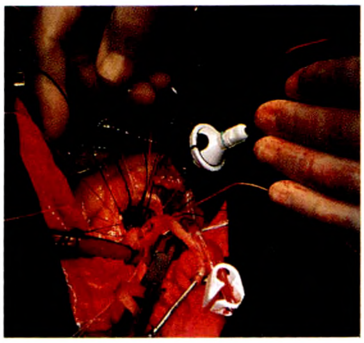

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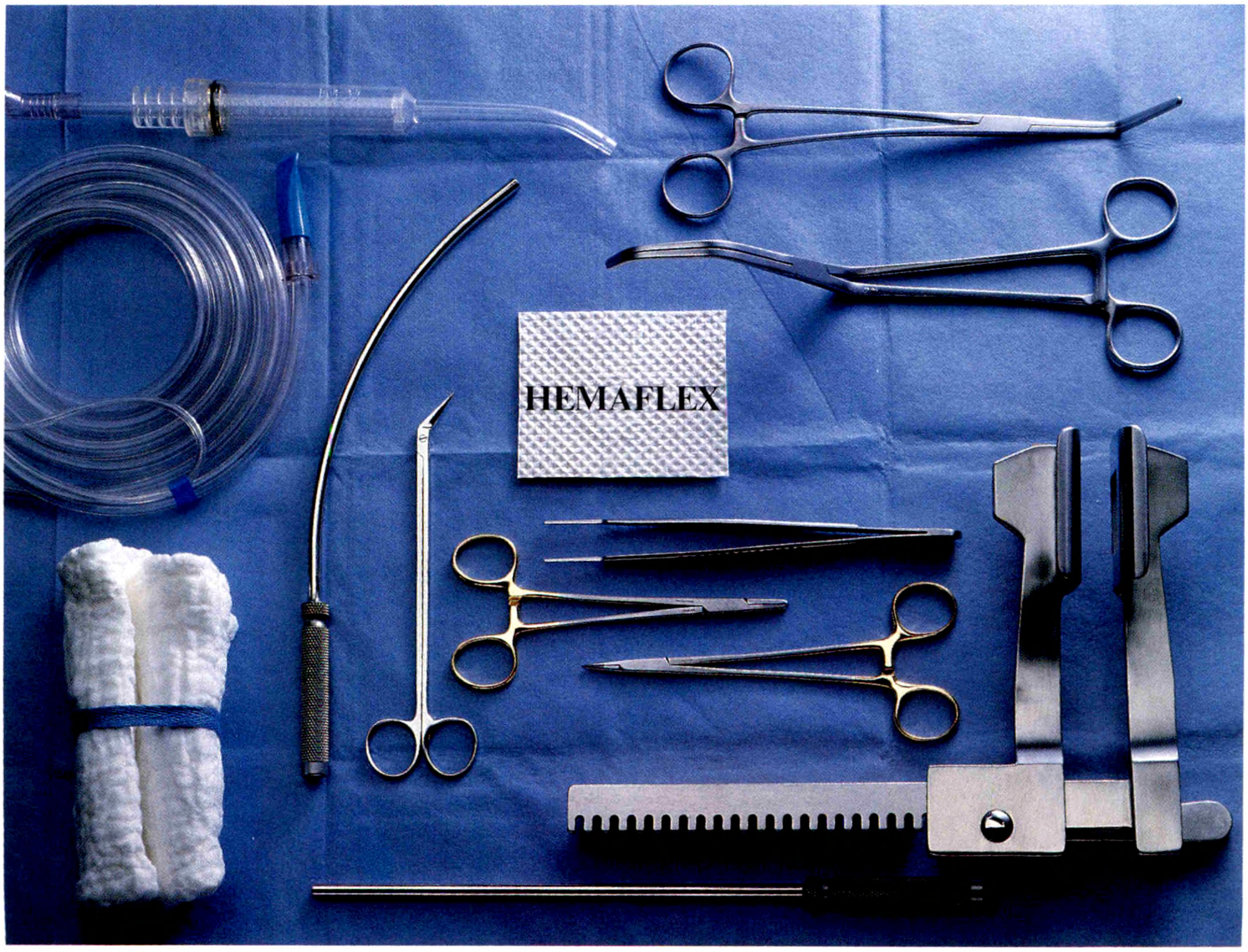

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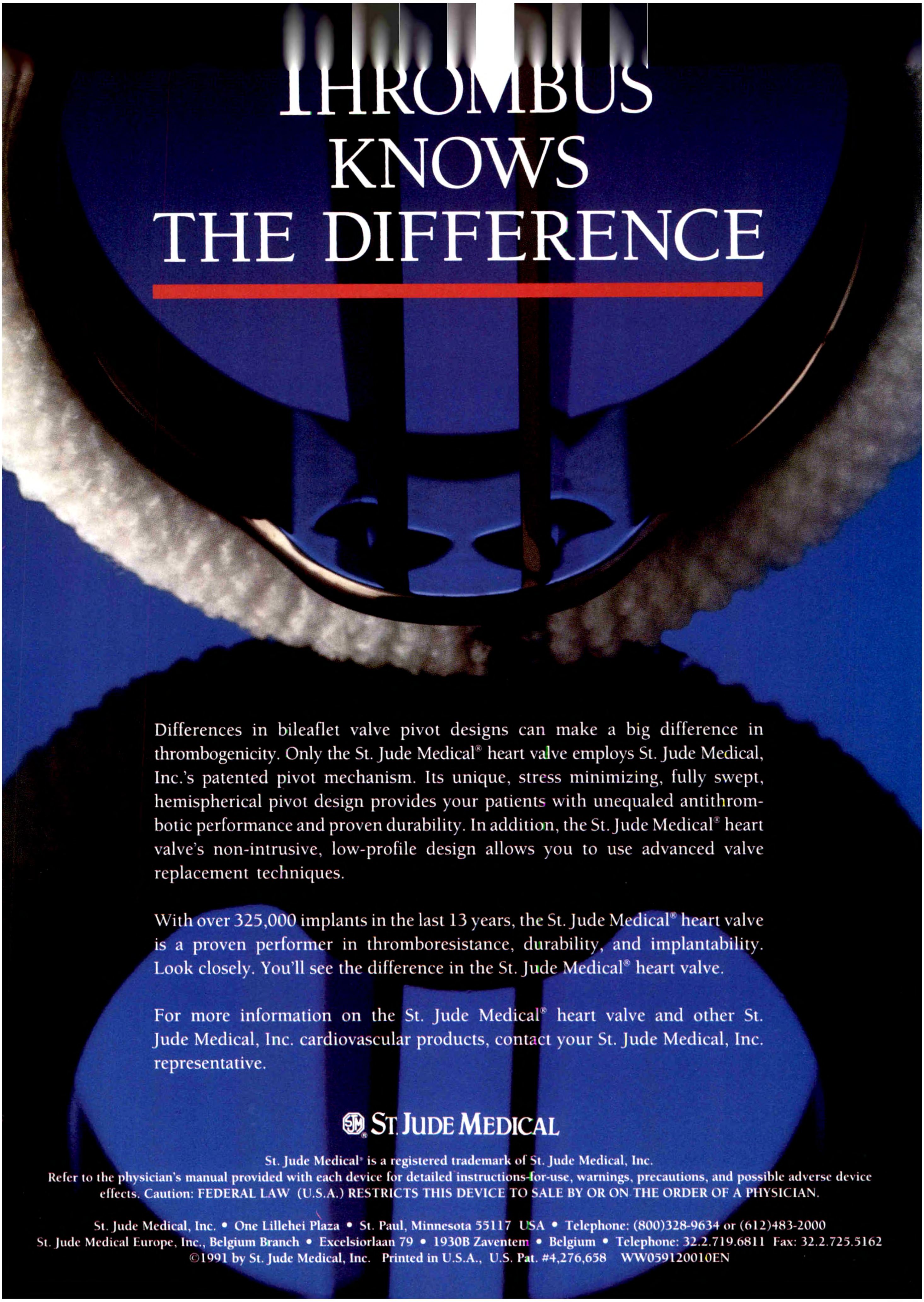

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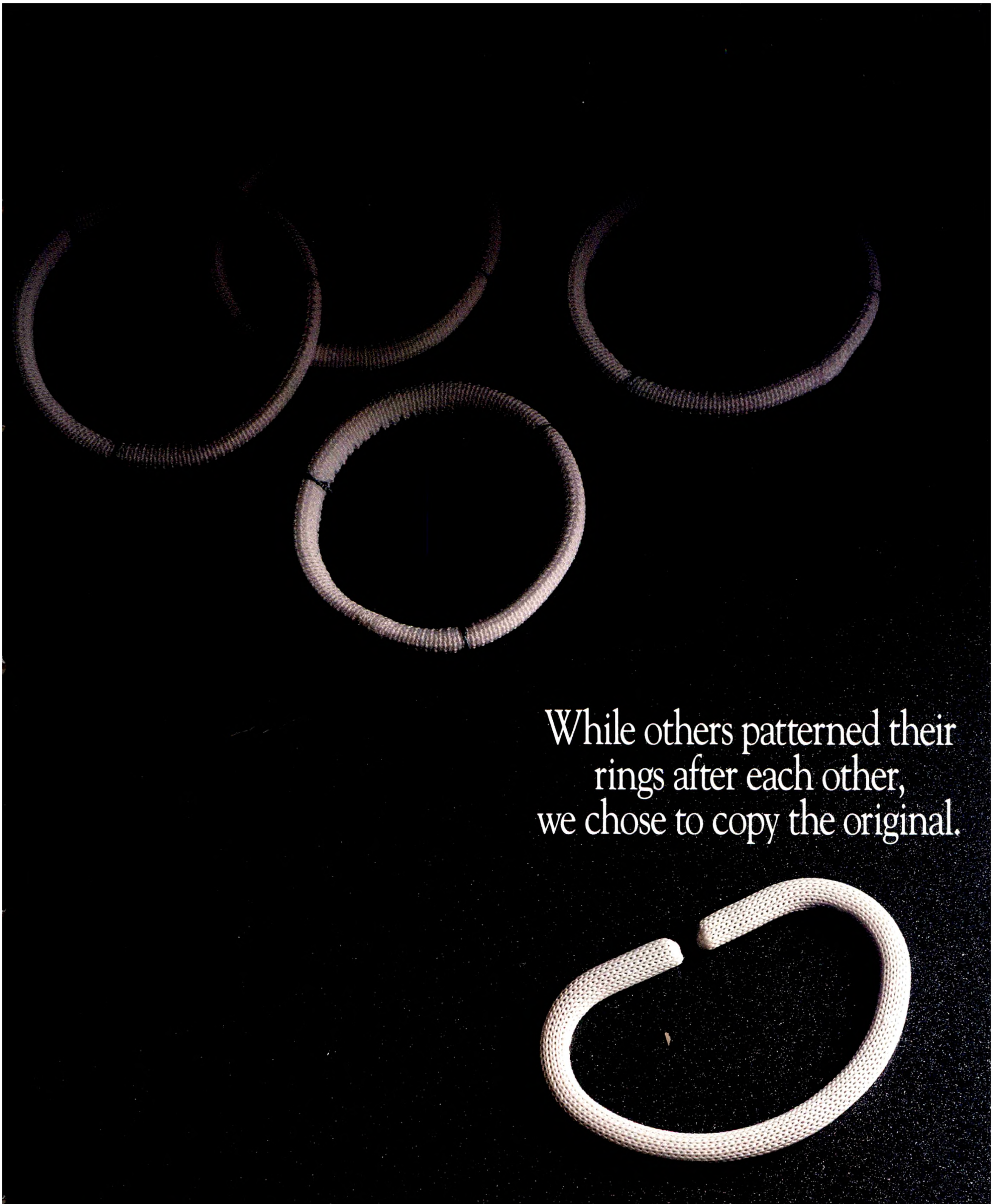

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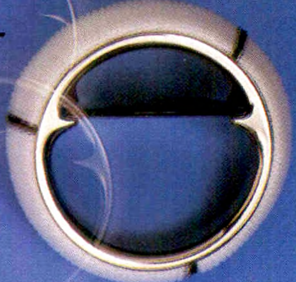

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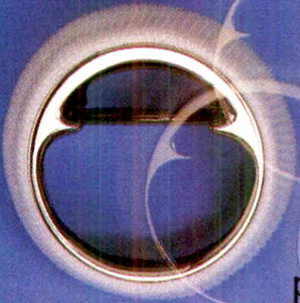

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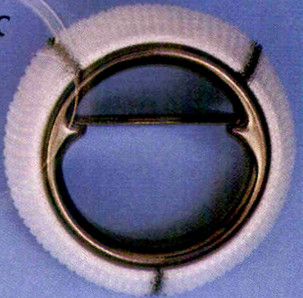

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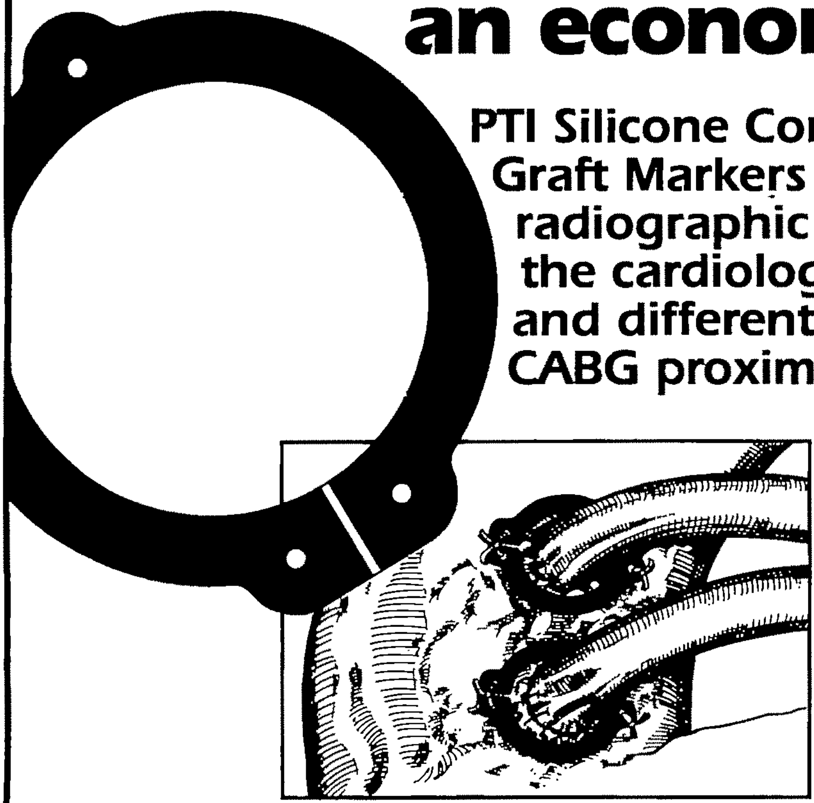

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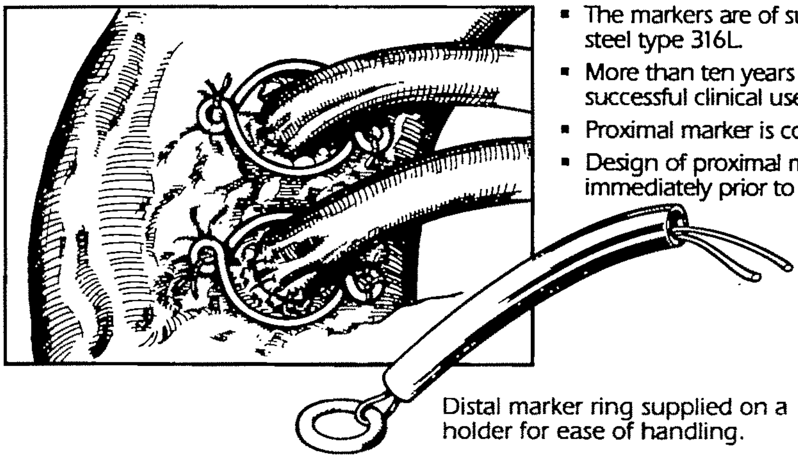

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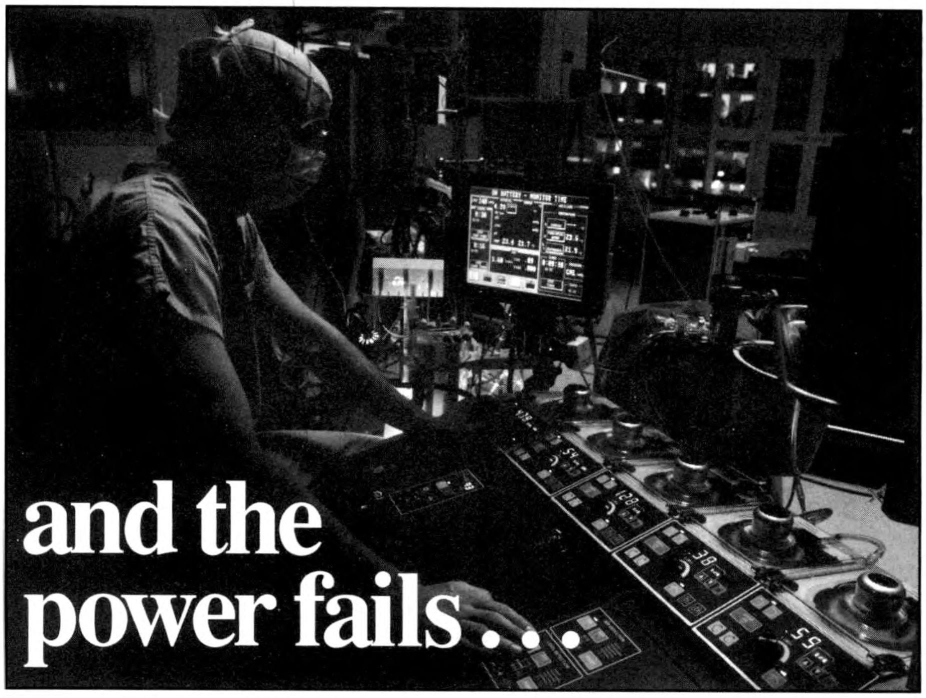

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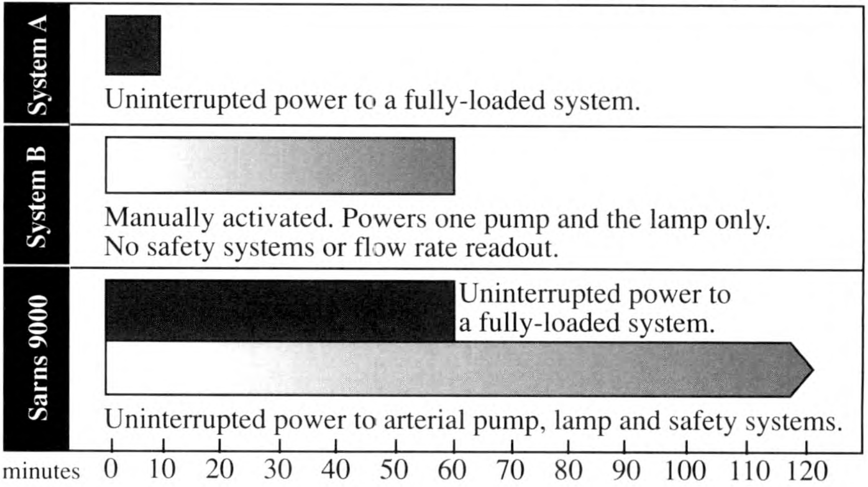

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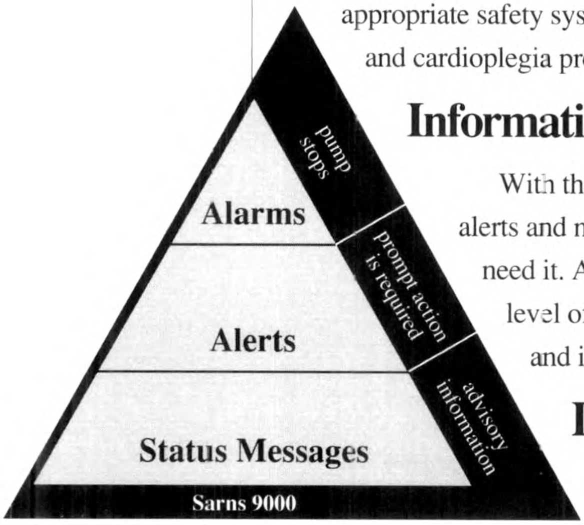

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Footnotes

[1] Greenhalgh D, Thomas W. Blackout during cardiopulmonary bypass. *Anaesthesia.* 1990;45:175. This article reports on power failure accompanied by back-up generator failure.

[2] Kurusz M, Conti VR, Arens JF, et al. Perfusion accident survey. *Proc Amer Acad Cardio Perfusion.* 1986;7:57–65.

[3] Human factors issues with surgical devices. *Tech for Perfusion.* 1991;3:1.

[4] Schabel RK, Berryessa RG, Justison GA, et al. Ten common perfusion problems: Prevention and treatment protocols. *J Extra-Corpor Tech.* 1987;19:392–398. These authors identify the perfusion problems "most likely" to occur, recommend preventive measures, and give examples of emergency protocols.

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The frame is designed to be flexible at the orifice as well as at the commissures. The compliance of the stent's commissure supports is intended to reduce the loading shock at the valve commissures and free margin of the leaflets. The flexibility of the orifice is intended to reduce the loading shock at the base of the leaflet. The flexible orifice concept is based on the physiology and mechanics of natural heart valves and reported experience with implantation of unstented homografts.

The lightweight frame is made of Elgiloy,* a corrosion-resistant alloy, chosen because of its superior spring efficiency and fatigue-resistant characteristics.

The metal frame is covered with a porous, knitted polytetrafluoroethylene cloth to facilitate tissue ingrowth and encapsulation. The inflow orifice is contoured to support the right coronary cusp of the porcine valve. The commissure supports are not equidistant to conform to the anatomical configuration of the porcine aortic valve. The suture ring has a soft, silicone-rubber insert that is covered with a porous, seamless polytetrafluoroethylene cloth. The aortic sewing ring has been scalloped to conform to the natural aortic root. The compliant nature of the suture ring facilitates coaptation between the valve and an often irregular or calcific tissue bed.

The porcine aortic valves are fixed in buffered glutaraldehyde, and then mounted on cloth-covered frames. After mounting, the valves are tested *in vitro* to ascertain their competency and flow characteristics. After final inspection, the valves are sterilized, rinsed, and then transferred into fresh glutaraldehyde and packaged under aseptic conditions.

Indications

Bioprostheses are indicated for use in patients suffering from valvular heart disease. Aortic and mitral valvular heart disease are conditions involving any of the following: obstructions of the heart valves, or stenosis; leakage of the valves, known as regurgitation or insufficiency; and combinations of the two, sometimes referred to as mixed disease or combined lesions.

Valvular heart disease may be caused by any number of factors, including congenital abnormalities, infection by various microorganisms, degenerative calcification, rheumatic heart disease and rupture or dysfunction of the mitral subvalvular mechanism secondary to myocardial ischemia.

Bioprostheses are used particularly in those patients for whom long-term anticoagulation is contraindicated or who may be difficult to maintain on anticoagulation therapy.

The Carpentier-Edwards Bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of th[illegible]al valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis.

Warnings

For Single Patient Use Only

In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, and in patients who are on maintenance hemodialysis.

The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient and consideration of alternative methods of treatment. Overall durability, especially long-term, has not been established for bioprostheses.

Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery.

Note: Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis (see Clinical Experience).

Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed.

Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection.

Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombus. The appropriate anticoagulation therapy must be determined by the physician on an individual basis.

Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc., should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result.

Carpentier-Edwards Bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.

Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards Bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided.

Both the valve and glutaraldehyde storage solution are sterile. The outside of the jar is not sterile and must not be placed in the sterile field. Furthermore, the valve and container should never be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation.

Storage between 10°C and 25°C (50-77°F) is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue.

Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with the eyes, seek medical attention.

Surgical Precautions

1. When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result (Ref. 50). The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient.
2. Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts.
3. Left ventricular perforation or ulceration has been reported (Ref. 16). Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage.
4. The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15 mm, produced a pursestring effect, causing compression of the valve orifice.
5. When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures.
6. Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue.
7. As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs, which would interfere with proper valvular function.
8. The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence.

   Note: The contrasting suture material should not be removed from the prosthesis.
9. In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia.
10. A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar and implantation data card; if any differences are noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal.
11. Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation.

Complications

As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days), necessitating reoperation and replacement of the prosthetic device.

Complications associated with the use of Carpentier-Edwards Bioprostheses compiled from the literature and from reports received through the complaint handling system in accordance with the United States (Federal) regulations establishing Good Manufacturing Practices, 21 CFR Section 820.198, include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, degeneration, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct.

Note: Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies (Ref. 10) show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes a potential relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the post-operative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards Bioprostheses to calcification as compared to other commercially available porcine valves.

Clinical Experience

Clinical data, available on 288 patients requiring isolated aortic valve replacement (AVR) and on 241 patients requiring isolated mitral valve replacement (MVR) with Models 2625 and 6625 Carpentier-Edwards Bioprostheses, respectively (mean follow-up of 2.6 and 2.2 years, respectively), indicate operative (< 30 days) and postoperative (> 30 days) survival rates of 97.2% and 95.0% per patient year, respectively, for the isolated AVR population, and 90.9% and 93.2% per patient year, respectively, for the isolated MVR population.

In the isolated AVR population there were a total of 208 (72.2%) males and 80 (27.8%) females with a mean age (± standard deviation) of 65.2 (± 10.1) years and a range of 24-88 years. The indications for valve replacement were stenosis (52.4%), regurgitation (13.5%), mixed disease (18.4%), and previous prosthetic mitral valve dysfunction (2.1%). For 13.5% of the patients, the diagnosis for replacement is unknown.

In the isolated MVR population there were a total of 79 (32.8%) males and 162 (67.2%) females with a mean age of 63.3 (± 10.5) years and a range of 30-84 years. The indications for valve replacement were stenosis (27.0%), regurgitation (36.1%), mixed disease (19.9%), and previous prosthetic aortic valve dysfunction (7.5%). For 9.5% of the patients, the diagnosis for replacement is unknown.

The follow-up methods used at each clinic included hospital visits, office visits, and contact by telephone or letter with either the patient himself, his family, or his local doctor.

These data were compiled from a multicenter clinical trial conducted by Edwards CVS Division.

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☐ Arrange manuscript as follows: (1) title page, (2) abstract, (3) text, (4) references, (5) tables, and (6) legends. Number pages consecutively, beginning with the title page as page 1 and ending with the legend page.

☐ A manuscript for an *original article* ordinarily should not exceed 26 double-spaced typewritten pages, and should be proportionally shorter the more illustrations and tables that are included.

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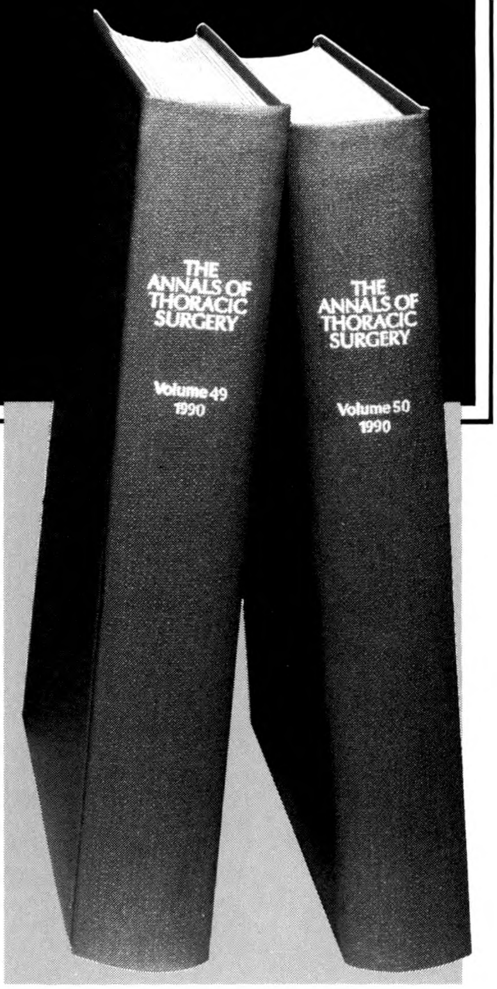
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Cardioplegia: From the Bedside to the Laboratory and Back Again

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One of the most important applications of the dictum *primum non nocere* in cardiac surgery is the protection of the myocardium to prevent its impairment while simultaneously achieving a quiet and bloodless operative field to facilitate the surgical procedure. For this obvious reason, myocardial protection has been a prime concern for surgeons since the dawn of cardiac surgery. The technique has evolved over the years from Melrose's original potassium cardioplegia to systemic and topical hypothermia, continuous coronary perfusion with electrically induced ventricular fibrillation, cold potassium cardioplegia, and more recently to blood cardioplegia. This latter technique is reviewed in detail by Barner [1] in this issue of *The Annals*. The considerable amount of scientific data reviewed by the author in this article represents only the tip of the iceberg of the massive amount of literature on cardioplegia. Much of these data come from sophisticated laboratory studies. The issue clinicians must face is how and when to apply this information obtained in the scientific laboratory to their routine clinical practice. Not too long ago, three experts on a panel discussed and reviewed the pros and cons of crystalloid and blood cardioplegia. At the end of this discussion, the moderator asked each expert on the panel to state which technique he actually used in clinical practice. The first one said blood cardioplegia, whereas the second used crystalloid cardioplegia. The third replied that he used something in between—a pink cardioplegia! How do we extrapolate and apply basic laboratory data into clinical practice? Are there any pitfalls?

See also page 1354.

Indeed, many such pitfalls are well known [2]. Species differences, statistical design, and limitations of various experimental models are obvious and are therefore readily recognized. One of the issues that has not been discussed explicitly and guarded against is the question of various "surrogate endpoints" often used for the experimental studies.

All scientific studies need clear endpoints to allow for quantitative analysis. We need to remember, however, that what is really important to the patients is the clinical outcome, represented by "clinical endpoints." These clinical endpoints include mortality and morbidity, functional capacity, quality of life, and cost. Unfortunately, quite often it is logistically very difficult to show a difference in these clinically highly relevant endpoints. A very large sample size may be required, and in some cases a long time may be required for the follow-up data. Thus, the "sensitivity" of clinical endpoints to discriminate between different modality of therapies, such as various techniques of cardioplegia, is quite low. Investigators may, therefore, be forced to use more sensitive "surrogate endpoints," which are assumed to reflect the ultimate clinical outcome. Conceptually, there are two groups of "surrogate endpoints" available (Fig 1). The "basic science endpoints" are those usually obtainable only in the research laboratory, which in the study of cardioplegia include endpoints such as the levels of high-energy phosphates, the membrane degradation products by free radicals, and the ultrastructural changes, to name a few. These parameters are highly sensitive in detecting differences between one intervention and another, but their clinical relevance needs to be proven. These endpoints could be trivial, insignificant, or simply irrelevant. Another group of surrogate endpoints is the clinical "laboratory endpoints," which are intermediate in their clinical relevance and sensitivity. In cardioplegia research, these include changes in various parameters of ventricular function and leakage of creatine kinase-MB isoenzyme, which are usually available in a clinical setting. It is important to emphasize that these sensitive parameters are of paramount importance in research, particularly in the elucidation of the pathophysiology involved. Nevertheless, when they are applied clinically, they need to be recognized as "surrogates," and their power to represent the clinical outcome needs to be critically scrutinized. Indiscriminate acceptance of the validity of surrogate endpoints could lead to wasteful, premature, and sometimes inappropriate clinical applications of laboratory data. A recent fiasco associated with the trial of encainide and flecainide for arrhythmia suppression comes to mind in this regard [3]. The importance of well-designed, prospective, randomized clinical studies cannot be overemphasized.

Another timely point brought out in the overview by Barner [1], and recently elucidated eloquently by Kirklin [4] (Fig 2), is the effect of the nature of patient population. In reviewing the results of several prospective studies comparing blood versus crystalloid cardioplegia, Barner noted that blood cardioplegia showed superiority in a number of laboratory endpoints but no detectable difference in clinical endpoints. He pointed out that this could be a reflection of the "good risk" of the patients studied. In "poor-risk" patient populations, differences in the clinical endpoints may indeed exist [1]. In recent years,

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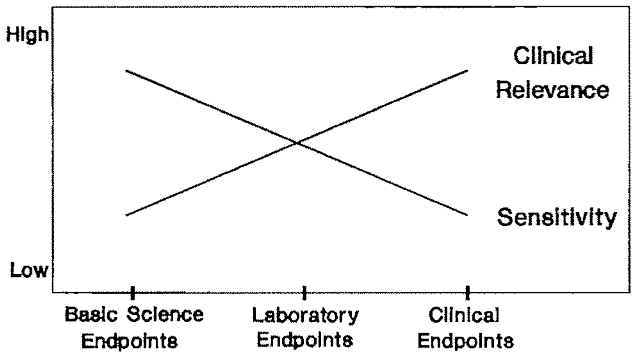

Fig 1. Surrogate endpoints: "sensitivity" = the sensitivity in establishing a difference comparing two treatment strategies; "clinical relevance" = relevance to the clinical outcome of the patients. See text for the definition of various endpoints.

several large series have shown that with the current technique of myocardial protection, overall, there is no longer a correlation between the length of aortic cross-clamp time and operative mortality. This probably represents the excellent survival rate of patients in these series. In the subgroup of patients with compromised myocardial reserve and increased vulnerability to ischemic insults, the study may discriminate better a superior technique from a less optimal one, even when the less sensitive "clinical endpoints" are used for study. This consideration is important in interpreting some of the negative clinical comparative studies. With the advent of percutaneous transluminal coronary angioplasty and other catheter interventions, patients undergoing cardiac operations are becoming older, with more compromised myocardial function and requiring more complex interventions [5]. One can expect that in these patients, superiority of certain techniques that cannot be detected in previous patient population may become apparent. We may have to return to the laboratory to find even better ways of protecting the heart.

The progress in cardioplegia, as in many other areas of medicine, will come from back-and-forth interaction between the research laboratories and the patients' bedside.

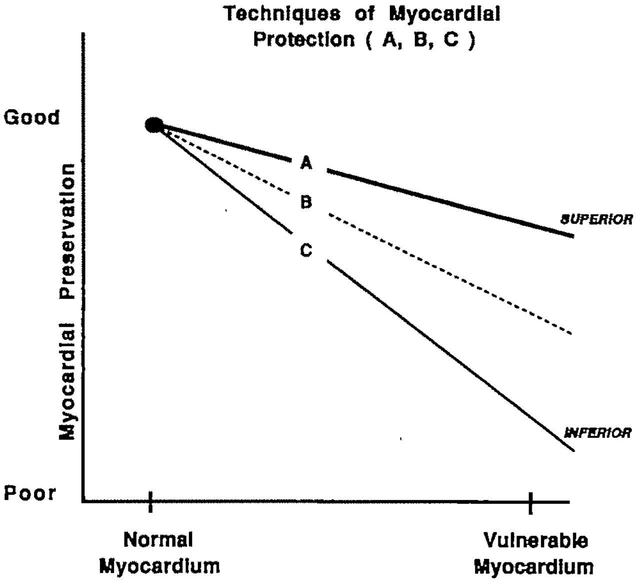

Fig 2. Differences in clinical effectiveness of various cardioplegic techniques may become apparent in poor-risk patients. (Modified from Kirklin JW [4].)

Clear recognition of pitfalls in these shuffling pathways can only facilitate such interaction, ultimately leading to better care of our patients.

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Human Coronary, Internal Mammary, and Gastroepiploic Artery Reactivity

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Pharmacological studies on human tissues are quite important to extrapolate the obtained data to healthy or disease humans, devoid of species variations. However, there are many problems in harvesting and using the materials. Gastroepiploic and internal mammary arteries are relatively easy to obtain for experimental study and usually in good condition for research, because fresh specimens are available from the stomach excised because of cancer or ulcer and from the mammary artery isolated for coronary artery bypass grafting. On the other hand, it is very difficult to obtain blood vessels from vital organs such as the heart and brain. Heart transplantation has made it possible to use coronary arteries from the disease heart for study. However, such arteries may show severe histological lesions, such as atheromatous changes, and the effects of prolonged, intensive drug therapy before operation. An alternative source of human coronary artery is the body during autopsy. Here the problems are postmortem functional and histological changes. Because of ethical, social, medical, biological, and economical problems in harvesting the human materials, experimental animals have been widely used for the determination of drug effects and analysis of the mechanisms of action. Responsiveness of blood vessels is quite variable in different animals. For example, in coronary arteries, norepinephrine causes contraction in humans and monkeys [1] but relaxation in dogs and pigs; acetylcholine causes contraction in humans [2] and pigs and relaxation in monkeys and dogs. Therefore, one has to know how the data obtained from experimental animals can be extrapolated to humans. Because of this, comparative studies of humans versus monkeys and subprimate mammals become very important.

See also page 1310.

In this issue, O'Neil and associates [3] compare the contractile responsiveness to endogenous substances and those commonly used as therapeutic agents in human gastroepiploic and internal mammary arteries obtained during operation. They found that norepinephrine and dopamine produce comparable dose-related contractions, but 5-hydroxytryptamine (5-HT) and a thromboxane A_2 analogue are more efficacious in the mammary artery. Histamine contracts the mammary artery but does not alter the tone of gastroepiploic artery under resting conditions. From these findings, they suggest that the internal mammary artery is more predisposed to the events that initiate vasospasm. These data provide important information on drugs used during and after bypass operations and show that endogenous vasoconstrictor substances influence the lumen size of bypass grafts differently, thus altering the prognosis for the grafts and ultimately the patients.

In addition to response comparisons between these arteries, thoracic surgeons are interested in knowing the similarities and differences in responses between these two vessels and the coronary arteries. Norepinephrine [1] and dopamine contract human coronary arteries; however, the magnitude is less than that in the gastroepiploic and mammary arteries. Histamine seems to be equally effective in producing contractions in coronary [2, 4] and mammary arteries, although the integrity of endothelium in the latter was not determined. Thromboxane A_2 analogues [5] and 5-HT [2, 6] are also potent vasoconstrictors in coronary arteries, as they are in the other two. Drug receptors and their subtypes involved in the response have not yet been determined in the gastroepiploic and mammary arteries and are only partially known in coronary arteries. Alpha$_1$- and α_2-adrenoceptor subtypes [7], H_1 histaminergic receptor subtype [4], and 5-HT$_2$- and 5-HT$_1$-like serotonergic receptor subtypes [6] are involved in the contraction caused by norepinephrine, histamine, and 5-HT.

It is quite important to determine the reasons for different magnitudes of contraction in different arteries. The possibilities are (1) the quantity and affinity of receptors responsible for the smooth muscle contraction and functioning of intracellular processes after receptor activation (G-protein, adenylate cyclase, guanylate cyclase, phospholipase C, protein kinase, and Ca^{2+} availability) and (2) how the vasodilator response counteracting the contraction is induced by the agents. Biochemical studies on receptor binding and intracellular events are not easy to perform in human material because of the limited quantity and the inhomogeneity. However, the vasodilator responses can easily be determined following the methods used by O'Neil and associates. They postulate the importance of endothelium-derived relaxing factor in modifying the agonist-induced contraction. This is true in the case of histamine actions on human coronary arteries [4]; however, norepinephrine and 5-HT do not release endothelium-derived relaxing factor in the human artery [2] but do in the porcine artery [8]. In addition to endothelium-derived relaxing factor, one has to take into account of the role of prostacyclin, a potent vasodilator in

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coronary, gastroepiploic, and mammary arteries, which is liberated from endothelial and subendothelial tissues.

It should be noted that the responsiveness of a variety of human arteries to endogenous vasoactive substances and to drugs differs in quality, quantity, and mechanism. Such heterogeneity plays an important role in regulating blood supply to the various organs and tissues. It is to be expected, then, that gastroepiploic and internal mammary arteries, when transferred to the heart, would not behave functionally like the coronary artery.

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University of Wisconsin Solution for Human Donor Heart Preservation: Initial Clinical Experience

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Although in vitro and primate orthotopic transplant experiments have suggested the superiority of University of Wisconsin solution (UWS) compared with crystalloid cardioplegia and saline solution storage for hypothermic heart preservation, concerns about the viscosity and the high potassium concentration of UWS have precluded its use in human cardiac transplantation. To test the safety and efficacy of UWS, 16 patients received hearts arrested with, flushed with, and stored in UWS at 4°C for a mean ischemic time of 153.3 ± 30.7 minutes. After reperfusion, the hearts contracted vigorously and attained a stable sinus rhythm within 4.0 ± 2.4 minutes, and the patients were weaned from bypass in 24.5 ± 8.0 minutes. There was no evidence of acute or chronic ischemic myocardial injury by enzymatic analysis, electrocardiography, or biopsy specimen histology. The results suggest UWS can be safely used, within currently accepted limits of donor ischemic time, to arrest and preserve human hearts for transplantation. Further studies of preservation are required to compare UWS with crystalloid cardioplegia and saline solution storage and to test the ability of UWS to prolong the period of safe donor hypothermic ischemia in clinical heart transplantation.

(Ann Thorac Surg 1991;52:1213–6)

Improving and extending the period of hypothermic preservation could expand the available donor pool and provide a wider safety margin for long-distance organ procurement. Four to 5 hours is currently the accepted time limit for preservation in human cardiac transplantation; longer periods of ischemia are associated with higher perioperative morbidity and mortality [1, 2].

University of Wisconsin solution (UWS) improves and prolongs hypothermic preservation for clinical transplantation of the pancreas, kidney, and liver [3–5]. In vitro experiments [6–9] using animal hearts preserved with UWS have shown improved preservation after 12 hours, better maintenance of high-energy phosphate levels, and improved structural preservation on histological analysis compared with crystalloid cardioplegic solutions and saline solution storage. Furthermore, we [10, 11] have previously demonstrated the ability of donor baboon hearts preserved for 18 hours in UWS to maintain circulation after orthotopic transplantation into immunosuppressed baboons until they were killed at 6 weeks. Preserved myocardial function was shown by short-term and long-term animal survival, histological studies, enzymatic analysis, electrocardiogram (ECG), right heart catheterization, and echocardiogram. Because of the success with nonhuman primate cardiac preservation, UWS was studied in human donor heart procurement. This study details our initial use of UWS in humans and addresses safety and efficacy during preservation before heart transplantation.

Material and Methods

We reviewed the records of 16 consecutive adult patients at Columbia-Presbyterian Medical Center who received hearts arrested with and stored in UWS between May and September 1990. Donor criteria at our institution include the following: age less than 50 years, normal echocardiogram and ECG, minimal (<10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) dopamine hydrochloride support, negative human immunodeficiency virus titer, and no systemic infection. The UWS was prepared according to DuPont protocol (ViaSpan with 40 units of regular insulin and 16 mg of methylprednisolone). The composition was as follows:

Hydroxyethyl starch (Pentafraction) (g/L) 50
Lactobionic acid (mmol/L) 100
Potassium phosphate (mmol/L) 25
Magnesium sulfate (mmol/L) 5
Raffinose (mmol/L) 30
Adenosine (mmol/L) 5
Allopurinol (mmol/L) 1
Glutathione (mmol/L) 3
Dexamethasone (mg/L) 16
Glucose (mmol/L) 0.06
Potassium (mEq/L) 113
Sodium (mEq/L) 30
Total osmolarity 323
pH 7.4

Cardiectomy was performed in standard fashion [12], and UWS was used for cardioplegia. It was administered

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Table 1. Myocardial Enzyme Levels of Patients Undergoing Heart Transplantation After Graft Preservation With University of Wisconsin Solution[a–c]

Time	AST (IU)	CK[d] (IU)
Preoperative	55.0 ± 46.2	95.8 ± 99.6
Postoperative (h)		
0	79.2 ± 37.0	334.7 ± 283.5 (1.1, 2.1)
24	82.2 ± 47.1	300.9 ± 179.7 (5.4)
48	60.0 ± 23.9	157.1 ± 111.4

[a] Data are shown as the mean ± standard deviation. [b] There was no significant difference between the preoperative and postoperative values for either AST or CK. [c] The following values are considered signs of critical myocardial injury after open heart operations at our institution: AST, >100 IU; CK, >400 IU; and MB fraction, >5% or >20 IU. [d] Data in parentheses are percentages of myocardial bands on CK fractionation.

AST = aspartate aminotransferase (serum glutamic-oxaloacetic transaminase); CK = creatine kinase.

through standard intravenous tubing at a pressure of 80 mm Hg, a total volume of 15 mL/kg, and a temperature of 4°C. Arrested donor hearts were stored and transported in UWS at 4°C.

During transplantation, after each of the left atrial, right atrial, and pulmonary artery anastomoses, the coronary circulation was flushed with 250 mL of cold blood cardioplegia (4:1). Patients were weaned from cardiopulmonary bypass with dopamine and isoproterenol hydrochloride as inotropic support. Temporary myocardial pacing and defibrillation were used as indicated. Eleven patients received cyclosporine, azathioprine, and steroids for immunosuppression, and 5, because of preoperative renal dysfunction, received OKT3 and steroids. Myocardial preservation was assessed postoperatively by: (1) cardiac enzyme analysis in all patients at 0, 24, and 48 hours; (2) ECG, endomyocardial biopsy, and right heart catheterization in all patients at 1 week; and (3) echocardiography in 8 patients at 1 week.

Results

The study population consisted of 12 male and 4 female patients with a mean age of 53.7 ± 9.9 years (range, 29 to 63 years). Mean donor ischemic time was 153.3 ± 30.7 minutes (range, 96 to 189 minutes). After reperfusion, stable sinus rhythm was attained in 4.0 ± 2.4 minutes (range, 2 to 12 minutes), and patients were weaned from cardiopulmonary bypass in 24.5 ± 8.0 minutes (range, 18 to 46 minutes). Temporary pacing in the operating room was required in 2 patients for 5 and 30 minutes, and 3 patients required defibrillation. Dopamine and isoproterenol were discontinued after 3.5 ± 0.8 days (range, 2 to 5 days).

Aspartate aminotransferase (AST) and creatine kinase (CK) levels were obtained preoperatively and at 0, 24, and 48 hours postoperatively (Table 1). The AST level peaked at 82.2 ± 47.1 IU (range, 32 to 170 IU) within 24 hours and returned to baseline by 2 days. In 2 patients, the AST level rose to more than 100 IU postoperatively. The CK level peaked at 334.7 ± 283.5 IU (range, 156 to 1,320 IU) immediately postoperatively. The MB fractions were determined in the 3 patients whose CK peak values were greater than 400 IU, and only one was elevated. The CK and AST elevations from baseline were not significant.

One week after transplantation, the ECG showed sinus rhythm at rates ranging from 75 to 105 beats per minute without Q waves or ST-T wave abnormalities. Four patients had an intraventricular conduction delay without specific bundle-branch block pattern. Right heart catheterization revealed mixed venous oxygen saturation of 69.1% ± 5.2% with a cardiac index of 3.0 ± 0.6 and a pulmonary capillary wedge pressure of 9.7 ± 3.3. Echocardiography, performed on 8 patients, showed normal contractility with fractional shortening greater than 36%. Endomyocardial biopsy specimens had no evidence of major ischemic injury. Four of the patients had minimal areas (<10% of the subendocardium) of healing subendocardial coagulation necrosis.

Up to 8 months after transplantation 10 rejection episodes requiring treatment have been detected in 9 patients. One patient with normal cardiac function died of a cerebral aneurysm. No patient has needed a permanent pacemaker.

Comment

Extending the period of preservation beyond the currently accepted 4 hours may expand the donor pool, will allow easier planning of the operation, will increase the margin of safety for long-distance procurement, and may allow time for human lymphocyte antigen matching. In addition, improved preservation techniques may decrease the need of postoperative inotropic support, shorten the stay in the intensive care unit, decrease morbidity, and help alleviate the critical donor shortage by permitting use of marginal donor hearts that were previously rejected for transplantation.

University of Wisconsin solution is used for solid-organ preservation (kidney, pancreas, liver) and has been shown to improve and extend the period of safe preservation. Studies in our laboratory and by others have demonstrated superior preservation and extended ischemic periods up to 16 hours using UWS. Although longer preservation periods have been reported experimentally, no other static hypothermic preservation technique has been reported to be as consistent as UWS regarding graft function and recipient survival [13–15]. This study addresses the issue of safety and efficacy of UWS for human heart preservation as a prelude to further questions regarding superiority and duration of preservation.

University of Wisconsin solution contains impermeants that prevent cellular swelling and components that are directed at reducing the amount of oxygen free radical injury both during storage and reperfusion [16, 17]. Although the actual contribution of each component to myocardial protection is unknown, theoretically they combine to improve preservation. Lactobionate, raffinose, and the hydroxyethyl starch component reduce the edema associated with hypothermic storage by decreasing transcapillary and osmotic fluid flow. Glutathione acts by reducing the levels of cytotoxic oxidants generated on reperfusion. Allopurinol suppresses the generation of oxygen free radicals by inhibition of xanthine oxidase. Adenosine facilitates the regeneration of adenosine triphosphate during reperfusion, and magnesium has membrane-stabilizing properties. Compared with extracellular solutions, the intracellular composition of electrolytes in UWS decreases the gradient for sodium and potassium across the cell membrane. This prevents sodium influx into the cells with the obligatory transport of water and the consequent cellular edema [18]. Because the movement of sodium and potassium across the cell membrane is minimized, adenosine triphosphate utilized in maintaining the sodium-potassium adenosine triphosphatase pump is conserved.

The UWS-preserved hearts in this study arrested quickly because of the adenosine and the high potassium content and were soft and pliable after preservation. The hearts rapidly converted to sinus rhythm, contracted vigorously after reperfusion, and required minimal defibrillation (n = 3) or temporary pacing in the operating room (n = 2). There was no evidence of ischemic injury or of major conduction abnormalities on the ECG. Four patients (4/16, 25%) had a mildly prolonged QRS interval of 12 to 15 ms (normal, 8 to 12 ms) without clinical significance. None had bundle-branch or atrioventricular block, and none required a permanent pacemaker.

An AST level greater than 100 IU and a CK level greater than 400 IU with an MB fraction of greater than 5% or greater than 20 IU have been arbitrarily set in our institution as signs of serious myocardial injury after all open heart operations. The mean AST level rose to more than baseline levels but not to the point considered indicative of myocardial injury. Two patients had a rise in AST values of greater than 100 IU. One received the heart from a heart-lung block; the heart had a brief period of exposure to room temperature and immersion in saline solution before transport. In the other patient, a coronary artery air embolism occurred on reperfusion and necessitated multiple defibrillation attempts. Three patients had CK levels greater than 400 IU, the 2 patients just mentioned and another in whom the cross-clamp was released while the temperature was 28°C, thus necessitating defibrillation. The patient with the air embolus had a peak CK value of 510 IU with a 5.4% MB fraction.

At 1 week postoperatively, echocardiograms from 8 patients demonstrated good uniform contractility and fractional shortening greater than 36% in all 8 patients. Right heart catheterization revealed good mixed venous oxygen saturation and satisfactory cardiac index and pulmonary capillary wedge pressure. Endomyocardial biopsy specimens showed good myocardial preservation without evidence of major ischemic injury [19], such as subendocardial fibrosis, coagulation necrosis, or contraction bands. Four patients had small, patchy areas of subendocardial healing (<10% of subendocardium studied) without further evidence of myocardial injury. Two of these patients required defibrillation in the operating room, perhaps resulting in the injury seen on histology. All 4 patients had normal postoperative hemodynamics and right heart catheterization at 1 week, findings suggesting that the minimal damage seen on histology is clinically insignificant.

During follow-up of 6 to 8 months, 1 patient died of a ruptured cerebral aneurysm but had normal cardiac function. There have not been an excessive number of rejection episodes, and long-term myocardial function remains stable.

The high potassium concentration in UWS is potentially of concern, as there are reports [20–22] of possible damage to cardiac cells caused by previously employed hyperkalemic cardioplegic solutions, such as the Melrose solution. These solutions contained more than 180 mEq/L of potassium as a citrate and were hyperosmolar. Initial and late (after 1 month) cardiac function was depressed, resulting in withdrawal of the solutions for use in cardioplegia. Potassium is thought to injure endothelial cells and cause vasospasm and increased capillary permeability. Gharagozloo and associates [20] demonstrated that solutions with a potassium concentration between 20 and 30 mEq/L are best for cardioplegia without cellular damage, but their model used normothermic ischemia. In controls without a period of ischemia, solutions with a high potassium concentration did not alter cellular damage. Kohno and co-workers [18] reported better myocardial function after initial arrest with a lower potassium solution and then storage in an intracellular (Collins) solution. Mankad and colleagues [23] perfused rat hearts in a Langendorff preparation with solutions containing greater than 30 mEq/L of potassium and demonstrated decreased endothelial response to vasodilators. This model used continuous perfusion at 20°C and may not be directly applicable to static preservation.

Using hypothermic static arrest, techniques employing potassium concentrations of 126 mEq/L have been successful in achieving preservation periods up to 24 hours [13]. Follow-up to 8 months in our study demonstrates no depression in initial or long-term myocardial contractility and no major conduction abnormalities, arrhythmias, or ultrastructural damage. Although there is no evidence of endothelial injury leading to accelerated graft atherosclerosis, this can be documented only by annual coronary angiograms. Perhaps the attenuation of the hyperkalemic effect can be attributed to (1) isosmolarity, (2) adenosine, which arrests the heart before potassium and may prevent possible potassium-induced coronary vasospasm [24], and (3) impermeants that prevent cellular injury, which can result in an influx of calcium and contraction damage to the myocytes.

The high viscosity of UWS, especially when cold, did not pose a problem for delivery. The solution was administered through standard intravenous tubing without an in-line filter at a perfusion pressure of 80 mm Hg. There was uniform myocardial distribution of UWS as demonstrated by excellent postoperative function and insignificant enzyme leakage.

This study demonstrates the efficacy and safety of UWS in achieving adequate hypothermic preservation within currently accepted time limits in humans with excellent return of cardiac function, graft survival, and no demonstrable deleterious long-term effects. Further studies are required to (1) evaluate extended long-term effects of UWS (greater than 1 year), (2) compare UWS with crystalloid cardioplegia and saline solution storage, and (3) study the ability to prolong the period of donor ischemia past 4 hours in clinical heart transplantation.

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DISCUSSION

DR DAVIS C. DRINKWATER, JR (Los Angeles, CA): My colleagues and I at UCLA have just completed a similar study, which was randomized and double-blind, comparing Stanford cardioplegia with UWS. Basically, we found the same things you did, ie, an early return of function and particularly sinus rhythm. There have been no early significant differences in ventricular function, early survival, or need for inotropic support. However, the data are still being evaluated statistically. I think that we will have to follow up the patients for a greater time to see possible long-term benefits of the UWS. Also, I suspect, and I wonder if you are going to embark on this based on your results, longer ischemic times will perhaps demonstrate substantial benefit. We have performed transplantation in patients with ischemic times of 8 hours using UWS and modified reperfusate without noticeable detriment in ventricular function.

DR JEEVANANDAM: Most of the donor hearts at our institution come from the local area, so we do not have a real need to extend preservation times. However, should the need arise, we are confident enough with our baboon and human data to extend ischemic time up to 10 hours.

Improved Management of the Paget-Schroetter Syndrome Secondary to Thoracic Outlet Compression

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For years, patients with "effort" thrombosis were treated by anticoagulants and conservative exercise; and if recurrent symptoms developed when they returned to work, they were considered for first-rib resection. Recently, use of thrombolytic agents with early surgical decompression of the neurovascular compression has markedly reduced morbidity, such as postphlebitic syndrome and the necessity for thrombectomy. A review of 67 patients seen over 25 years showed that 34 were initially treated with heparin sodium and then Coumadin (crystalline warfarin sodium). Recurrent symptoms developed in 21 after they returned to work and necessitated transaxillary first-rib resection to relieve symptoms. Eight also underwent thrombectomy. Recently, 33 patients were initially treated with thrombolytic agents and heparin, followed promptly by early first-rib resection. The evaluation and efficacy of this therapy have been established by frequent and repetitive venograms and careful follow-up of patients. Most of the patients showed improvement with thrombolytic agents. Remaining stenoses that suggested intravascular thrombosis were usually secondary to external compression of the vein by the clavicle, costoclavicular ligament, rib, or scalenus anterior muscle. Venous thrombectomy was necessary in only 4 patients in whom the clot was not controlled by thrombolytic therapy and operative release of compression. There were no deaths in the series.

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"Effort" thrombosis of the axillary-subclavian vein (Paget-Schroetter syndrome) is usually secondary to unusual or excessive use of the arm in addition to the presence of one or more compressive elements in the thoracic outlet [1, 2]. For many years, therapy included elevation of the arm and use of anticoagulants with subsequent return to work. If symptoms recurred, the patient was considered for a first-rib resection, with or without thrombectomy [3], as well as resection of the scalenus anterior muscle and removal of any other compressive element in the thoracic outlet, such as the cervical rib or abnormal bands [4–6].

Recent availability of thrombolytic agents [7–9] combined with prompt surgical decompression of the neurovascular compressive elements in the thoracic outlet [10] have reduced morbidity and the necessity for thrombectomy and have substantially improved clinical results, including the ability to return to work. The purpose of this report is to define the evolution of therapy for and the advantages of improved management of patients with effort thrombosis secondary to thoracic outlet syndrome [11, 12].

Material and Methods

Clinical manifestations of effort thrombosis of the axillary-subclavian vein in the acute phase or in the subacute phase (from 2 to 30 days) in 71 extremities in 67 patients were reviewed. There were 28 male and 39 female patients ranging in age from 16 to 51 years (mean age, 32 years). Trauma was involved in 37 patients. Fifty-four patients had unusual occupations that involved excessive repetitive muscular activity of the shoulder, arm, and hand; and symptoms were usually exacerbated when working over the head. Potentially aggravating occupations included sports such as golf, tennis, baseball, and football. Painters, beauticians, linotype operators, weight lifters, cheerleaders, and drill team members also were seen.

Diagnosis

All 71 extremities demonstrated swelling and venous distention over the chest, arm, and hand (Fig 1), which suggested the clinical diagnosis of venous obstruction. Elevation of the arm or hand did not change the configuration of the veins or swelling acutely. Bluish discoloration of the extremity was present in 64 extremities and aching pain at rest or with exercise, in 45 arms. Palpable cords were present in the neck of 27 patients. Cervical ribs occurred in 19 patients. Only minimal symptoms were present in 12 extremities. More than 80% of extremities demonstrated a positive Adson's test or hyperadduction sign with the various compressive positions. The syndromes occurred bilaterally in 4 patients, simultaneously in 2 (1 with bilateral old clavicular fractures) and sequentially in 2.

Diagnostic tests performed included venography, ulnar and median nerve conduction velocities, plethysmogra-

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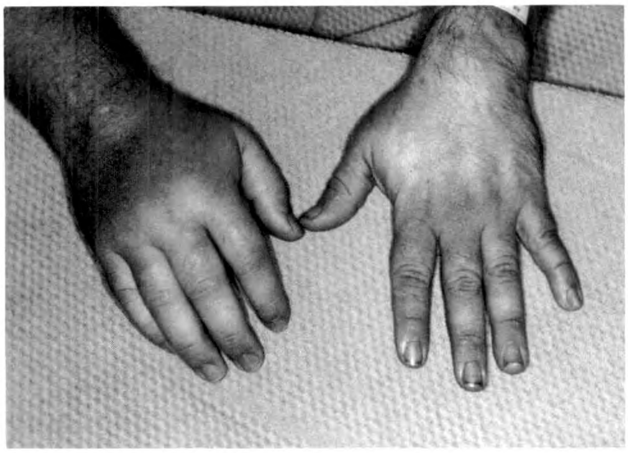

Fig 1. Swollen right hand on the side of subclavian vein occlusion.

phy, temperature studies of the hand, venous Doppler measurements, and magnetic resonance imaging [13, 14]. The diagnosis was established by clinical history, physical examination, and venogram performed through the antecubital vein. Initially a needle was used for the venogram, but with the advent of fibrinolytic agents, indwelling plastic catheters in the antecubical vein have been used to perform serial venograms and therapeutic injections.

Substantial narrowing or occlusion of the axillary-subclavian vein, usually in the area of the first rib or clavicle, was observed in all patients. Partial recanalization occurred in 3 patients seen 2 to 3 weeks after the acute episode. In 48 patients there was evidence of some collateral circulation, although it was obviously not adequate as long as the swelling was present. Pathways for the most efficient collateral communications were between the cephalic veins, transverse cervical veins, transverse scapular veins, and tributaries to the internal jugular veins.

Differential diagnoses included lymphangitis, intramuscular hemorrhage, and intermittent venous obstruction without thrombosis as well as vena cava or innominate vein obstruction.

Therapy

Two groups of patients were treated, one seen generally before 1980 and one seen after 1980 (Table 1). Group 1 comprised 35 extremities in 33 patients treated with anticoagulant therapy (heparin sodium followed by Coumadin [crystalline warfarin sodium]). Patients in this group were instructed to elevate the arm, the management usually being initiated in the hospital. All patients were encouraged to return to work after the alleviation of the symptoms. Recurrent symptoms similar to those of the initial syndrome developed in 25 of the 35 extremities after the patients returned to work. The 21 patients were readmitted, and Coumadin therapy was stopped. A transaxillary resection of the first rib was performed with removal of all other compressive elements including congenital or adhesive bands, the costoclavicular ligament, and the scalenus anterior and subclavius muscles. In addition, 8 patients required thrombectomy. The axillary-subclavian vein was opened through a transverse incision, and a Fogarty catheter was used to remove the clot from inside the vein. It was not necessary to patch the venotomy.

Clavicular resection was carried out in 1 patient bilaterally because of callous formation from old bilateral fractures of the clavicle, which had caused the syndrome initially. Although clavicular resection tends to be disfiguring when performed unilaterally, a bilateral resection is less cosmetically objectionable [15]. The patient had excellent function of the arms and shoulders and was able to touch her shoulders together anteriorly.

Group 2 comprised 36 extremities in 34 patients treated since 1980. They were managed initially with heparin and a venogram. Thrombolytic agents were administered (in most instances) through an indwelling catheter in the antecubital vein; and after lysis of the thrombus, a first-rib resection was performed promptly. In the first 10 patients, the thrombolytic treatment was streptokinase, with an initial loading dose of 250,000 U followed by 100,000 U/hour intravenously for 24 to 48 hours or until lysis of the clot occurred. This was monitored with prothrombin time and partial thromboplastin time. Operation was "safe" 4 hours after the discontinuation of streptokinase. No streptokinase was given postoperatively for at least 10 days because of the potential bleeding hazard. If a bleeding state developed, reversal of streptokinase was possible with Amicar (aminocaproic acid), fresh frozen plasma, cryoprecipitate, and fresh whole blood. All patients were tested for the hypercoagulable state before therapy.

Streptokinase acts directly on the plasminogen, which is converted to plasmin and which activates the body's thrombolytic mechanism. This is a systemic reaction and involves no heparin or antiplatelet therapy. The half-life of heparin is 90 minutes, and the half-life of streptokinase is 83 minutes. One drug can be exchanged for the other in approximately 4 hours.

Because of the disadvantage of the systemic effect of streptokinase, it was replaced by urokinase in 26 patients when this drug became available [9]. The loading dose was 4,400 U/kg by bolus, and a dosage of 4,400 $U \cdot kg^{-1} \cdot h^{-1}$ was given until clot lysis occurred. (Mean

Table 1. Therapy for the Two Patient Groups

Therapy	No. of Extremities
Group 1	
Initial anticoagulant treatment	35
Persistent or recurrent symptoms	21
Transaxillary first-rib resection	21
With thrombectomy	8
Group 2	
Initial thrombolytic treatment	36
Transaxillary first-rib resection	35
With thrombectomy	4

duration of treatment was 26 hours.) Assessment of the therapeutic effect included frequent clinical observation as well as serial venograms through the indwelling catheter. Most patients showed major improvement in less than 24 hours after administration of the thrombolytic agent.

Because of the experience with recurrence after discharge in the first series, 35 of the 36 extremities in the 34 patients were treated surgically. The heparin and thrombolytic therapy were stopped, and the first rib was removed through a transaxillary (33 patients) or combined transaxillary and supraclavicular approach (2 patients) [12]. In addition, complete division of the costoclavicular ligament, the subclavius and scalenus anterior muscles, and any bands or adhesions was performed. Scalenectomy was extensive to minimize the possibility of the muscle fibers reattaching. Any congenital or compressive bands were removed from the axillary-subclavian vein. Venograms were occasionally obtained when the patient was on the operating table, but usually direct observation assured patency of the vein. If there was any question regarding complete patency, thrombectomy was carried out. This was necessary in only 4 patients. The apparent suggestion of retained clot or failure of the thrombolytic agent to completely lyse the clot was usually explained at operation by external compressive elements even though they appeared to be "residual clot" on the venogram (Fig 2). The patients were discharged after several days and were followed up clinically without anticoagulants. After 6 weeks, they were allowed to return to work.

Table 2. Results of Treatment[a]

Group[b]	Good	Fair	Poor	Recurrence
1 (7–25 y)	10	21	4	1
2 (0–7 y)	32	2	2	0

[a] Results are shown by number of extremities. [b] Numbers in parentheses indicate length of follow-up.

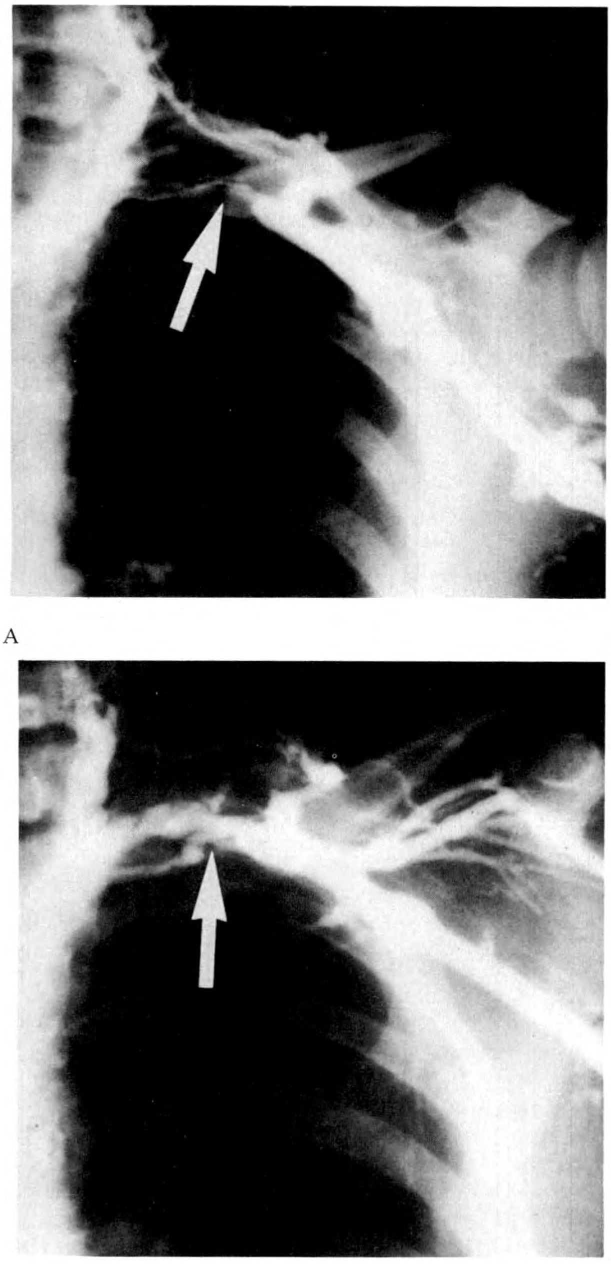

Fig 2. (A) Venous angiogram demonstrating total obstruction (arrow) *of the subclavian vein at the clavicle and first rib. (B) Venous angiogram made 24 hours after fibrinolytic therapy showing dissolution of clot except for questionable small amount of thrombus remaining* (arrow). *At operation, this was explained by extrinsic compression from the costoclavicular ligament.*

Results

In group 1, 10 extremities of the 35 showed substantial improvement with anticoagulation and elevation therapy (Table 2). In 25 extremities, symptoms either were not completely improved or recurred. The pain, swelling, and dysfunction usually occurred when the patient returned to the demanding job that had produced the difficulty initially. Twenty-one of these required first-rib resection, and 8 also underwent thrombectomy to completely remove the clot. The long-term results demonstrated 21 patients with intermittent swelling of the arm with use; they were categorized as having fair results. Two patients had constant symptoms, and 2 others had development of phlegmasia cerulea dolens with marked chronic edema of the arm and were unable to return to work. They were considered to have poor results. One of the patients who underwent surgical intervention had recurrence.

Because of these less than ideal results and the observation that a first-rib resection was usually necessary for patients to return to demanding work, therapy for the second group was modified. The patient was hospitalized and, through an antecubital indwelling catheter, a venogram was performed and thrombolytic therapy initiated. After lysis of the clot, prompt first-rib resection with removal of compressive elements including the costoclavicular ligament and scalenus anterior and subclavius muscles was carried out. Thrombectomy was necessary in only 4 extremities, and long-term results indicated that 32 of the 36 extremities had good results, ie, the patient returned to work without symptoms. Two patients were evaluated as having fair results, ie, intermittent swelling but able to work, and 2 patients were regarded as having poor results, ie, chronic swelling. No patient had phlegmasia cerulea dolens. There were no deaths in either group.

Comment

Historically, Sir James Paget [16] in 1875 in London and Von Schroetter [17] in 1884 in Vienna described this syndrome of thrombosis of the axillary-subclavian vein, which bears their names. The word *effort* [18] was added to thrombosis because of the frequent association with exertion producing either direct or indirect compression of the vein. The thrombosis is caused by trauma [19] or unusual occupations requiring repetitive muscular activity as has been in professional athletes, linotype operators, painters, and beauticians. Cold and traumatic factors such as carrying skis over the shoulder tend to increase the proclivity for thrombosis [20]. Elements of increased thrombogenicity also increase the incidence of this problem and exacerbate its symptoms on a long-term basis.

Adams and colleagues [1, 3] reported long-term results in patients treated conservatively with elevation and Coumadin. There was a 12% incidence of pulmonary embolism. Development of occasional venous distention occurred in 18%, and late residual arm symptoms of swelling, pain, and superficial thrombophlebitis were noted in 68% of the patients (deep venous thrombosis with postphlebitic syndrome). Phlegmasia cerulea dolens was present in 1 patient.

These findings substantiate our observations in group 1 that a more aggressive operative approach after thrombolytic therapy is indicated. This is particularly true for younger patients in "precipitating" occupations.

One advantage of urokinase over streptokinase is the direct action of urokinase on the thrombosis distal to the catheter, producing a local thrombolytic effect [21–23]. Streptokinase produces a systemic effect involving the alteration of serum plasminogen and increasing potential complications. Heparin is given postoperatively until the catheter is removed. A decrease in the need for thrombectomy after use of the thrombolytic agent followed by aggressive surgical intervention is another advantage, as some of the long-term disability is related to morbidity from thrombectomy as well as recurrent thrombosis [24–26].

The results in group 2 patients (aggressive thrombolytic therapy with urokinase and surgical resection of the first rib) are in marked contrast to those in group 1 patients. There was a lack of serious complications in group 2, and a high number of patients was able to return to work after 6 weeks.

The natural history of Paget-Schroetter syndrome suggests moderate morbidity [27–29] with conservative treatment alone. Bypass with vein or other conduits [30–32] has limited application. Causes other than thoracic outlet syndrome must be treated individually [33, 34] using the basic principles mentioned. Intermittent obstruction of the subclavian vein [35] can lead to thrombosis, and decompression should be employed prophylactically [31, 32].

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DISCUSSION

DR PETER C. PAIROLERO (Rochester, MN): Dr Urschel, in following up these patients, is there a role for ultrasound? Can ultrasound visualize this area, and can it determine if there is any recurrent thrombus?

DR URSCHEL: Yes, I think that ultrasound is a good way to follow up these patients. It is more difficult in the axilla and easier down the arm. After the indwelling venous catheter has been removed, it is the preferential technique combined with clinical observation.

DR PAIROLERO: What about the baseball player whom you showed us? You resect his first rib. Is he still going to pitch?

DR URSCHEL: Following the proper therapy many athletes can return to their profession. However, some cannot tolerate the repetitive, aggravative movements over prolonged time and must retire.

DR PAIROLERO: Do you recommend any type of shoulder or girdle exercise postoperatively to keep that clavicle off the second rib? In other words, do you teach these patients to do anything different about their posture?

DR URSCHEL: Conservative physiotherapy embodies good posture, strengthening the shoulder girdle, and loosening the neck muscles, all of which are important after as well as before operation.

DR ANTHONY S. PATTON (Peabody, MA): What was the age group that you operated on, and have you seen any trouble with urokinase complications such as bleeding into the brain?

DR URSCHEL: This is a local treatment with a catheter right in the clot, and that is the advantage of it. Regarding your other question, the age group is very young; the oldest patient was 51 years old.

FROM THE AUDIENCE: Usually the thrombolytic therapy does not work after 4 or 5 days; that is what we are told. Why can you give it after 20 days? Even after 20 days it works, you say.

DR URSCHEL: What happens after 20 days? You mean giving streptokinase after operation?

FROM THE AUDIENCE: Yes.

DR URSCHEL: After 10 days you can usually safely give a thrombolytic agent. Before that time it may lyse some of the clots that had formed after operation and produce bleeding. Urokinase is preferred because it has a more localized effect.

In clinical situations fibrinolytic therapy may be effective selectively for many days after the initial thrombosis. Cases have been reported to have responded for more than 30 days.

Malignant Esophagorespiratory Fistula: Management Options and Survival

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The development of a malignant esophagorespiratory fistula is a devastating complication. Data comparing various treatment options in a large group of patients are sparse. To assess the results of therapy, we reviewed our experience in 207 patients with malignant esophagorespiratory fistula. Records of 207 patients admitted to our institution with malignant esophagorespiratory fistula from 1926 to 1988 were reviewed and results of management analyzed. Age ranged from 21 to 90 years (median, 59 years); the male/female ratio was 3:1. Primary tumor site was esophagus in 161 (77%), lung in 33 (16%), trachea in 5 (2%), metastatic nodes in 4 (2%), larynx in 3 (1%), and thyroid in 1. Symptoms and signs of malignant esophagorespiratory fistula included cough in 116 (56%), aspiration in 77 (37%), fever in 52 (25%), dysphagia in 39 (19%), pneumonia in 11 (5%), hemoptysis in 10 (5%), and chest pain in 10 (5%). Respiratory location of fistula included trachea in 110 (53%), left main bronchus in 46 (22%), right bronchus in 33 (16%), lung parenchyma in 13 (6%), and multiple sites in 5 (2%). The percentage of patients alive at 3, 6, and 12 months by treatment modality was 13%, 4%, and 1% for supportive care (n = 104); 17%, 3%, and 0% for esophageal exclusion (n = 29); 21%, 14%, and 0% for esophageal intubation (n = 14); 30%, 15%, and 5% for radiation therapy (n = 20); and 46%, 20%, and 7% for esophageal bypass, respectively. Patients treated with radiation therapy and esophageal bypass had a significantly prolonged survival compared with patients treated with the other modalities. Age, sex, site of primary tumor, or site of fistula did not affect survival. In conclusion, the diagnosis of malignant esophagorespiratory fistula carries a grave prognosis. All therapy is aimed at palliation of respiratory tract contamination. Radiation therapy and esophageal bypass appear to offer survival advantage over supportive care or other modalities.

(Ann Thorac Surg 1991;52:1222–9)

The development of a malignant esophagorespiratory fistula is a devastating complication for both patient and physician. Once symptoms of fistula develop, untreated, the natural history is one of continued respiratory tract contamination, pulmonary sepsis, and death, with median survival ranging from 1 to 6 weeks [1–4]. Therapy directed at malignant esophagorespiratory fistulas is palliative, with the intent to prevent continued contamination of the respiratory tract. Various methods have been reported and included esophageal intubation (pulsion or traction techniques); esophageal exclusion with esophagostomy and gastrostomy; esophageal bypass using stomach, colon, or jejunal interposition; and occasionally resection and repair of the fistula [5]. Even after effective palliation of the fistula, there are few survivors at 1 year, with most succumbing to pulmonary sepsis or progression of disease. When reviewing the literature, one finds few series reporting more than 50 patients, and most reports describe approximately 12 to 16 patient. To evaluate the management options available to help deal with the patient with a malignant esophagorespiratory fistula, we reviewed and analyzed 207 patients with this problem at our institution.

Material and Methods

A search of our medical records from 1926 to 1988 was conducted, and 212 patients with esophagorespiratory fistula were identified. These records were reviewed, and five fistulas were determined to be benign and excluded from analysis. Therefore, data of 207 patients with malignant esophagorespiratory fistula were available for review and analysis, and make up this report.

Actuarial survival was calculated by Kaplan-Meier method [6]. All survival curves are calculated from initiation of therapy of the malignant esophagorespiratory fistula. Differences in survival were determined by log-rank analysis [7]. Significance was defined as a p value less than 0.05.

Results

For the 207 patients with malignant esophagorespiratory fistula, age ranged from 21 to 90 years (median, 59 years). There were 152 men and 55 women (male/female ratio approximately 3:1).

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Table 1. Modality Used to Treat the Primary Malignant Neoplasm[a]

Modality	Esophagus	Lung	Other
Radiation therapy	112 (70)	24 (73)	10 (77)
Resection	13 (8)	7 (21)	3 (23)
Chemotherapy	8 (5)	1 (3)	0
Bypass	3 (2)	0	0
Exclusion	3 (2)	0	0
Supportive care	22 (14)	1 (3)	0
Total	161	33	13

[a] Numbers in parentheses are percentages.

Primary Malignant Neoplasm

The majority of the patients had an esophageal primary tumor (161/207, 78%), of which 28 (17%) were cervical, 114 (71%) upper thoracic, and 19 (12%) lower thoracic. During this same period 3,543 patients were seen with primary esophageal malignant neoplasms; therefore, the incidence of fistula occurring in patients with esophageal malignant neoplasms in our series is 4.5% (161/3,543). Of the 161 patients with fistula and an esophageal primary tumor, 152 (94%) had squamous cell carcinoma, 5 (3%) adenocarcinoma, 3 (2%) leiomyosarcoma, and 1 (1%) primary melanoma.

Primary lung carcinoma accounted for 16% (33/207) of the fistulas, of which 18 (55%) arose in a main bronchus and 15 (45%) in lung parenchyma. During this study period 10,840 patients were seen with primary lung carcinoma; therefore, the incidence of fistula in patients with primary lung carcinoma in our series is 0.3% (33/10,840). Of the 33 patients with fistula and a lung primary tumor, 19 (58%) had squamous cell carcinoma, 12 (36%) adenocarcinoma, and 2 (6%) oat cell carcinoma.

A fistula developed in 5 primary tracheal neoplasms, of which 3 (60%) were squamous cell carcinoma, 1 (20%) anaplastic carcinoma,and 1 (20%) adenocarcinoma. There were 4 patients with metastatic mediastinal lymph nodes as the underlying cause of the fistula; 2 were secondary to metastatic breast carcinoma and 2 to Hodgkin's disease. There were 3 primary laryngeal squamous cell carcinomas and 1 patient with diffuse histiocytic lymphoma arising in the thyroid as the underlying malignant cause of the fistula.

Table 2. Signs/Symptoms Related to the Fistula[a]

Sign/Symptom	Number	Percent
Cough	116	56
Aspiration	77	37
Fever	52	25
Dysphagia	39	19
Pneumonia	11	5
Hemoptysis	10	5
Chest pain	10	5

[a] Twenty-one patients were asymptomatic.

Treatment of the primary malignant neoplasm was radiation therapy in 146 of the 207 patients (71%), resection in 23 (11%), and chemotherapy in 9 (4%) (Table 1). Of interest, 29 patients (14%) did not receive antineoplastic therapy, but either had supportive care alone (n = 22), exclusion (n = 3) or bypass (n = 3) as the only therapy of their primary malignant process.

Of the patients in whom the dose of radiation to the primary tumor was available (n = 126), the median was 4,766 cGy: 4,808 for esophageal (n = 101), 4,438 for lung (n = 20), and 5,220 for the other (n = 5) primary malignant neoplasms.

Malignant Esophagorespiratory Fistula

Symptoms of the malignant esophagorespiratory fistula occurred in 186 of 207 patients (90%); in 21 (10%) it was asymptomatic, being found serendipitously or at autopsy. Cough (56%), aspiration (37%), and fever (25%) were the most common symptoms, and most patients had multiple complaints (Table 2). The average duration of symptoms from onset to diagnosis was 12 days (median, 3 days; range, 0 to 238 days).

The diagnosis of an esophagorespiratory fistula was made by a single modality in 170 patients (82%) and included: contrast radiography (99/170, 58%), clinical grounds (methylene blue) (42/170, 25%), autopsy (15/170, 9%), esophagoscopy (8/170, 5%), and bronchoscopy (4/170, 2%). The remaining patients had documentation by more than one of the above diagnostic modalities.

The respiratory sites of the esophagorespiratory fistula in the 207 patients were as follows: The trachea accounted for 53% (110/207) and the left and right main bronchi for 38% (79/207) of the fistulas (left main bronchus, 46, right, 33). In 13 of 207 patients (6%) lung parenchyma itself was the site of fistulization. In 5 patients there were two sites of fistulization.

The median time from treatment of the primary malignant neoplasm to development of a fistula was 347 days. For those treated with radiation therapy (n = 146) it was 367 days, for chemotherapy (n = 9) 291 days, and resection (n = 23) 575 days. For those patients whose primary

Table 3. Therapy Directed at the Malignant Esophagorespiratory Fistula

Therapy	Number	Percent
Supportive care	104	50
Esophageal exclusion	29	14
Esophageal bypass	28	13
Radiation therapy	20	10
Esophageal intubation	14	7
Chemotherapy	5	2
Resection	5	2
Direct closure	2	1
Total	207	

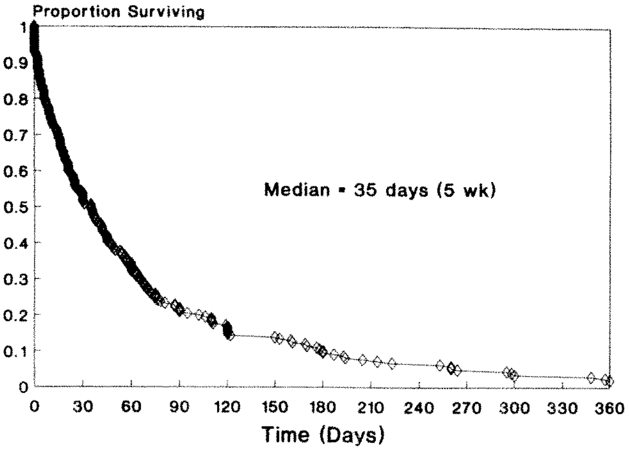

Fig 1. Overall survival of 207 patients with malignant esophagorespiratory fistula.

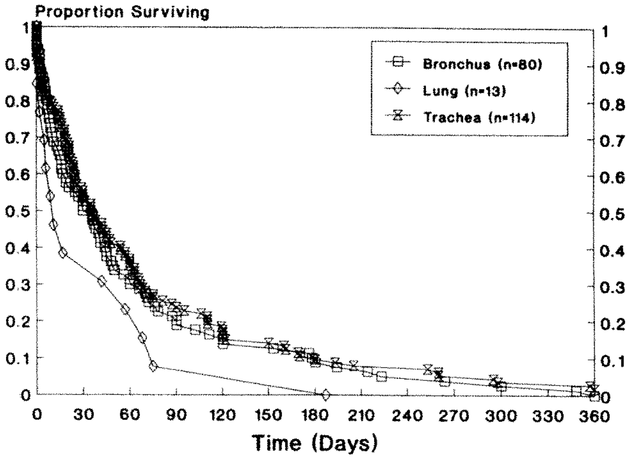

Fig 3. Actuarial survival curves of patients by respiratory location of fistula; there are no significant differences in survival.

neoplasm was untreated (n = 23), the median time to fistulization was 43 days.

Treatment of Fistula and Survival

The methods of treatment are listed in Table 3. The overall survival of the 207 patients is depicted in Figure 1. As shown, the median survival is 35 days (5 weeks).

We analyzed various parameters to search for factors that might predict a good or poor prognosis. Age, sex, site of the primary malignant neoplasm (Fig 2), and respiratory location of the fistula (Fig 3) were not significant factors in predicting favorable or unfavorable outcome. When we compared the survival in those patients receiving supportive care (median, 22 days) (n = 104) to those receiving specific therapy directed at the fistula (median, 47 days) (n = 103), survival was significantly longer in those receiving specific therapy ($p < 0.001$) (Fig 4).

One half of the patients (104/207) received only supportive care. Supportive care was defined as one or more of the following: intravenous hydration, antibiotics, jejunostomy, gastrostomy, ligation of cardia, esophagostomy, nasogastric tube, or tracheostomy. To be considered in the supportive care group, no combination of the above was allowed to functionally exclude the fistula.

Twenty-five patients (12%) underwent antineoplastic therapy as the primary therapy directed toward the malignant esophagorespiratory fistula, without mechanical diversion of the fistula. Patients who received chemotherapy (n = 5) alone had a median survival of 71 days (range, 2 to 187 days). This was not significantly better than supportive care, but the number receiving chemotherapy alone is small. There were 20 patients (10%) who received radiation therapy as treatment of their malignant esophagorespiratory fistula. Fifty percent of these had received radiation therapy for their primary neoplasm. The dose of radiation used to treat the fistula was available in 16 patients; the median was 3,151 cGy (range, 735 to 6,100 cGy). The survival of these 20 patients (median, 57 days)

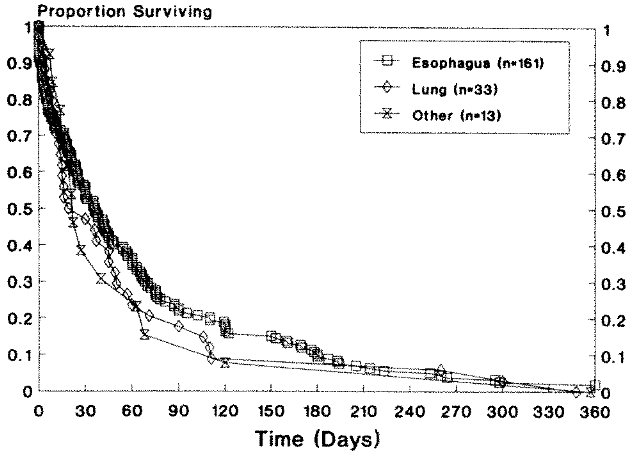

Fig 2. Actuarial survival curves of patients by primary neoplasm; there are no significant differences in survival.

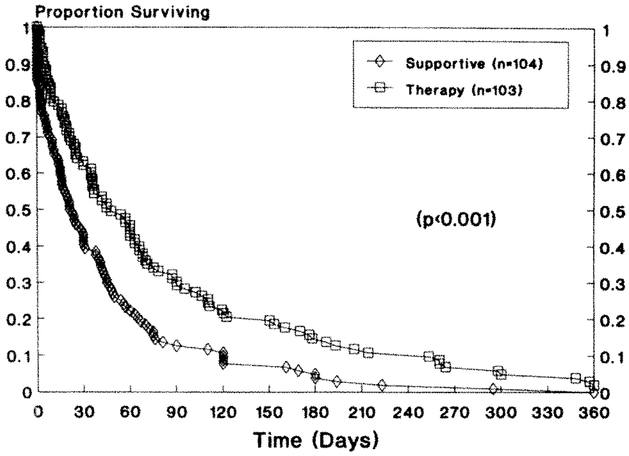

Fig 4. Actuarial survival of the 103 patients given specific therapy directed at the fistula versus the 104 patients given supportive care only. There is a significant prolongation of survival in those given specific therapy.

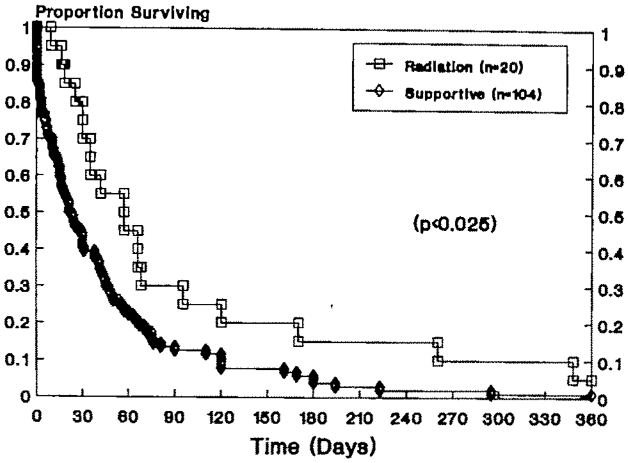

Fig 5. Actuarial survival of 20 patients with esophagorespiratory fistula treated with radiation therapy compared with 104 patients receiving supportive care. The survival of those receiving radiation therapy is significantly longer.

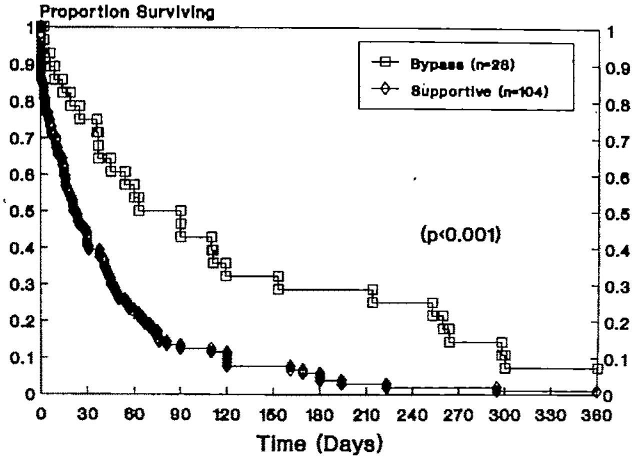

Fig 7. Actuarial survival of the 28 patients undergoing esophageal bypass compared with those undergoing supportive care. The patients undergoing bypass have a significantly longer survival.

was significantly longer than that of those treated with supportive care (median, 22 days) ($p < 0.025$) (Fig 5).

Seventy-eight patients (38%) had therapy directed toward mechanically preventing continued soilage of the respiratory tract. Twenty-nine patients (14%) underwent esophageal exclusion. This included esophagostomy and gastrostomy with closure of the esophagus above and below the fistula site. Figure 6 depicts the survival (median, 29 days) of these patients compared with that of those treated with supportive care; there was no significant difference.

Twenty-eight patients (13%) underwent esophageal bypass using either stomach ($n = 12$) or colon ($n = 16$). The survival (median, 77 days) in this group was significantly longer compared with that of those undergoing supportive care ($p < 0.001$) (Fig 7). Of note, the patients undergoing gastric pull-up survived significantly longer (17% at 1 year) than those having colon interposed (0% at 1 year) ($p = 0.04$).

Figure 8 demonstrates that the survival of the 14 patients undergoing placement of an esophageal endoprosthesis (median, 35 days) is not significantly different than that of patients who received supportive care alone.

Resection was performed in 5 patients. Two had esophagectomy with pulmonary resection to include the fistula to lung parenchyma. Three had very high cervical fistulas and underwent pharyngolaryngoesophagectomy. Survival ranged from 9 to 75 days (median, 25 days) in this select group of patients.

Direct closure of the fistula was attempted in 2 patients. Fibrin glue was attempted in 1; this eventually failed and the patient died 110 days afterwards. In another, the fistula was closed with a split-thickness skin graft, which failed.

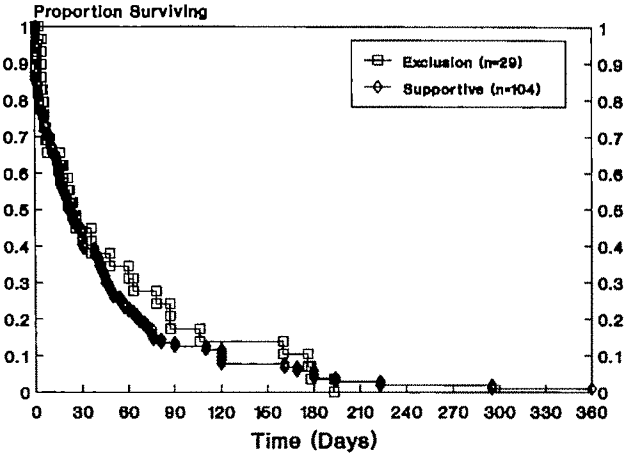

Fig 6. Actuarial survival of the 29 patients undergoing esophageal exclusion compared with those undergoing supportive care. There is no significant difference.

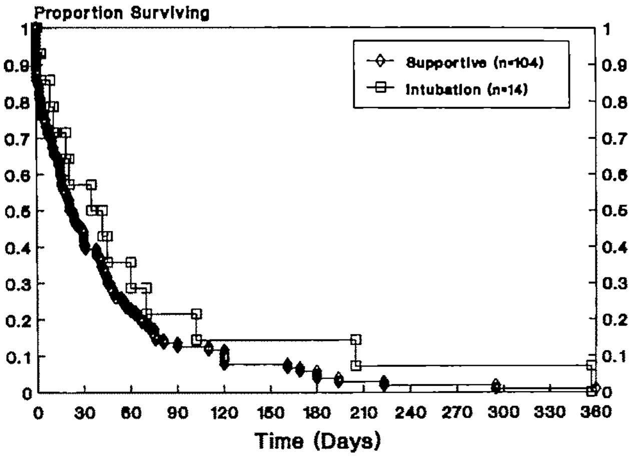

Fig 8. Actuarial survival of the 14 patients undergoing placement of an esophageal endoprosthesis compared with those having supportive care. There is no significant difference in survival.

Table 4. Treatment Mortality: 30-Day

Treatment	Number	Percent
Resection	3/5	60
Supportive care	58/104	56
Exclusion	16/29	55
Intubation	6/14	43
Bypass	7/28	25
Radiation therapy	4/20	20
Chemotherapy	1/5	20
Direct closure	0/2	0
All therapies	95/207	46

Table 6. Cause of Death as Related to Therapy[a,b]

Treatment	Pneumonia	Other
Bypass (n = 20)	10 (50)	10 (50)
Exclusion (n = 25)	20 (80)	5 (20)
Supportive (n = 77)	68 (88)	9 (12)
Tube (n = 11)	10 (91)	1 (9)
Radiation (n = 15)	14 (93)	1 (7)

[a] Total less than 207 because only those whose specific cause of death could be ascertained are included. [b] Values in parentheses are percentages.

Table 4 lists the mortality within 30 days of start of therapy in 207 patients with malignant esophagorespiratory fistula. It demonstrates an overall 30-day mortality of 46%. Resection, exclusion, intubation, and supportive care have 30-day mortalities ranging from 43% to 60%. Esophageal bypass, radiation therapy, chemotherapy, and direct closure are slightly less morbid, with a 30-day mortality ranging from 0% to 25%.

The cause of death could be determined in 154 of the 207 patients (74%) and is listed in Table 5. Pulmonary sepsis was by far the leading cause of death, occurring in 82% of the patients. Bleeding, as the cause of death, occurred in 12% of patients. Table 6 correlates death due to pneumonia versus all other causes and the therapy of the fistula in the patients in whom the cause could be ascertained. It is of note that death from pneumonia is less frequent for those undergoing esophageal bypass compared with other therapies.

A very interesting correlation was found between the interval from diagnosis of fistula to time of therapy and survival and is illustrated in Figure 9. It demonstrates clearly that the earlier treatment is instituted after diagnosis of a fistula, the greater the number of patients surviving for 35 days or more.

Table 5. Cause of Death

Cause	Number	Percent
Pulmonary sepsis	126	82
Bleeding	19	12
Suicide	2	1
Sepsis	2	1
Myocardial infarction	1	<1
Peritonitis	1	<1
Aspiration	1	<1
Malnutrition	1	<1
Stroke	1	<1
Total	154[a]	

[a] Does not equal 207 because only those whose cause of death could be ascertained are included.

Comment

The development of a malignant esophagorespiratory fistula, at any point in the treated or untreated course of a malignant neoplasm, is a devastating and life-threatening complication [1]. Treatment should be instituted rapidly once the diagnosis is confirmed, because almost all patients die of continued soilage of the respiratory tract with resultant pulmonary sepsis and death. Palliation should be directed at diverting or obstructing the flow of saliva and gastric contents through the fistulous communication. Even if successful palliation is effected, the outcome of these individuals is measured in weeks, or occasionally, months [1–5, 8–13].

The incidence of malignant esophagorespiratory fistula in this series was 4.5% for primary malignant esophageal and 0.3% for primary malignant lung neoplasms. Data in the literature concerning the incidence of fistula formation with primary lung carcinoma are sparse, and a report of incidence could not be found. There are data concerning the incidence of fistula secondary to esophageal carcinoma, and this ranges from 4.3% to 8.1% [1–4, 11].

In our series, the majority of patients (approximately 70%) received radiation therapy as antineoplastic therapy of their original primary malignant neoplasm. In a retrospective analysis such as this, it is impossible to say for sure, but it appears that radiation therapy to a primary thoracic neoplasm, especially esophageal, may increase

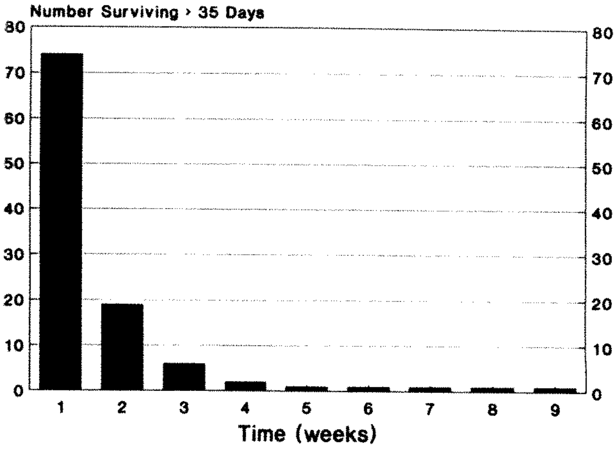

Fig 9. Number of patients surviving greater than 35 days versus the interval of time from diagnosis of fistula to start of therapy.

the incidence of malignant esophagorespiratory fistula in this population. Of note, we found that neither the site of the primary malignant neoplasm nor the respiratory site of the fistula had any impact on survival. It appears that once the fistulous communication develops between any portion of the esophagus and any site within the respiratory tract, the prognosis is dismal.

In our series of 207 patients, treatment of the malignant esophagorespiratory fistula was addressed by either a direct attack on the tumor causing the fistula (radiation therapy or chemotherapy) in 25 patients (13%) or an attempt to obstruct or divert contamination from the respiratory tract (esophageal exclusion, bypass, resection, direct closure, or intubation) in 78 patients (38%). One hundred four patients (50%) received only supportive care (nasogastric drainage, tracheostomy, gastrostomy, intravenous hydration, or antibiotics). When we compared the 103 patients having specific therapy aimed at the malignant esophagorespiratory fistula (whether antineoplastic or mechanical) with the 104 having supportive care, we found a significant increase in survival in those receiving specific therapy. More importantly, when we analyzed the timing of therapy in relation to the diagnosis of a malignant esophagorespiratory fistula, we found that if therapy was not instituted within a week of diagnosis, the ability to palliate the situation decreased dramatically.

When attempting to analyze the various treatment modalities to propose general treatment guidelines, we found that radiation therapy and esophageal bypass were the only treatments that significantly prolonged survival compared with supportive care. Within the marked constraints of a retrospective study, these data are obviously subject to interpretation. The following is a discussion of the results of each treatment compared with supportive care.

There were 5 patients who received chemotherapy as the only therapy after diagnosis of a malignant esophagorespiratory fistula. Although not significantly different than the survival of those receiving supportive care, the median survival of this group was 71 days compared with the overall median survival of 35 days and the median of 22 days for the group receiving supportive care. Recommendations concerning the use of chemotherapy in this condition cannot be made because of the almost anecdotal nature of the data, but it would not seem unreasonable to consider the use of chemotherapy in this group after adequate mechanical palliation of the fistula.

Reports of radiation therapy as the only treatment of a malignant esophagorespiratory fistula are sparse. In our series, 20 patients were treated with radiation with a significant increase in survival compared with supportive care alone. In a report by Yamada and associates [14], radiation therapy was used in 14 patients with a malignant esophagorespiratory fistula secondary to esophageal carcinoma. Two patients (14%) had long-term closure of the fistula, and 1 patient lived 74 months; the overall median survival was 150 days. Although permanent closure or healing of a malignant esophagorespiratory fistula after radiation therapy has been reported [1, 14], most patients have temporary relief. As in our series, survival is prolonged, but ultimately 83% die of pulmonary sepsis as the fistula reopens and soilage of the respiratory tract continues. One mechanism of the temporary benefit of radiation may be soft-tissue swelling in the area of the fistula with either temporary closure or substantial narrowing, and therefore, decreased contamination of the respiratory tract.

Mechanical obstruction or diversion of saliva and gastric contents has been advocated by many as the best palliation for a malignant esophagorespiratory fistula. There are few reports of resection or direct closure in this setting. As noted in the Results section, 2 patients in our series had an attempt at direct closure, 1 with fibrin glue and 1 with a split-thickness skin graft. Both methods failed and cannot be recommended. Five patients had resection of the fistula either by pharyngolaryngoesophagectomy for very high cervical fistula (n = 3) or by esophagectomy with resection of lung (n = 2). These have to be considered heroic attempts at palliation with high operative mortality and poor long-term results.

Esophageal exclusion by cervical esophagostomy and gastrostomy with closure of the esophagus above and below the fistula would allow diversion of saliva and gastric contents. Although it would seem that this should allow palliation and therefore prevent pulmonary sepsis and death, this was not the case in our 29 patients undergoing exclusion. There was no significant prolongation of survival compared with the supportive care group, and 80% still died of pulmonary sepsis. In our retrospective analysis we could not determine whether this group was a "sicker" group, and therefore, did not benefit from exclusion. But in this regard, this group had no higher incidence of pneumonia, fever, or aspiration when first seen than those undergoing any other treatment. Although exclusion would appear to mechanically palliate the fistula, normal intake of nutrients obviously is impaired and therefore palliation of swallowing is inadequate. This has been confirmed by other reports using exclusion as palliation [2, 3].

Placement of an esophageal endoprosthesis was used in 14 of our patients with relatively poor results. There was no prolongation of survival compared with supportive care: 43% died within 30 days of placement, and 91% died of pulmonary sepsis. These data would suggest that, at least in our hands, the ability to obstruct the flow of saliva and gastric contents through the fistula was not obtained by placement of an esophageal endoprosthesis. Although there are reports of better success in larger numbers of patients, the mortality ranges from 25% to 48% [9]. In addition, the impact on palliation in the survivors is unclear [9, 15–17].

Esophageal bypass with gastric, colonic, or jejunal interposition has been reported by others [1–4, 8, 10–13, 18–21]. For the most part, these reports favor esophageal bypass as the palliation of choice for patients with malignant esophagorespiratory fistula who are able to tolerate operation, except one [18], which reported a mortality of 50%. In our series, 28 patients underwent esophageal bypass with gastric (n = 12) or colonic (n = 16) interposition. This group had a significantly prolonged survival compared with the supportive care group and with all other treatment groups except those receiving radiation therapy. The procedure carried a 30-day mortality of 25%, which was equal to that of radiation therapy but significantly less than that of resection, exclusion, or intubation. It is hard to judge effective palliation in this large retrospective series, but after bypass only 50% of the patients died of pulmonary sepsis and 50% of other causes. Eighty percent or more of the patients undergoing other therapies died of pulmonary sepsis. We, therefore, think effective mechanical palliation was achieved with esophageal bypass, although with a high but not prohibitive initial mortality in this difficult group of patients.

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DISCUSSION

DR EARLE W. WILKINS, JR (Williamstown, MA): If I were to summarize this report, I would start by saying that there are three unarguable facts. To quote their leading sentence, "The development of a malignant esophagorespiratory fistula is a devastating complication," and I like the phrase they have added, "for both patient and physician." Second, the therapy is directed at palliation, which includes interruption of contamination of the pulmonary tree and, ideally, restoration of the ability to swallow. Third, the results in general have been dismal. It is a tough subject.

The report purports to show, first of all, that radiation and esophageal bypass are significantly better methods of therapy, but I would remind you that there were only 20 patients with irradiation and 28 undergoing bypass. That is a total of only about 20% of the patients involved. The second point is that the earlier the treatment is instituted after diagnosis of fistula, the better the results.

My thoughts are these. First of all, I would guess that the results after bypass are better because the patient selection is better. These are the patients who are less sick, who have less extensive disease, and who are judged able to undergo this extensive operation. Second, in the question of radiation therapy, I really cannot comment because our radiation therapy department categorically refuses to see these patients unless you do a bypass first.

My own bias in this field is to individualize patients. First, I would consider radiation and chemotherapy for a restricted few, those with oat cell carcinoma, those with lymphoma—there were 2 of each in this series—or possibly those with metastatic breast carcinoma, of which also there were 2. I might use the endoesophageal prosthesis if the tumor were bulky and I thought it could be properly seated. The problem with these tubes, however, is that they tend not to stay in place and they tend to enlarge the fistula and often increase the magnitude of the problem. I would consider an en bloc resection in an occasional case, particularly where the fistula is into the parenchyma of the lung, and would consider the possibility in certain patients of doing an esophagolobectomy. My greatest experience has been with the use of bypass, but the patient has to be otherwise healthy, in decent nutritional status (which usually means early diagnosis), and free of distant metastases.

I was asked belatedly to discuss this report and received it just this weekend, so I am not up to date on our own statistics, but in a 1980 report on our first 100 cases of using the colon to bypass the esophagus, there were 3 patients who had malignant fistulas of this type. All survived operation; they were in the hospital an average of 9 days, and they survived an average of 3 months. Is this good treatment or is this good judgment? I really cannot answer that.

There is an enormous amount of data in this report. Unfortunately, I am not sure that we in the audience can take away information on how we treat the next patient we see tomorrow. So I would ask Dr Burt, as this represents a series over 60 years, is the incidence increasing, and could you tell us how you go through your thought process on taking care of the next patient you see with this complication?

DR BURT: I thank Dr Wilkins for his comments and would like to address his questions. As far as the incidence of malignant esophagorespiratory fistula increasing, at our institution in the early part of this series we saw approximately 3 patients per year with this problem; presently we are seeing 5 patients a year.

As far as the selection of which patients receive what therapy in this series, obviously it is biased. It is a retrospective analysis of a large number of patients over a long period of time. I can say that before 1970, 70% of the patients received supportive care. Since 1970, 70% of the patients have received some form of therapy directed at the fistula, either by mechanical or antineoplastic therapy.

The efficacy of radiation therapy was somewhat of a surprise. There is a report by Yamada and associates [14] in which they reported 14 patients receiving radiation therapy. Two patients were long-term survivors, 1 surviving 74 months.

As far as how to treat the next patient, I agree completely with Dr Wilkins that it has to be individualized. The patient presenting to you with pulmonary sepsis late in the course of the fistula obviously should be supported, and no heroic measures should be done. The patient seen 2 or 3 days after diagnosis of a fistula in otherwise good condition should be offered gastric bypass with esophageal exclusion. This appears to be able to palliate these patients and seems to prolong their survival.

Weight Is Not an Accurate Criterion for Adult Cardiac Transplant Size Matching

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Owing to the limited availability of donor hearts, standard donor criteria for heart size matching need to be reexamined. The current practice at most centers is to match the donor's body weight to within ±20% of the recipient's. Our hypothesis was that minimal differences exist in heart sizes of the adult donor population, and therefore, the donor pool could be expanded for any given patient. M-mode echocardiographic measurements of left and right ventricular internal dimensions, left ventricular mass, and percent fractional shortening were reviewed in 235 normal adult subjects (101 men, 134 women). Low correlation coefficients and a high degree of variance were consistently observed between cardiac parameters and body size. There were no significant differences in left ventricular internal dimension when women weighing 40 to 109 kg were compared with men weighing up to 80 kg. Left ventricular size was not statistically different among men weighing 50 to 99 kg. No difference was noted in right ventricular size among men and women. Echocardiography is a simple and accurate technique to assess cardiac dimensions. Body weight does not correlate well with adult cardiac size and should not be used as an exclusion criterion for a donor heart.

(Ann Thorac Surg 1991;52:1230–6)

As heart transplantation evolves into an established treatment modality for end-stage heart disease, the need to maximize donor utilization becomes critical. Limited donor availability contributed substantially to the recent plateau in the number of heart transplantations performed in 1989, as compared with the period of exponential growth during the early 1980s [1].

Investigators have advocated the extension of donor criteria to meet the increasing lists of recipients. Traditional dogmas of high-risk variables have come under serious scrutiny. These include donor age, ischemic time, requirement for inotropic support, systemic infection, extent of coronary artery disease, and donor-recipient size mismatch [2–8].

The current practice at most centers is to match the donor's body weight to within ±20% of the recipient's [2, 4, 8, 9]. An inherent assumption to this criterion is that the donor/recipient body weight ratio adequately reflects the donor/recipient heart size ratio. Furthermore, definitions of "oversized" and "undersized" donor hearts, especially in the context of recipients with pulmonary hypertension, have not been standardized. Our hypothesis was that minimal differences exist in heart sizes of the adult donor population, and therefore, the donor pool could be expanded for any given recipient.

Material and Methods

Echocardiographic Measurements

M-mode echocardiography, a well-established technique for anatomic measurements of heart sizes and evaluation of cardiac function, was used in this study to assess cardiac parameters [10, 11]. M-mode measurements of left ventricular internal diameter at end-diastole and at end-systole (LVID-ED, LVID-ES), right ventricular internal diameter at end-diastole (RVID-ED), interventricular septal thickness at end-diastole (IVS-ED), left ventricular posterior wall thickness at end-diastole (LVPW-ED), and left atrial and aortic root dimensions were obtained from echocardiograms performed in 235 normal adults (age range, 18 to 60 years) at the University of Virginia Health Sciences Center from 1988 to 1990. These subjects (134 women, 101 men) had no echocardiographic evidence of structural or functional heart disease. Weight ranged from 42 to 118 kg (mean, 73 ± 9.5 kg [standard error of the mean]). Height ranged from 152 to 196 cm (mean, 170 ± 6.2 cm), and body surface area (BSA) ranged from 1.37 to 2.46 m^2 (mean, 1.84 ± 0.01 m^2) [12].

Echocardiographic studies were performed with an Advanced Technology Laboratories Ultramark 9 ultrasound system using a 2.25-MHz transducer. Images of the heart were obtained with the patient in the partial left decubitus position and the transducer in the standard chest interspace. M-mode echocardiograms were derived from the two-dimensional images under direct anatomical visualization and were recorded at 50 mm/s. Cardiac chamber dimensions and wall thicknesses were measured on the M-mode by on-line analysis in accordance with the

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Table 1. Distribution of Physical and Cardiac Parameters for All Subjects (n = 235)

Variable	N	Mean	SEM	Range
Height (cm)	235	170	0.6	152–196
Weight (kg)	235	73	0.9	42–118
BSA (m^2)	235	1.84	0.01	1.37–2.46
LVID-ED (mm)	198	50	0.37	32–70
LVID-ES (mm)	199	31	0.30	20–42
RVID-ED (mm)	122	20	0.38	12–34
IVS-ED (mm)	199	9.3	0.12	5.4–12
LVPW-ED (mm)	199	8.8	0.11	5.6–12
FS (%)	198	37	0.45	16–60
LV mass (g)	198	205	3.78	68–374
LA (mm)	162	36	0.38	22–48
AO (mm)	229	29	0.29	20–42

AO = aortic root; BSA = body surface area; FS = fractional shortening; IVS-ED = interventricular septal thickness at end-diastole; LA = left atrium; LV = left ventricular; LVID-ED, LVID-ES = left ventricular internal diameter at end-diastole and end-systole; LVPW-ED = left ventricular posterior wall thickness at end-diastole; RVID-ED = right ventricular internal diameter at end-diastole; SEM = standard error of the mean.

Table 3. Linear Regression Analyses of Cardiac Parameters[a]

Parameter	LVID-ED	RVID-ED	%FS	LV Mass
Weight				
r	0.43	0.28	0.25	0.45
r^2	0.17	0.09	0.06	0.20
Slope	0.16	0.08	0.11	1.69
Height				
r	0.48	0.31	−0.04	0.23
r^2	0.24	0.11	0.02	0.05
Slope	0.29	0.14	−0.09	1.32
BSA				
r	0.52	0.34	0.18	0.43
r^2	0.26	0.13	0.02	0.19
Slope	14.6	7.05	4.22	117.7

[a] Correlation (r), variance (r^2), and slopes when body size was related to cardiac parameters. Low correlation and high variance (low r^2) were consistent observations.

BSA = body surface area; LV = left ventricular; LVID-ED = left ventricular internal diameter at end-diastole; %FS = percent fractional shortening; RVID-ED = right ventricular internal diameter at end-diastole.

recommendations of the American Society of Echocardiography [13]. Left ventricular mass was calculated from the following equation [14, 15]: LV mass = 1.05 [(LVID-ED + IVS-ED + LVPW-ED)^3 − (LVID-ED)^3]. Percent fractional shortening (%FS) as an estimate of left ventricular function was calculated from the following equation [16, 17]: %FS = (LVID-ED − LVID-ES)/LVID-ED × 100%. All M-mode studies were performed by two experienced echocardiographic technicians.

Statistical Analysis

Correlation of cardiac parameters to weight (in 10-kg increments), height (in 10-cm groups), and BSA (in 0.2-m^2 groups) was determined using linear regression lines constructed with the least-squares method [18]. Index of association between the variables was measured by Pearson's correlation coefficient (r). The strength of the linear relationship between the variables as an indication of variance (or degree of scatter) was expressed by the square of the sample correlation coefficient (r^2). Properties of the regression line were described by the intercept and slope. Slope is defined as a function of r and variance [r = (slope)(standard deviation of x/standard deviation of y)]. Significance of the slope as different from zero was determined with Student's t test. Data sets were also analyzed using analysis of variance with Duncan's multiple range tests for comparison of means. Differences were considered significant when the p value was 0.05 or less.

Results

Tables 1 and 2 show the distribution of physical and cardiac parameters used in the data analysis. Maximum difference in LVID-ED in men and women was 3 cm and

Table 2. Distribution of Physical and Cardiac Parameters for 101 Men and 134 Women

	N		Mean		Range	
Variable	M	F	M	F	M	F
Height (cm)	101	134	178	165	157–196	152–180
Weight (kg)	101	134	80	68	55–118	42–107
BSA (m^2)	101	134	1.97	1.74	1.59–2.46	1.37–2.13
LVID-ED (mm)	85	113	53	48	40–70	32–55
RVID-ED (mm)	82	40	21	18	12–34	12–29
FS (%)	85	113	38	37	16–52	19–60
LV mass (g)	85	113	225	190	122–374	68–313

BSA = body surface area; FS = fractional shortening; LVID-ED, RVID-ED = left and right ventricular diameter at end-diastole.

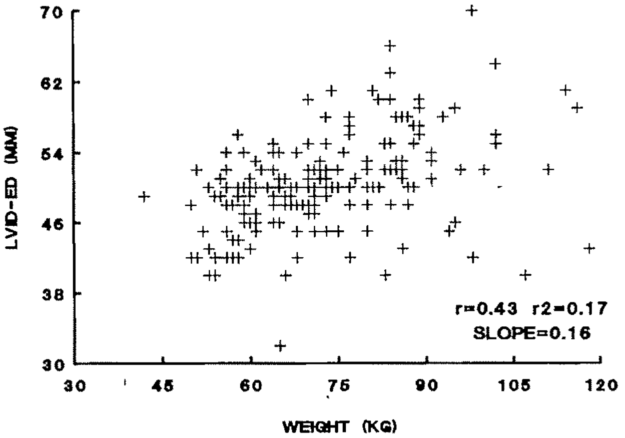
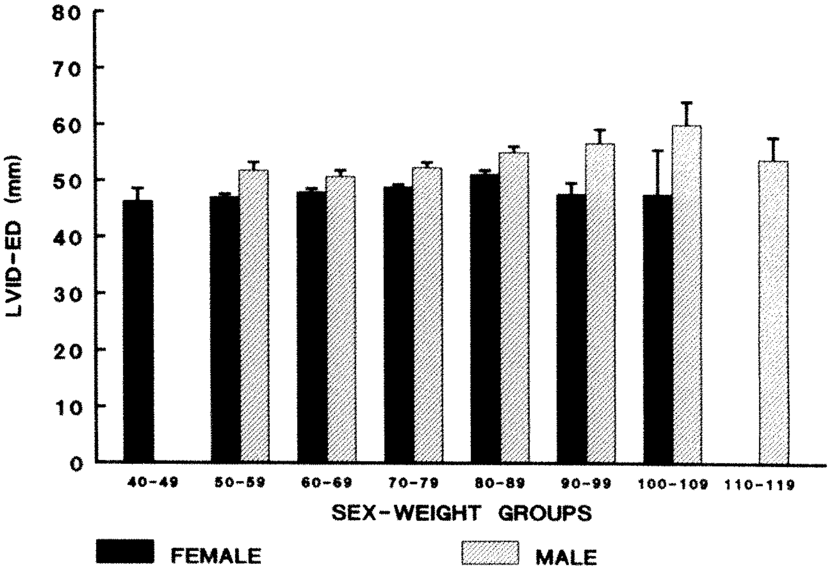


Fig 1. Scattergram of left ventricular internal diameter at end-diastole (LVID-ED) versus body weight. (r = coefficient of correlation; r^2 = index of variance.)

Fig 2. Comparison of left ventricular internal diameter at end-diastole (LVID-ED) with body weight at 10-kg increments for men and women. No significant differences were noted among women of any weight group (40 to 109 kg) and when all women were compared with men weighing 50 to 80 kg. No differences were observed in left ventricular size in men weighing 50 to 99 kg. Comparisons were made with Duncan's multiple range tests. Differences were considered significant when the value of p was 0.05 or less.



2.3 cm, respectively. Right ventricular inner dimension at end-diastole varied by 2.2 cm and 1.7 cm, respectively.

Table 3 summarizes the linear regression analyses of ventricular dimensions and left ventricular function. It demonstrates the relatively weak correlation between cardiac parameters and body size ($-0.04 < r < 0.52$). Variance is consistently high, with r^2 ranging from 0.02 to 0.26. Slopes are generally minimal, except for BSA, which exhibited markedly positive slopes with cardiac dimensions, %FS, and left ventricular mass.

The r values of 0.43 and 0.48 when LVID-ED was correlated with weight and height, respectively, indicate a weak association of left ventricular dimensions with these variables. The r^2 of 0.17 and 0.24 and slopes of 0.16 and 0.29 further supported a poor correlation in addition to a high degree of scatter (Fig 1). No significant differences in LVID-ED were noted among women of any weight group (40 to 109 kg) and when all women were compared with men weighing 50 to 80 kg (Fig 2). In addition, there were no differences in LV size in men weighing 50 to 99 kg (see Fig 2). When comparison was made with height groups, there were no differences in LVID-ED except for the tallest men (190 to 200 cm) and the shortest women (150 to 160 cm). Similarly, r values of 0.28 and 0.31 and slopes of 0.08 and 0.14 indicate poor correlation of right ventricular dimensions with weight and height, respectively (Fig 3). No significant differences were observed in RVID-ED among men and women of any weight and height group. Percent fractional shortening also correlated poorly with weight and height ($r \leq 0.25$) (Fig 4). Percent fractional shortening did not differ significantly among men and women except for the largest (100 to 109 kg) and smallest women (40 to 49 kg).

Relationship of right and left ventricular dimensions and left ventricular function to BSA exhibited markedly positive slopes (4.2 to 117), even though the degree of correlation remained relatively weak, as r ranged from 0.18 (%FS) to 0.52 (LVID-ED) (see Table 3). This is consistent with the definition of slope as a function of

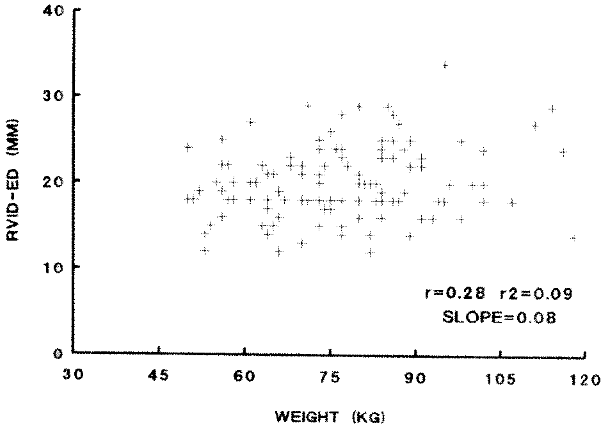


Fig 3. Scattergram of right ventricular internal diameter at end-diastole (RVID-ED) versus body weight. (r = coefficient of correlation; r^2 = index of variance.)

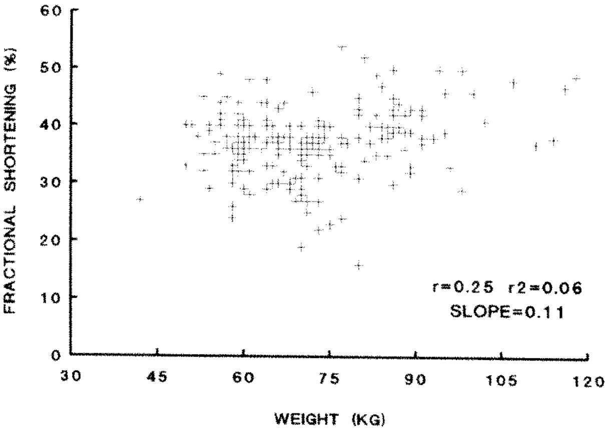


Fig 4. Scattergram of percent fractional shortening versus body weight. (r = coefficient of correlation; r^2 = index of variance.)

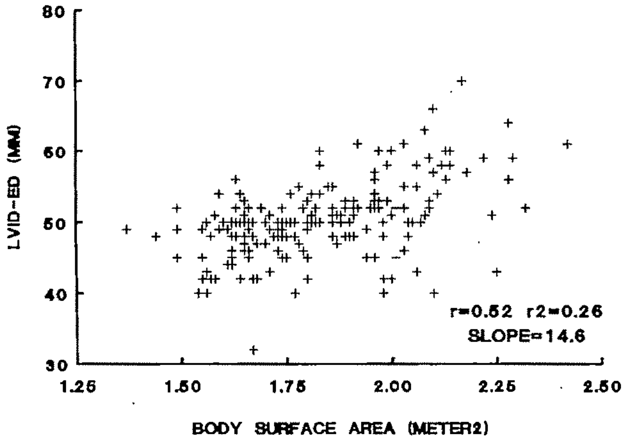


Fig 5. Scattergram of left ventricular internal diameter at end-diastole (LVID-ED) versus body surface area. (r = coefficient of correlation; r^2 = index of variance.)

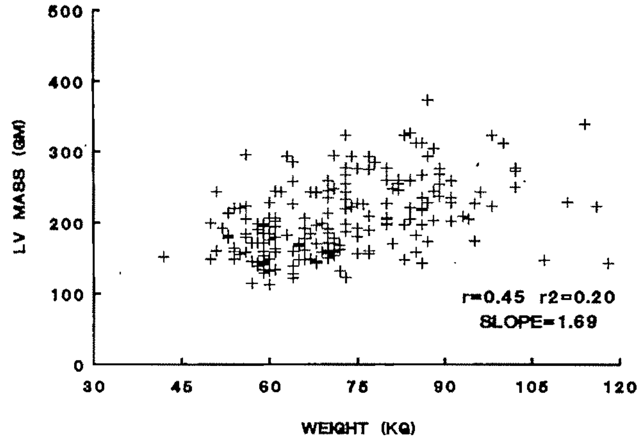


Fig 6. Scattergram of left ventricular (LV) mass versus body weight. (r = coefficient of correlation; r^2 = index of variance.)

both r and variance [18]. Low r^2 values from 0.02 (%FS) to 0.26 (LVID-ED) indicate that a high degree of scatter was present (Fig 5). No significant differences were noted in LVID-ED, RVID-ED, and %FS among men and women when grouped by BSA, except when comparing the largest men (2.25 to 2.5 m^2) with the smallest women (1.2 to 1.5 m^2). Our findings are consistent with data in the cardiology literature that show that left ventricular measurements are most closely related to BSA [11, 19]. Similarly, left ventricular mass displayed markedly positive slopes when related to weight, height, and BSA, even though the correlation (r) was relatively weak (Fig 6). Aside from the extremes of weight (women, 40 to 49 kg; men, >100 kg), no significant differences were noted in left ventricular mass among male and female subjects (Fig 7).

Comment

The shortage of donor hearts demands that the standard criteria for adult cardiac transplant size matching be reexamined. The practice of matching donor heart size based on ±20% of the recipient's body weight may be due to tradition rather than to scientific data.

We have provided echocardiographic evidence to suggest that physical parameters such as body weight, height, and BSA do not correlate well with cardiac dimensions and with left ventricular function. Low correlation coefficients, high degree of variance, and minimal slopes were consistently observed. Specifically, in regard to body weight, there was no significant difference in LVID-ED when women of all weight groups were compared with men weighing 50 to 80 kg. Furthermore, left ventricular size was not statistically different in men weighing 50 to 99 kg. No difference was noted in right ventricular size among men and women, irrespective of body weight.

Although this is primarily an anatomic study, we have attempted to provide some data correlating left ventricular function with physical parameters. Both %FS and left ventricular mass correlated poorly with weight and height. Except for the largest (100 to 109 kg) and smallest

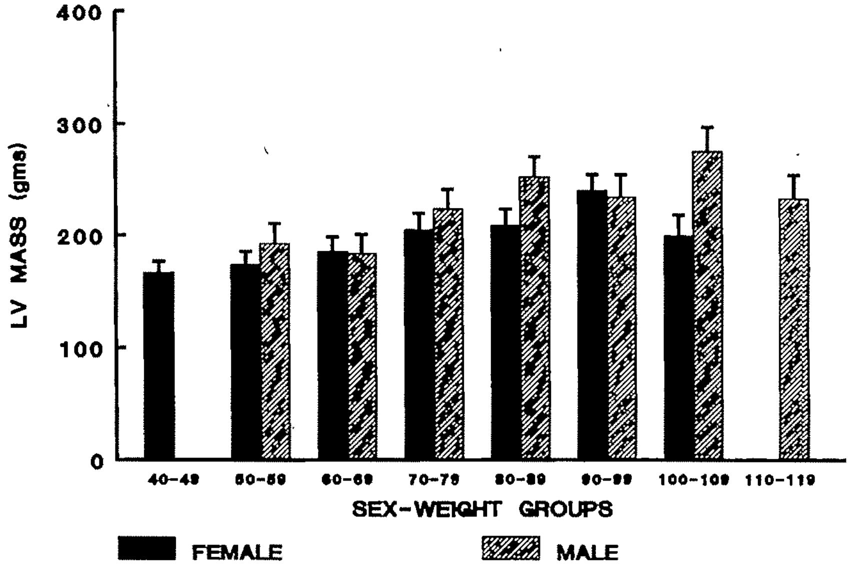


Fig 7. Comparison of left ventricular (LV) mass with body weight at 10-kg increments. No significant differences were noted among male and female subjects aside from the extremes of weight: 40 to 49 kg for women and greater than 100 kg for men. Comparisons were made with Duncan's multiple range tests. Differences were considered significant when the value of p was 0.05 or less.

(40 to 49 kg) women, there were no differences observed in %FS among men and women of all weights and heights (see Fig 6). Concern for the ability of a heart from a relatively "smaller" donor to generate adequate cardiac output in light of these data may therefore not be valid. It has been observed that cardiac output and hemodynamics are maintained at levels appropriate for recipient size after heart transplantation using an "undersized" heart [20, 21].

Many investigators have been successful in using clinical cardiac allografts in the orthotopic position with a donor-recipient weight discrepancy greater than 20%. As early as 1970, Thomas and colleagues [22] demonstrated a remarkable survival in dogs after orthotopic heart transplantation of donor hearts that weighed 50% to 70% less than the recipient's heart weight. It is important to note in the study that donor-recipient heart weight ratio was used. Actual body weights of the dogs were not reported. More recently, Sweeney and associates [2] did not observe any correlation between the number of high-risk donor variables and a poor postoperative result. These variables included age greater than 40 years, systemic infection, prolonged cardiopulmonary resuscitation, ischemic time longer than 5 hours, and donor body weight more than 20% less than that of recipient. Guerraty and other [4, 5] have also been effective in using hearts from donors who weighed 50% less than the recipient. Large weight mismatches are often accepted in infant and pediatric heart transplantation owing to the scarcity of infant and pediatric donors [8]. Gundry and co-workers [8] reported in their series that infant donors averaged 60% larger than their recipients (range, 41% smaller to 308% larger); pediatric donors averaged 57% larger (range, 32% smaller to 238% larger).

On the other hand, Hosenpud and associates [20] suggested that broadening the standard donor to recipient body match criteria may have a negative impact on long-term cardiac allograft hemodynamics and function. Their data demonstrated that although cardiac output is maintained at levels appropriate for recipient size after heart transplantation, "smaller" hearts tend to rely on an increased heart rate and elevated filling pressure to achieve this end. Similarly, Schuler and associates [23] observed that a weight discrepancy greater than 20% may lead to deterioration in postoperative graft function.

Results of this study are also relevant to heart transplantation in recipients with elevated pulmonary vascular resistance. Most centers will use an "oversized" donor heart, or consider heterotopic heart transplantation, for patients with a pulmonary vascular resistance greater than 4 to 6 Wood units (or a transpulmonary gradient greater than 15 mm Hg) to prevent acute graft distention and right heart failure [24–28]. "Oversized" and "undersized" hearts have yet to be defined. Our data suggest the right ventricular dimensions correlate poorly with body weight and height. Furthermore, no significant differences were observed in RVID-ED among all weight and height groups, irrespective of sex, implying that the terms "oversized" and "undersized" may have been misused. Recent reports have challenged the physiology behind the use of these "mismatched" grafts, suggesting that the use of "oversized" donor hearts based on a donor/recipient weight ratio of +20% does not improve outcome of orthotopic heart transplant recipients with or without preoperative pulmonary hypertension. These reports also suggest that "undersized" hearts (>20% less than recipient's body weight) are not detrimental to allograft function and recipient survival, and do not produce restrictive hemodynamics at rest [22, 29].

Although the criteria for heart size matching remain controversial, there is an increasing body of literature to suggest that the donor/recipient weight ratio of ±20% may be excessively restrictive. One reason that this 20% figure may be irrelevant is that body weight does not predict the actual dimensions, mass, and function of a given heart. Echocardiography is now routinely performed in most centers to evaluate donor hearts for structural defects and for cardiac function [17]. It could be used to assess cardiac dimensions and heart size of donors who would otherwise have been excluded based on body weight discrepancy. Even though one cannot compare the recipient's cardiac dimensions with the donor's, our results suggest that minimal differences exist in heart sizes of the normal adult population. If there is an extreme difference in body weight or height, one may refer to standardized echocardiographic nomograms to estimate the expected average heart size for a given recipient [10]. In light of the critical shortage of donor hearts, our data provide evidence that body weight does not correlate well with adult cardiac size and therefore should not be used as an exclusion criterion for a donor heart.

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DISCUSSION

DR RICHARD J. NOVICK (London, Ont, Canada): I congratulate Dr Chan and associates on a very well-presented paper. At University Hospital in London, Ontario, Canada, we have performed 281 heart transplantations in the past 10 years. In about 15% of cases, the donor weighed less than 70% of the weight of the recipient. There were no significant differences in either perioperative mortality, 3-month mortality, or rest or exercise hemodynamic/performance at 1 year in these recipients as opposed to other patients who received a more evenly weight-matched heart. We would therefore generally support Dr Chan and associates' conclusions, except we would still hesitate to use an undersized heart in recipients with a very high pulmonary vascular resistance or if the estimated ischemic time is beyond 5 hours.

I have two questions for Dr Chan. First, did you measure the thickness of the right ventricular wall in patients of widely discrepant weights? Second, have you had any experience in correlating echocardiographic indices of donor heart size versus subsequent hemodynamic performance and inotropic requirement in heart transplant recipients?

DR CHAN: Thank you for your comments, Dr Novick. Pertaining to your first question, left ventricular mass was determined from the interventricular septal and posterior wall thickness. Analyzing these entities individually, we did not observe any significant differences except for the extremes of weight group. Right ventricular wall thicknesses were not analyzed because it is difficult to consistently measure right ventricular wall thickness from echocardiography owing to transducer artifacts.

In regard to your second question, we agree with you that the ideal study would be to prospectively correlate echocardiographic indices of donor heart size versus posttransplantation cardiac function. The limiting factor to the study would be the consistency in echocardiographic measurements, as most donor hearts are procured from outside hospitals. Furthermore, even though our report does not address the issue of appropriate heart size for recipients with pulmonary hypertension, our data do suggest that because there is such a poor correlation between body size and heart size, it may be difficult to assess which heart is oversized and which heart is undersized.

DR R. MORTON BOLMAN III (Minneapolis, MN): Our observation also has been that if you use a small heart in patients with high pulmonary vascular resistance, the patient may survive the operation but may end up with more tricuspid regurgitation than is usually the case.

DR HENRY M. SPOTNITZ (New York, NY): This is a very interesting study, although your data appear to be at variance with some of the early angiographic measurements. An alternate hypothesis would be that the problem here is not a lack of correlation but a lack of reproducibility of your echocardiographic measurements. What efforts did you make to try to be certain that your echocardiographic measurements were reproducible? For example, did you do cross-correlations between independent observers, did you do serial measurements of the same information in the same patient, and were echocardiographic measurements done by more than one observer in the course of the study?

DR CHAN: Dr Spotnitz, this was a retrospective study with the echocardiograms performed by the same two echocardiographic technicians. Cardiac parameters were measured according to the recommendations of the American Society of Echocardiography. Furthermore, I understand that there is some discrepancy between angiographic and echocardiographic determination of heart size. However, the purpose of this study was not to compare different techniques in the estimation of cardiac parameters. We believe that the same standard technique applied by the same two echocardiographers on an adequate sample size of 235 subjects should provide a high degree of confidence.

First Successful Bridge to Cardiac Transplantation Using Direct Mechanical Ventricular Actuation

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Currently available ventricular assist devices are technically difficult to implant, require continuous anticoagulation, and are associated with hemorrhagic and thromboembolic complications. Direct mechanical ventricular actuation is a biventricular assist device that can be applied in 3 to 5 minutes through a left anterior thoracotomy and has no direct blood contact or need for anticoagulation. The present study was designed to determine the effects of direct mechanical ventricular actuation in total biventricular circulatory support. Cardiogenic shock refractory to standard therapy developed in 2 patients awaiting cardiac transplantation. Direct mechanical ventricular actuation was applied and provided immediate hemodynamic stabilization in both. All inotropic agents and intraaortic balloon support were then discontinued. Fifty-six hours of circulatory support bridged the first patient to successful cardiac transplantation without complication. The patient is alive and well more than 1 year later without incident of infection or rejection. The second patient suffered cardiac arrest and required closed chest cardiopulmonary resuscitation before device application. After 45 hours of support, it was determined that irreversible neurologic injury had occurred and direct mechanical ventricular actuation was discontinued. Neither patient's native heart exhibited any histologic evidence of device-related trauma. Direct mechanical ventricular actuation has undergone limited clinical investigation since its original description 25 years ago, but in these initial trials, the device has proved effective. The concept of mechanically actuating the ventricles appears to be a valuable, yet under-utilized method of total circulatory support.

(Ann Thorac Surg 1991;52:1237–45)

The concept of applying rhythmic, external mechanical compression and decompression to both ventricles of the failing heart was first described in 1965 [1]. Extensive laboratory investigations have shown that direct mechanical ventricular actuation (DMVA) produces cardiac outputs of 80% to 110% of control values compared with outputs of 20% to 40% using open chest bimanual compression [1–6]. Animal studies also have shown that DMVA results in minimal epicardial trauma, does not require sophisticated electrophysiologic timing, and can produce normal cardiac outputs in the failing, asystolic, and even fibrillating heart. Conceptually, this technology is simple and elegant, and offers several advantages compared with other existing ventricular assist devices as well as partial cardiopulmonary bypass. First, there is no blood contact or need for anticoagulation, which should reduce thromboembolic, hemorrhagic, and infectious complications. Second, the support cup can be rapidly applied through a left anterior thoracotomy to achieve biventricular support within 3 minutes [1–6]. The anterior thoracotomy application preserves the sternal approach for subsequent cardiac operations including cardiac transplantation. Third, physiologic and pulsatile flow rates are rapidly achieved without the need for inotropic support.

Until the present study, clinical applications of DMVA were primarily limited to heroic efforts of resuscitation after prolonged periods of failed standard external cardiopulmonary resuscitation [2–6]. These initial clinical applications did not result in long-term survivors but did convincingly demonstrate that previous experimental results could be duplicated in humans.

The present investigation was designed to determine whether DMVA could be used to support patients with refractory cardiogenic shock who were otherwise candidates for cardiac transplantation. This particular group of patients was chosen to attempt to answer a number of important questions: can the technology provide prolonged circulatory support without systemic complications, is there serious trauma to the native heart, and finally, what are the present limitations in device design and construction that could lead to device failure? Based on extensive laboratory testing, the Institutional Review Board at Duke University Medical Center approved DMVA for use in patients with refractory cardiogenic shock or cardiac arrest including those awaiting cardiac transplantation.

Material and Methods

Direct mechanical ventricular actuation employs an elliptically contoured cup which fits over both ventricles. The cup consists of an outer shell or housing and an inner flexible diaphragm. The housing can be made of either a

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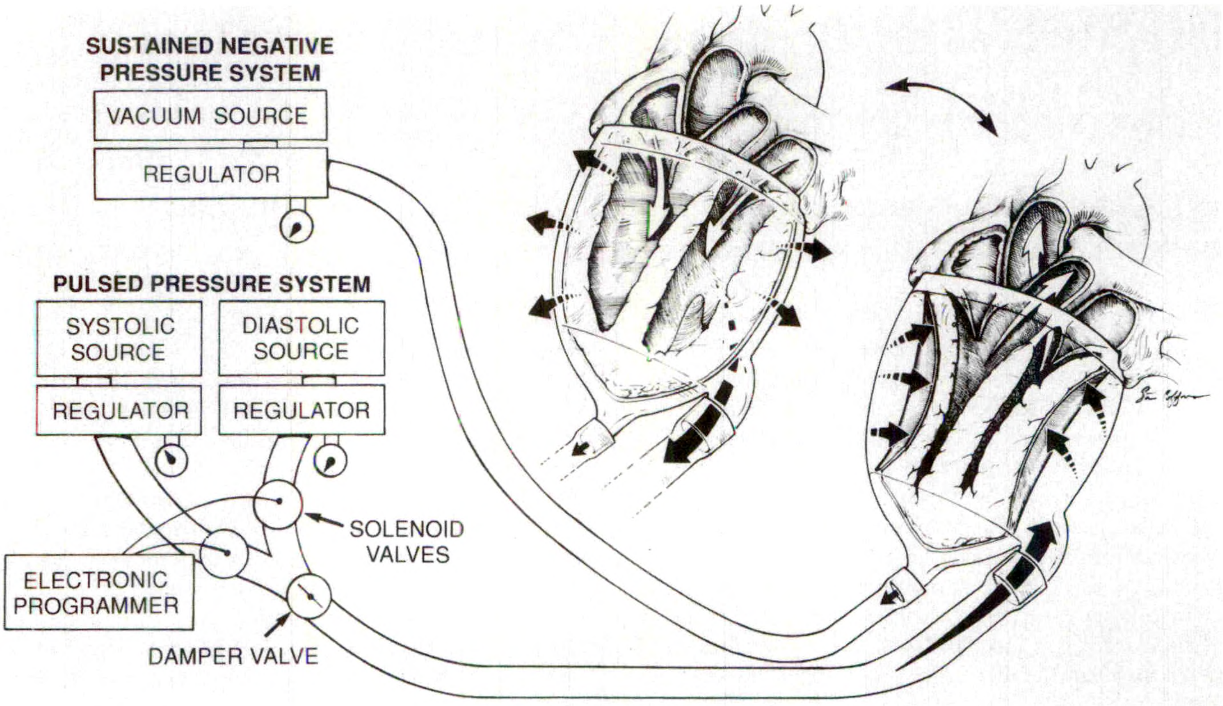

Fig 1. Schematic diagram of direct mechanical ventricular actuation drive system and cup. The cup is shown actuating the ventricles into both systolic (right) and diastolic (left) configurations.

hard translucent material (Pyrex) or a soft material (Dacron-reinforced Silastic) which produces a semirigid structure. The advantage of the soft shell is its ease of application through a smaller incision, while the rigid shell has the advantage of allowing visualization of the diaphragm's action on the heart. The flexible diaphragm currently used in both the hard and soft cups is a Silastic membrane (Dow Corning Corp).

The cup attaches itself to both ventricles via a continuous vacuum at the apex of the cup. Once the cup is in position, positive and negative pneumatic forces operate a diaphragm within the cup to "actuate" the ventricles into their normal systolic and diastolic configurations. Appropriate delivery of uniform positive and negative forces to the ventricles rapidly returns the failing, asystolic, or fibrillating heart to its function as a blood pump (Fig 1). The apical vacuum source provides device attachment, but more importantly and critical to the concept, it seals the actuating diaphragm to the myocardial surface. Consequently, the device not only compresses the ventricles to create systole but also enhances cardiac filling by decompressing the ventricles into a diastolic configuration.

The massaging action of DMVA is controlled by a pneumatic drive unit that has both pulsed pressure and sustained vacuum systems (see Fig 1). The vacuum system is responsible for device attachment and can be adjusted to the least degree of suction (approximately −70 mm Hg line pressure) that will maintain a constant diaphragm-to-epicardium seal. The pulsed pressure system consists of a positive pneumatic source for systolic compression and a negative pneumatic source for diastolic filling.

The internal diameter of each assist cup is identified by a numerical label in millimeters. An appropriate cup size is one that approximates the greatest transverse diameter of the unassisted heart. Cups varying in size from 90 to 150 mm in transverse diameter were prepared for the present study. This particular range was chosen by evaluation of chest radiographs of cardiac transplant candidates on the waiting list at Duke University Medical Center. Once a general size has been selected, the assist cup can rapidly be positioned through a small left anterior, sixth intercostal space thoracotomy. After the heart is adequately exposed, the device is positioned over the ventricular apex while set in a diastolic mode. The drive system's continuous vacuum source causes the device to aspirate itself onto the heart. Once attached, the drive system is switched into an actuating mode which cycles positive and negative forces from the pulsed pressure source resulting in ventricular systole and diastole. The process of DMVA application takes seconds after adequate exposure. The entire procedure has been performed in less than 3 minutes for emergency resuscitation in both the clinical [3–6] and the laboratory setting [1].

After acute hemodynamic stabilization, systolic (+120 mm Hg to +130 mm Hg) and diastolic (−100 mm Hg to −115 mm Hg) forces and flow rates can be adjusted to maximize hemodynamics. Cycle rates are generally set between 80 per minute (large hearts) to 120 per minute (small hearts). Adequate left ventricular emptying is achieved by systolic durations set at 50% of the

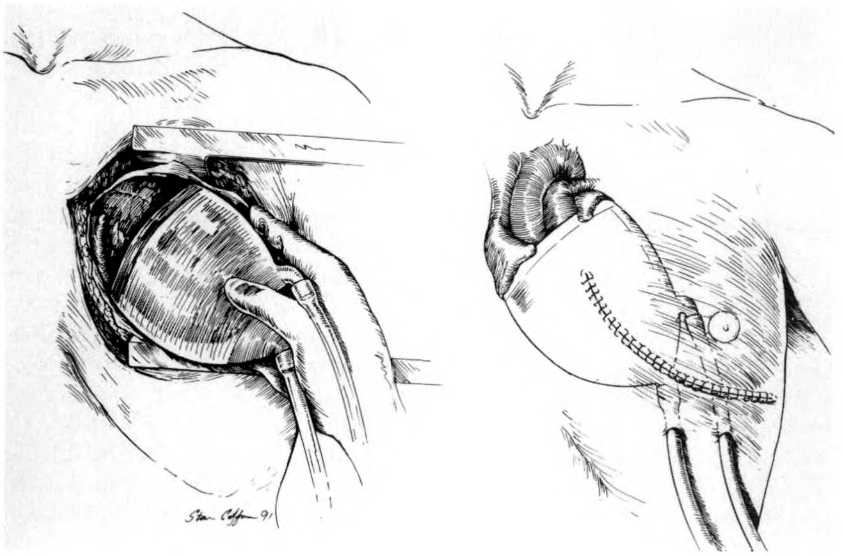

Fig 2. Cup application is made by exposing the heart through a left anterior thoracotomy; positioning the cup over the ventricular apex results in self attachment (left). The chest is then closed after the drive lines are brought out through separate stab incisions (right).

DMVA actuating cycle. This is important to prevent any potential for pulmonary edema, which could result if an abbreviated systolic force effectively emptied the right ventricle but not the left. Assurance of avoiding this situation is possible by monitoring pulmonary wedge pressures. An increase in wedge pressures during support indicates the need for increased systolic durations.

Other conceivable complications can be circumvented by the usual preparations for any mechanical failures. If a leak develops in the actuating diaphragm, it is easily detected by fluctuations in drive pressures and vacuum attachment pressures. Although such an event requires device replacement, initial hemodynamic stabilization can be maintained by increasing drive pressures. The possibility of a pneumothorax is prevented by placement of a chest tube at the time of initial thoracotomy. The pneumatic drive units are an unlikely source of mechanical failure but were monitored as with any other device. For these reasons, an additional drive unit and set of devices were available for emergency back-up in the present study.

Institutional Review Board approval was obtained in August 1989. After informed consent, DMVA was applied to 2 patients in whom refractory cardiogenic shock developed while they were awaiting cardiac transplantation. Patients with preexisting infection and multiorgan failure were excluded from the investigation.

Patient 1

In January 1990, a 56-year-old woman, diagnosed 2 years previously with an idiopathic dilated cardiomyopathy, was seen in acute congestive failure. The patient had been previously approved as a candidate for cardiac transplantation. Cardiac catheterization revealed an ejection fraction of 0.08 with normal coronary arteries, 3+ mitral regurgitation, and a pulmonary vascular resistance of 3 Wood units. At the time of presentation to the emergency room, she was in pulmonary edema with a cardiac index of less than 2.0 L/m^2. After admission to the intensive care unit, the patient was successfully resuscitated with inotropic agents and vigorous diuresis. Shortly after being transferred to a step-down unit, the patient required readmission to the intensive care unit for hypotension and pulmonary edema. She was placed on a regimen of maximal doses of three inotropic agents yet continued to deteriorate hemodynamically; she became oliguric, and episodes of nonsustained ventricular tachycardia developed. Cardiac index dropped to 1.8 L/m^2 with mean pulmonary artery pressures of more than 40 mm Hg and arterial pressures of 70/40 mm Hg. After informed consent, the patient was taken on an emergency basis to the operating room for device application. During transport, cardiac index (<1.8 L/m^2) and arterial pressures (60/40 mm Hg) fell further, with increases in pulmonary diastolic pressures to more than 45 mm Hg.

Patient 2

In November 1990, a 46-year-old man with no past medical history was seen at the emergency room with an acute, massive myocardial infarction. The patient was given thrombolytic therapy, which did not result in electrocardiographic evidence of reperfusion. Therefore, he was taken to the cardiac catheterization laboratory for emergency percutaneous transluminal coronary angioplasty. Catheterization revealed an ejection fraction of 0.10, severe global hypokinesis, and 100% occlusions of the proximal anterior descending and circumflex arteries with a 75% proximal right coronary occlusion. Multiple attempts to open the anterior descending artery using percutaneous transluminal coronary angioplasty techniques were unsuccessful and resulted in hemodynamic deterioration requiring placement of an intraaortic balloon pump and initiation of inotropic support. The patient's condition stabilized over the next 24 hours, and he was placed on the active cardiac transplant list. The patient's hemodynamic status subsequently deteriorated. Inotropic

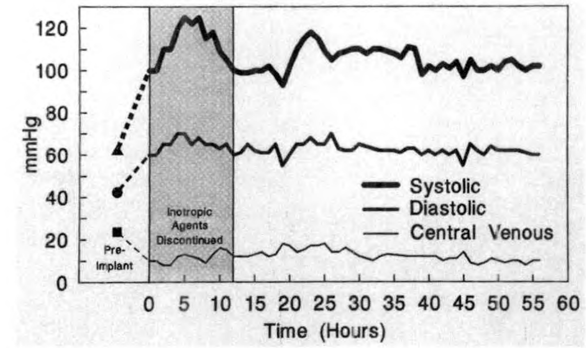

Fig 3. Systemic arterial and venous pressures before and during 56 hours of support in the first direct mechanical ventricular actuation recipient. The patient was weaned from all inotropic agents during the first 12 hours of support.

agents were increased to maximal doses along with continued intraaortic balloon pump support. However, the patient became oliguric with a cardiac index worsening to less than 1.8 L/m^2. Systemic pressures fell to 80/60 mm Hg and pulmonary artery diastolic pressure was greater than 40 mm Hg. After informed consent, the patient was taken on an emergency basis to the operating room where he suffered cardiac arrest upon arrival. Cardiopulmonary resuscitation was begun using standard closed chest cardiac massage while the chest was prepared and draped. However, cardiac index (0.5 L/m^2) and mean arterial pressures (40 mm Hg) were severely depressed for 15 minutes before device application.

Results

Both patients had transesophageal echocardiography performed in the operating room immediately before device application. This allowed for determination of transverse cardiac dimensions, and an appropriate device size was estimated preoperatively. A 115-mm diameter cup was selected for the first patient based on a 115-mm transverse cardiac diameter. The device fit perfectly, and DMVA application through an anterior thoracotomy was completed in less than 3 minutes. In the second patient, a cup of larger size (125 mm) provided adequate support; however, a subsequent change to a slightly smaller size (120 mm) substantially improved hemodynamics.

Patient 1

After DMVA application, hemodynamics immediately improved to a cardiac output of 3.9 L/min (2.78 L/m^2). Systemic arterial pressure increased to 100/60 mm Hg, and pulmonary pressure dropped to 24/8 mm Hg. Drive lines and a single chest tube exited the chest through separate intercostal incisions (Fig 2). The entire operation from skin incision to closure took 38 minutes. During the subsequent 12 hours administration of all inotropic agents was discontinued. The patient remained hemodynamically stable with normal systemic arterial (106 ± 1.0/63 ± 0.4 mm Hg) and venous pressures (12 ± 0.3 mm Hg) (Fig 3). Cardiac outputs averaged 3.16 ± 0.05 L/min (2.26 ± 0.03 L/m^2) during support, with adequate tissue perfusion as evidenced by normal venous saturations (Fig 4) and return of normal urine output. Pulmonary artery pressures (33 ± 0.9/16 ± 0.5 mm Hg) remained normal during the entire support period (Fig 5). All end organs demonstrated normal function during the period of DMVA support. After 56 hours, a donor heart became available and the patient was returned to the operating room where cardiopulmonary bypass was instituted through a virgin sternotomy. The device was easily removed after the patient was placed on cardiopulmonary bypass by cutting the sterile, intrathoracic portions of the drive and suction lines (Fig 6). Cardiac transplantation was then performed with a subsequent uncomplicated recovery. The patient is alive and in functional class I more than 1 year after transplantation without incidence of either infection or rejection.

Patient 2

The heart could only be briefly defibrillated before cup application and remained fibrillating during the entire period of support. Initial cardiac outputs increased to 4.7 L/min (2.14 L/m^2) upon placement of DMVA. These results were dramatic in comparison with the patient's severely depressed outputs (approximately 1 L/min) during his resuscitation for cardiac arrest. Systemic arterial pressures increased to 120/80 mm Hg, whereas pulmo-

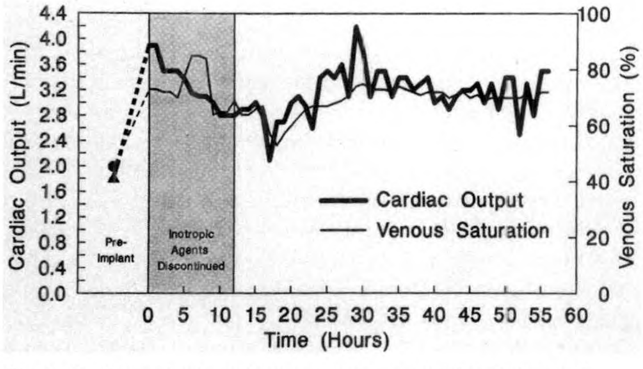

Fig 4. Cardiac outputs and venous oxygen saturations before and during 56 hours of support in the first direct mechanical ventricular actuation recipient.

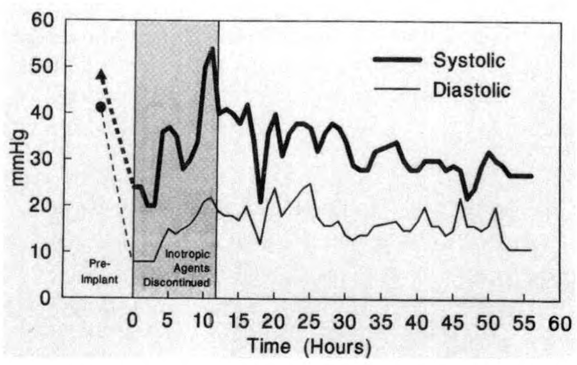

Fig 5. Pulmonary artery pressures before and during 56 hours of support in the first direct mechanical ventricular actuation recipient.

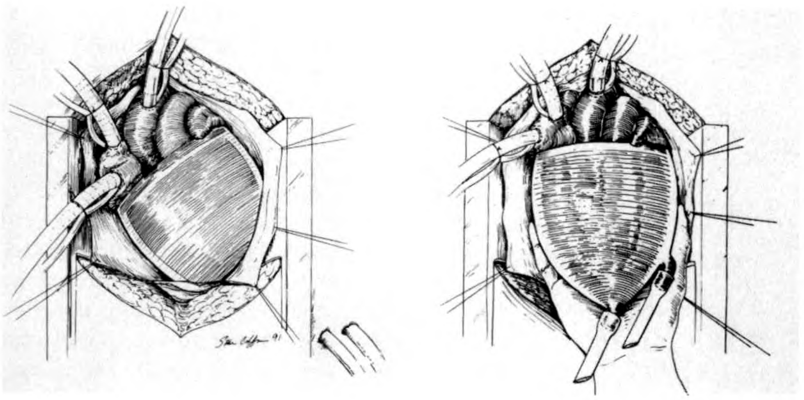

Fig 6. Before cardiac transplantation in patient 1, cardiopulmonary bypass was instituted using standard cannulation techniques through a virgin sternotomy (left). Device removal was performed by cutting the sterile drive lines within the chest and removing the cup through the sternotomy (right).

nary diastolic pressures decreased from 45 mm Hg to 22 mm Hg. As in the first patient, the chest was closed and the patient transferred to the intensive care unit. Hemodynamics remained stable during circulatory support with a mean cardiac output of 6.2 ± 0.3 L/min (2.8 ± 0.1 L/m^2) (Fig 7). A gradual decline in cardiac output occurred after 6 hours of support owing to a leak in a defective diaphragm. Placement of a new device resulted in initial cardiac outputs of 11 L/min. Systemic pressures (110 ± 2/66 ± 1 mm Hg) and central venous pressures (9.9 ± 1.5 mm Hg) remained adequate throughout support, despite the initial cup failure (Fig 8) and refractory ventricular fibrillation. Pulmonary diastolic pressures (21.3 ± 0.9 mm Hg) remained below device implant values (Fig 9). Venous saturations averaged 0.57 ± 0.04 during support, which indicated improved tissue perfusion (see Fig 7). Unfortunately, the patient remained neurologically unresponsive after his cardiac arrest and was pronounced brain-dead. Therefore, the device was removed after 45 hours of circulatory support, and the patient died.

Histologic Examination

Both patients' native hearts underwent extensive histologic examination. Despite the presence of recent underlying myocardial infarctions, neither heart showed any device-related trauma. In the first patient, myocardial infarction, predominantly in the right ventricle, was estimated to have occurred 3 to 4 days before device application. In the second patient, myocardial infarctions throughout both ventricles involved 60% of the myocardial mass. Approximately half of the infarcted myocardium was acute and represented two recent events that had occurred approximately 3 to 4 and 6 to 9 days before device implantation. The remaining infarctions appeared as well-defined scars that originated months to years earlier.

Complications

There were no complications during the entire period of support in the first patient. However, in the second patient, defective material led to premature diaphragm fatigue and a small leak in the cup diaphragm where it was attached to the housing. Cardiac outputs gradually declined at a rate of approximately 0.5 L · min^{-1} · h^{-1} after onset of the leak. This slow clinical failure of the cup confirmed failures observed during prolonged experimental in vivo and in vitro studies. Because sudden, complete cup failure has not been encountered, there is adequate time for cup replacement while hemodynamic support is

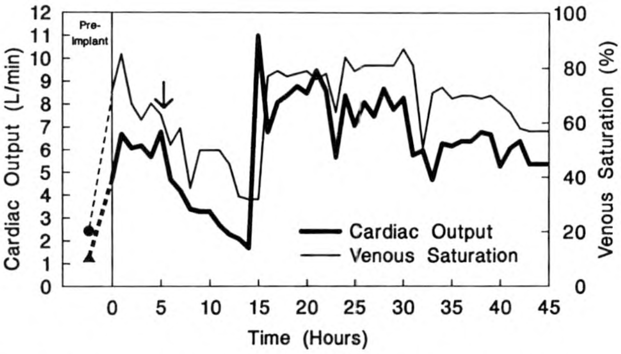

Fig 7. Cardiac outputs and venous oxygen saturations before and during 45 hours of support in the second direct mechanical ventricular actuation recipient. Use of the intraaortic balloon and all inotropic agents was discontinued immediately after direct mechanical ventricular actuation application. Arrow *indicates the onset of a leak in the cup diaphragm at 6 hours of support. Replacement of a new cup at 14 hours resulted in return of normal hemodynamics.*

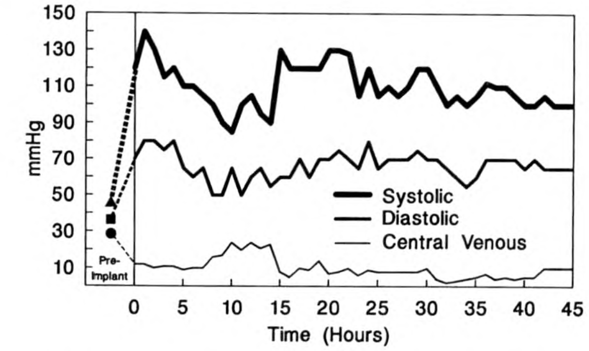

Fig 8. Systemic arterial and venous pressures before and during 45 hours of support in the second direct mechanical ventricular actuation recipient. Use of the intraaortic balloon and all inotropic agents was discontinued immediately after direct mechanical ventricular actuation application. Pressures remained relatively stable despite a device leak during the early support period.

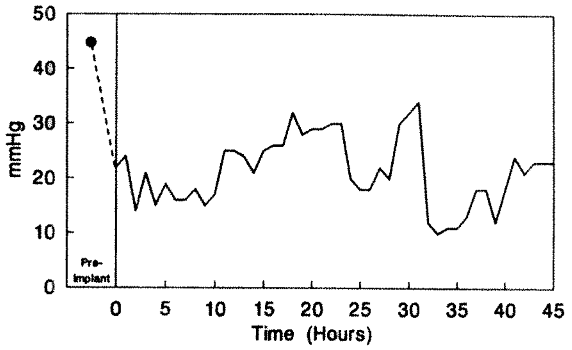

Fig 9. Pulmonary diastolic pressures before and during 45 hours of support in the second direct mechanical ventricular actuation recipient. Pulmonary systolic pressures could not be interpreted because of recording artifacts.

still adequately maintained. Furthermore, as demonstrated in the second patient, the cup can be replaced at the bedside using a standard thoracotomy tray. The initial hard-shell device was replaced in the second patient using a more pliable soft-shell device. The soft-shell device was very easy to apply through the small anterior thoracotomy.

Comment

These initial clinical results show that DMVA can be successfully used in patients with biventricular failure and refractory cardiogenic shock as a means of total circulatory support while awaiting cardiac transplantation. Based on numerous experimental studies and the present clinical results, we believe that this technology deserves further human application. Direct mechanical ventricular actuation has many proven and theoretical advantages compared with current blood contacting assist devices.

Because DMVA requires no anticoagulation or vascular cannulation, hemorrhagic and thromboembolic complications should theoretically be markedly reduced. In addition, implantation of DMVA does not require any vascular anastomoses. Reoperation for hemorrhage using current blood-contacting devices continues to be a major complication occurring in nearly 60% of patients [9–15]. Although newer devices employ "antithrombogenic" surfaces, thromboembolism remains an often catastrophic complication. Often these thromboemboli arise at sites of anastomosis or from mechanical valves or sewing rings and not from the moving membrane used to create blood flow [16–18].

In addition, DMVA application requires only a small left anterior thoracotomy, and complete biventricular support can be established in only 3 to 5 minutes. The installation of current blood-contacting univentricular and biventricular assist devices requires a major and time-consuming operative intervention complicated at times by technical problems [9–11, 17, 19]. These devices, unlike DMVA, also require an elaborate set-up and support team.

Finally, as shown in the present study, DMVA can restore physiologic, pulsatile flow rates even in the fibrillating heart, which is not possible with standard blood-contacting ventricular assist devices. This technology does not require an elaborate set-up or support team and does not necessitate prolonged training courses. Once DMVA is applied, a number of options become available: (1) support can be provided to determine whether end-organ injury is reversible or irreversible, (2) the device can serve as a temporary circulatory support system to bridge to implantation of a total artificial heart, or (3) the device can provide a definitive bridge to cardiac transplantation.

Through these options, DMVA can provide initial support and allow thorough patient assessment before committing to more elaborate and expensive devices. Such an approach may reduce early mortality and morbidity and minimize heroic and costly resuscitation efforts currently applied to patients who subsequently succumb early-on. For example, of those deaths occurring before transplantation while assist devices are being used, approximately 50% occur during the first hours to days of support [10, 14, 18]. Direct mechanical ventricular actuation may decrease early mortality by allowing rapid hemodynamic stabilization and avoidance of the complications associated with blood-contacting assist devices. In addition, if continued support is not justified secondary to end-organ failure, the unfortunate loss of the patient is not associated with tremendous expense.

Direct mechanical ventricular actuation requires further evaluation to determine its potential for providing long-term circulatory support. On the other hand, its ability to serve as a valuable adjunct in this regard has been shown in the present study. There are a number of potential future applications for DMVA:

Bridging to cardiac transplantation
Bridging to any other cardiac assist devices
Resuscitation from refractory cardiac arrest
Circulatory support for cardiogenic shock
Postcardiotomy circulatory support
Resuscitation from profound hypothermia
Noncardiac donor organ preservation after neurologic death

In certain situations, the device may actually prove critical in determining a patient's suitability for transplantation. For instance, some selection criteria for potential transplant candidates are difficult to adequately assess during acute cardiovascular collapse. Pulmonary vascular resistance is a particularly relevant example. Pulmonary vascular resistance is sometimes even more difficult to assess during conventional methods of circulatory support. This can be a major problem because substantial elevations in pulmonary vascular resistance have been found to be associated with right ventricular failure and subsequent mortality after transplantation [20]. Because DMVA creates hemodynamic conditions similar to the normal physiologic state, pulmonary vascular resistance can be calculated using standard techniques.

A major drawback of DMVA is its current state of cup manufacture. Presently, all devices are hand-fabricated using Silastic (Dow Corning) diaphragms. Industrial fabrication with careful quality control must be accomplished before widespread application of this technology. Another potential drawback may be trauma to previously placed coronary artery bypass grafts. However, we have used DMVA after coronary artery bypass grafting in 1 patient [3] as well as in experimental animal studies. In the acute setting, there was no evidence of graft trauma or interference with graft flow after 4 hours of device application. However, further experimental work will be required to determine DMVA's effect on bypass grafts during prolonged periods of circulatory support. Finally, it is unknown whether this particular technology can be routinely applied in patients who have had previous cardiac operations. In those patients who have had previous cardiac operations, the heart would have to be totally dissected free before device application. This would require a median sternotomy instead of the preferred left anterior thoracotomy approach.

In summary, we believe that our initial clinical results are most encouraging and justify further clinical application of this unique form of biventricular assistance. Although the technology was originally conceived to be applicable only to those patients who suffer acute refractory cardiac arrest, we believe that the technology can be perfected to provide a simple means for long-term circulatory support.

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DISCUSSION

DR DAVID B. SKINNER (New York, NY): I was delighted to hear this report. Back in the late 1960s, beginning in 1966 on until about 1972, George Anstadt and I and others had a chance to explore this device extensively. We were able to support dogs up to 72 hours with a fibrillating heart followed by defibrillation and normal organ function throughout that time, including regional blood flow studies. We were able to study coronary circulation while the cup was in place and found that it was not diminished by the device itself, and based on that our initial hope was that it might be useful in cardiac resuscitation. There were initial clinical trials with this at the Wilford-Hall US Air Force Hospital and at Johns Hopkins Hospital in the late 1960s and early 1970s. We had patients maintained as described here with good hemodynamics on the device but were unsuccessful in weaning anybody off the cup. We did not have the heart transplant option available at that time. So this heart transplant option is the necessary finish for this story.

Other possible applications of the device that were explored included the use of the device in a patient who was brain-dead with heart failure to sustain the brain-dead cadaver, if you will, while other organs were being harvested for transplantation as the perfusion of the other organs with this device is quite good. Dr Lowe, did you consider that possibility in this second patient who, at his young age, might have been a good donor for another organ?

We stopped the work in 1972 because the National Institutes of

Health, who had been funding the work up until that time, decided not to fund this avenue of research anymore. We were at a point where there were problems with using hand-made devices that were not standardized and could not be of certain reliability. It appears that this still remains a problem. Dr Lowe, what plans are there to try to take this device beyond a hand-made, experimental mode into something that can be standardized and reproducibly available for the rest of us?

This is an excellent report. I am delighted to see the successful outcome after 20 years of hoping this would happen. Congratulations.

DR LOWE: Thank you very much, Dr Skinner, for your comments. Drs Skinner and Anstadt did indeed prove that this technology could be used to preserve a brain-dead patient so that a kidney could be harvested for successful transplantation. I believe bridging to cardiac transplantation has now been established as another application.

Furthermore, in the immediate future, we hope to apply DMVA for resuscitation from refractory cardiac arrest. As Dr Skinner stated, however, before we can take this technology further, even within our own hospital, we must obtain appropriate industrial support. At the present time, all cups are hand-made. We believe that industrial techniques including injection molding and application of new biomers will eliminate inner membrane rupture or leak.

I personally believe that DMVA will become a means for long-term circulatory support. We plan to apply it for biventricular support in patients with cardiogenic shock after cardiac surgical operations. At the present time, we do not know whether or not we can apply DMVA technology in patients with recent coronary artery bypass grafts. We have done bypass grafts in animals and put the cup on for a number of hours and did not tear anastomoses or adversely affect blood flow. However, in a patient with a short or tight internal mammary artery, I would not recommend applying DMVA.

DR STEVEN F. BOLLING (Ann Arbor, MI): I see Dr Pennington in the audience, who has tried virtually all of these devices at one time or another. Dr Pennington, have you had any experience with this type of device, and what do you think about its potential for long-term support?

DR D. GLENN PENNINGTON (St. Louis, MO): Well, I have no experience whatsoever with this system. I suppose my own bias at this point would be to consider this as a bridge to a bridge. We have learned from our bridging experience that one of the major benefits is its great rehabilitative potential. This sort of short-term support you are reporting would not allow for that. The salvage rates now among bridged patients are almost as good as those among the nonbridged patients. And so what you would have to do is show that this device could last for weeks or months until one could have an elective transplantation under optimal circumstances. I suspect you are quite a way from that. But it is an exciting device, and I am looking forward to hearing more about it.

DR VALAVANUR A. SUBRAMANIAN (New York, NY): Around the same time as Drs Skinner and Anstadt were developing the ventricular cup and trying in the laboratory for cardiogenic shock support, we at Dr C. Walton Lillehei's lab at The New York Hospital-Cornell Medical Center, developed a left ventricular direct mechanical assistance cup made from a blood pressure cuff housed inside a hard polyurethane sleeve. Our results showed that indeed this device worked, but its use was limited by severe epicardial hemorrhages. Our device did not have any active diastolic relaxation mechanism in the cup, which I think is the most important feature of your device. Did you have any endocardial hemorrhage in the last patient shown? Was there anything on autopsy to show if there were any endocardial or epicardial hemorrhages?

This is a device that, I think, should be pursued clinically in many patients who may need it as an initial stage support.

I congratulate Dr Lowe and associates for this interesting new device or application.

DR LOWE: I sincerely thank Dr Pennington and Dr Subramanian for their comments. I agree with Dr Pennington that this is an exciting and new way to bridge to bridge, so to speak. I think our next challenge is to perform a series of long-term experiments. We hope to have cows or sheep fibrillating for long periods of time. Obviously, these are very expensive experiments and we are going to require substantial support. I believe that with currently available biotechnology we will be able to manufacture durable cups and we will be able to support patients for extended periods of time. Eventually, this device may have applicability as a long-term biventricular support device.

An interesting clinical study with a group like Dr Pennington's would be to use this device initially, in other words, instead of committing to very expensive biventricular assist devices. Current biventricular assist devices require a support team and careful anticoagulation of the patient. Direct mechanical ventricular actuation could be applied initially and clinical characteristics could be sorted out. We all know that 50% of deaths in these patients commonly occur within hours to 4 or 5 days after application. Therefore, we would not commit to very expensive technology early on, and we could use this device to select patients for more complex devices including total artificial hearts at some point in the future.

We agree with Dr Subramanian. A key concept that we have realized in the last several years is the importance of appropriate timing, and I do not mean electrophysiologic timing. One of the advantages is that you do not have to time this device because the heart will actually track with the mechanical actuation of the device. We can easily keep track of compressive forces to make sure that we do not collapse the right ventricle or left ventricle prematurely into the septum. The excised hearts from both of these patients were studied extensively in the cardiovascular pathology laboratory of Dr Keith Reimer. He found no epicardial or endocardial or midmyocardial trauma. We have performed more than 200 animal experiments in the laboratory, and the only consistent site of injury that we have seen is a small area of superficial epicardial hemorrhage or contusion over the right ventricular outflow tract. I think the reason is that the heart is allowed to eject with the forces of compression, so we do not have a vector of friction along the epicardial surface of the heart.

DR GERARD GUIRAUDON (London, Ont, Canada): Congratulations for a very stimulating presentation. The mechanical assist device seems to have controlled a serious mitral valve regurgitation. I presume, based on the data presented, that the device decreased the left ventricular diastolic volume and diastolic diameter, especially at the level of the papillary of muscles. Do you have any better speculation?

DR LOWE: That is an excellent observation, Dr Guiraudon, and we cannot explain it. I thought in our first patient, who had serious mitral regurgitation, that the device would not be effective and that we would be giving a lot of nitroprusside for left-sided afterload to allow successful left-sided ejection. However, this was not at all a problem during her entire 56-hour period of support.

I believe that there are two possible explanations. One is that we may be altering mitral valve geometry because the device is close to the base of the heart. In addition, we are driving the right and left sides of the heart equally, and so we are getting blood through the lungs whether it wants to go through the lungs or not, and we can get it out of the left ventricle whether it wants to or not. We have not yet applied DMVA to a patient with aortic insufficiency, and we do not know how the device will function with severe aortic insufficiency.

DR CARY W. AKINS (Boston, MA): I really enjoyed your presentation. I have three quick questions. You make the claim that this is biventricular support. All of your data in the 2 patients would seem to show that it is more left ventricular support than right ventricular support. What have you shown in the laboratory? In 1 patient part of the right ventricle appeared to be outside of the cup, and I am wondering if you have evidence that the right ventricle can be also supported. The second thing is that the size of the heart changes, presumably. Does this device ride higher up the heart, and will it cause damage to other areas? The third question is, can you design this device such that the inflation of the inner diaphragm begins at the apex and goes up toward the outflow tract, or does it all just squeeze as one unit?

DR LOWE: Those are all very good questions. In the laboratory, we have induced global ischemic insults to both ventricles. We have extensive experimental evidence showing that this technology does indeed work as a biventricular assist device. I think we have now proven this in patients. As you recall, the second patient was fibrillating during the entire period of device support. Therefore, to achieve normal left- and right-sided pressures, clearly, we were successfully pumping both ventricles.

I would answer "yes" to your last question. I believe that we could design a sequence inflator, but we do not believe it is necessary. The free walls of the left and right ventricles collapse into the septum in a physiologic fashion during device systole. The pulse pressure curves are certainly physiologic, which adds further support to the concept that DMVA results in physiologic ejection. It is very easy to achieve cardiac outputs from 80% to 130% of control, even in fibrillating hearts.

Mitral Valve Repair for Ischemic Mitral Insufficiency

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Over a 5-year period, 1,292 patients had operation on their native mitral valves. Ischemia was the cause of mitral insufficiency in 84 patients (6.5%). Sixty-five patients (77.4%) had mitral valve repair. Mean age was 66 ± 10 years; 35 patients (53.8%) were women. Mean degree of preoperative insufficiency was 3.2 ± 0.7; mean preoperative New York Heart Association functional class was 3.3 ± 0.7. Eleven patients (16.9%) had acute and 54 (83.1%) had chronic mitral insufficiency. Valve prolapse was present in 26 patients (40%). Restrictive leaflet motion secondary to regional or global left ventricular dilatation occurred in 39 patients (60%). All patients had associated myocardial revascularization followed by transatrial valvuloplasty. Multiple techniques were employed to achieve valve competence: leaflet resection (3), chordal shortening (15), papillary muscle reimplantation (10), papillary muscle shortening (3), and annuloplasty (63). There were six (9.2%) hospital deaths (acute, 9.1%; chronic, 9.3% [not significant]; prolapse, 11.5%; restrictive, 7.7% [not significant]). The mean degree of postoperative mitral insufficiency was 0.6 ± 0.8 in 51 patients. At a mean follow-up of 3.1 ± 1.6 years, patient survival was 96% for patients with valve prolapse and 48% for those with restrictive leaflet motion ($p = 0.02$). New York Heart Association functional class was improved in all groups. Ischemic mitral insufficiency is an uncommon cause of mitral valve disease that is amenable to repair in the majority of cases of both acute and chronic onset. The operative mortality is low, and operation is associated with superior survival in patients with valve prolapse.

(Ann Thorac Surg 1991;52:1246–52)

Ischemic mitral regurgitation remains one of the most challenging management problems in cardiac surgery and is associated with suboptimal results. The majority of patients with ischemic regurgitation are treated medically, but patients with severe regurgitation were found to benefit from surgical treatment [1–3]. However, the operative morbidity and mortality associated with operation for ischemic mitral regurgitation are higher than those for other forms of mitral valve insufficiency.

In the last decade, the operative approach to moderate or severe ischemic regurgitation has evolved from myocardial revascularization to surgical revascularization combined with mitral valvuloplasty. Recognized advantages of mitral valve repair compared with prosthetic valve replacement are lower operative mortality, better preservation of ventricular function, reduced valve-related complications, decreased requirement for anticoagulation therapy, improved long-term survival, and decreased cost [4]. To evaluate the efficacy of mitral valve repair for ischemic regurgitation, our experience over a 4½-year period was reviewed.

Patients and Methods

All patients undergoing operation for moderate to severe ischemic mitral insufficiency were included. Ischemic mitral regurgitation was defined as valve insufficiency secondary to myocardial infarction with angiographic or echocardiographic documentation of left ventricular wall motion abnormality in a region perfused by a critically obstructed coronary artery. Patients were excluded if there was any macroscopic or histologic evidence of primary mitral valve pathology. Ischemic regurgitation was considered acute if the interval between myocardial infarction and operation was less than 4 weeks and chronic if the interval was greater.

Between January 1985 and June 1989, 1,292 patients underwent valve procedures on their native mitral valves at The Cleveland Clinic Foundation. Eighty-four patients (6.5%) had ischemic mitral insufficiency (Fig 1). Nineteen patients (22.6%) underwent revascularization and valve replacement; 65 (77.4%) underwent revascularization combined with mitral valve repair and form the basis of this report. Eleven patients (16.9%) had acute ischemic insufficiency and 54 (83.1%) had chronic.

There were 30 men and 35 women. Patients ranged in age from 47 to 78 years (mean age, 66.0 ± 8.1 years). The mean interval from myocardial infarction to operation was 77.1 ± 134.6 weeks for the entire group and 2.6 ± 0.8

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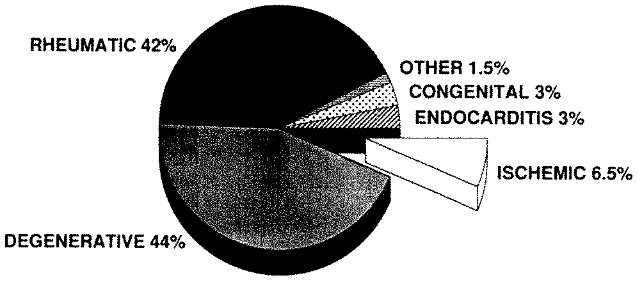

Fig 1. From 1985 through 1989, 1,292 patients underwent surgical procedures on their native mitral valve. Ischemic insufficiency was the cause of the mitral disease in 6.5%.

weeks and 98.0 ± 145.8 weeks in the acute and chronic groups, respectively.

Degree of mitral insufficiency was calculated by angiography or echocardiography and ranged from 2+ (moderate) to 4+ (severe) with a mean of 3.23 ± 0.66. Left ventricular function was assessed by echocardiography or ventriculography, or both, and categorized as normal, mild, moderate, or severe impairment. Twenty-three patients (35.4%) had mild impairment, 20 (30.8%) had moderate, and 22 (33.8%) had severe. Left ventricular function was more impaired in patients with chronic ischemic insufficiency than in those with acute insufficiency (Fig 2).

The mean preoperative New York Heart Association functional classification (NYHA FC) was 3.26 ± 0.69. In patients with acute ischemic mitral insufficiency, 5 (45.0%) were in cardiogenic shock preoperatively. Twelve patients (18.5%) had undergone a previous cardiac operation.

Preoperative echocardiography was performed in all patients. Valve dysfunction was classified according to the range of motion of the valve leaflets as described by Carpentier [5]. Valve prolapse was found in 26 (40.0%) of the patients. Valve prolapse resulted from papillary muscle rupture in 8 and papillary muscle infarction with elongation in 18. Restricted leaflet motion occurred in 39 patients (60.0%) and was secondary to regional or global ventricular dilatation. Of the patients with acute ischemic mitral regurgitation, 5 (45.0%) had prolapse and 6 (55.0%) had restricted motion. Of the patients with chronic regurgitation, 21 (39.0%) had prolapse and 33 (61.0%) restricted leaflet motion. Left ventricular function was significantly worse in patients with restricted leaflet motion (Fig 3).

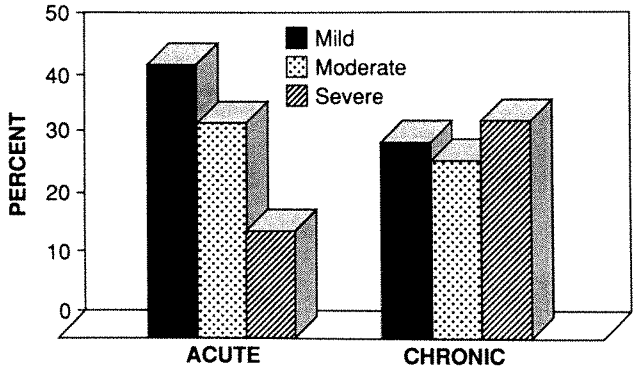

Fig 2. Compared with patients with acute mitral insufficiency, those with chronic mitral insufficiency had worse ventricular function.

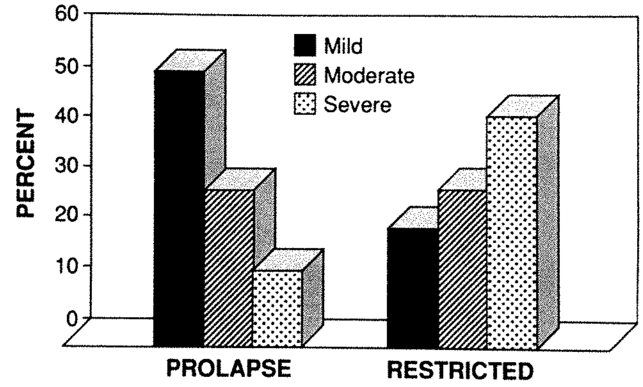

Fig 3. Restricted leaflet motion was secondary to regional or global ventricular dilatation. Left ventricular function was more impaired in patients with this valve classification compared with those with valve prolapse.

Color flow Doppler echocardiography was used intraoperatively before repair to evaluate the degree of and cause of insufficiency in all patients. All operations were performed using cardiopulmonary bypass with bicaval cannulation, systemic hypothermia, and cold potassium crystalloid cardioplegia during a single period of aortic cross-clamping. All patients had myocardial revascularization. The mean number of grafts was 2.4 ± 1.0 per patient. After completion of distal anastomoses, valve repair was performed transatrially using techniques described by Carpentier [5]. Multiple techniques were employed to achieve valve competence: leaflet resection (3), chordal shortening (15), papillary muscle reimplantation (8), papillary muscle shortening (3), and annuloplasty (63). All but 2 patients had annuloplasty. Mitral valvuloplasty was combined with simultaneous ventricular aneurysm resection in 3 patients. Three patients in the acute group and 5 in the chronic group had insufficiency resulting from papillary muscle rupture and were repaired by papillary muscle reimplantation. Patients demonstrating restricted leaflet motion had valve repair with annuloplasty alone. Early in the series, annuloplasty was performed with a Carpentier-Edwards ring. Recently, annular remodeling has been performed with posterior annular plication with a strip of glutaraldehyde-treated bovine pericardium.

Distention of the left ventricle with iced saline solution was used for initial testing of valve repair competence. Intraoperative Doppler echocardiography was used to assess definitive valve competence in all patients immediately after repair.

Follow-up data were acquired on all surviving patients. Univariate analyses of hospital and late survival were performed. The clinical variables in the analysis were as follows:

Preoperative Clinical Variables
- Age
- Sex
- Preoperative NYHA FC
- Duration of symptoms
- Duration of ischemic mitral regurgitation
- Diabetes
- Hypertension

Immediate Preoperative Condition
- Stable
- Shock
- Intraaortic balloon pump
- Blood urea nitrogen (mg/100 mL)
- Creatinine (mg/100 mL)
- Rhythm

Preoperative Catheterization and Echocardiographic Variables
- Number of stenotic vessels (greater than 50%)
- Angiographic/echocardiographic left ventricular function
- Leaflet motion
- Angiographic/echocardiographic quantification of mitral regurgitation
- Angiographic/echocardiographic wall motion abnormality

Operation-Related Variables
- Mitral anatomy
- Leaflet motion classification
- Number of coronary artery bypass grafts
- Type of repair performed (chordal procedure, papillary muscle procedure, annuloplasty)
- Associated operations
- Reoperation
- Completeness of revascularization

Immediate Postoperative Variables
- Transfusion requirement
- Perioperative myocardial infarction
- Cerebral vascular accident
- Wound complication
- Blood urea nitrogen (mg/100 mL)
- Dialysis
- Respiratory failure
- Prolonged inotropic support (greater than 48 hours)
- Reexploration for bleeding

Late Postoperative Variables
- Postoperative functional class
- Postoperative regurgitation
- Postoperative left ventricular function

For hospital survival, p values were calculated by means of a series of χ^2 tests of association (p values ≤ 0.05 were considered statistically significant). To identify risk factors for late mortality, the variables were tested for Wilcoxon rank sum tests (values ≤ 0.05 significant). Multivariate analyses were not used because of an insufficient number of overall deaths in the study group.

Results

There were six in-hospital deaths (9.2%) (acute, 9.1%; chronic 9.3% [not significant]; prolapse, 11.5%; restrictive, 7.7% [not significant]). Hospital death occurred in 1 patient with acute disease secondary to postoperative sepsis. Five hospital deaths occurred in patients with chronic ischemic insufficiency, 2 from postoperative sepsis and 3 from cardiac failure. Preoperative variables associated with increased risk for hospital death were poor preoperative NYHA FC ($p = 0.05$) and need for preoperative intraaortic balloon pump ($p \leq 0.01$). Acute presentation, valve motion classification, and technique of repair were not predictive risk factors. It was not possible to identify significant risk factors using multivariate analyses.

Table 1. Perioperative Morbidity ($n = 59$)

Cause	No. of Patients
Cerebral vascular accident	3 (5.1%)
Myocardial infarction	5 (8.5%)
Reexploration for bleeding	3 (5.1%)
Failed valve repair	2 (3.4%)

Perioperative morbidity in 59 surviving patients is listed in Table 1. Prolonged inotropic support was needed in 25 patients (38.5%). Two patients with chronic insufficiency underwent reoperation during initial hospitalization for failed repair. In 1 patient there was partial dehiscence of a Carpentier-Edwards ring and in a second there was suture dehiscence of a repaired leaflet.

Follow-up was complete at a mean of 36.6 ± 19.0 months. There were 18 late deaths in 59 hospital survivors resulting in 63% survival at 3 years. Patient survival was 96% for valve prolapse and 48% for restrictive leaflet motion ($p = 0.02$). Table 2 lists the causes of late deaths. The single late death in a patient with valve prolapse was noncardiac in origin. Univariate analysis identified the following as predictors of late mortality: incomplete revascularization ($p = 0.01$), perioperative stroke ($p = 0.002$), perioperative myocardial infarction ($p = 0.01$), perioperative renal failure ($p < 0.01$), prolonged postoperative respiratory insufficiency ($p = 0.006$), preoperative intraaortic balloon pump ($p < 0.001$), and restrictive leaflet motion ($p = 0.02$). It was not possible to identify signifi-

Table 2. Causes of Late Deaths

Cause	No. of Patients
Patients with leaflet prolapse ($n = 1$)	
Gastrointestinal bleeding	1 (100%)
Patients with restrictive leaflet motion ($n = 18$)	
Congestive heart failure	6 (35.3%)
Sudden death	4 (23.5%)
Multiorgan failure	2 (11.8%)
Arrythmia	1 (5.9%)
Renal failure	2 (11.8%)
Sepsis	1 (5.9%)
Cancer	1 (5.9%)

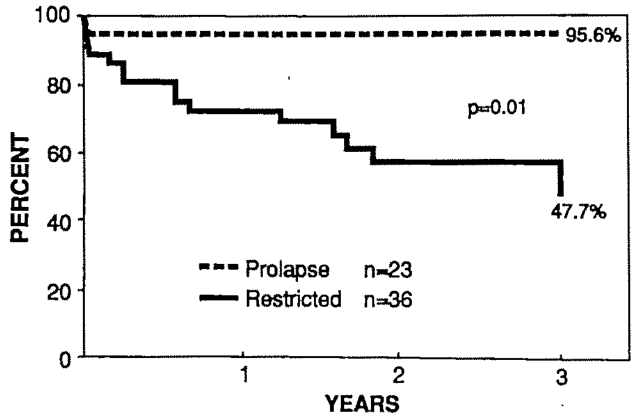

Fig 4. At 3 years' follow-up survival was 63% for all patients undergoing mitral valvuloplasty for ischemic dysfunction. Survival is superior in patients with valve prolapse compared with those with restricted leaflet motion.

cant risk factors using multivariate analysis. Restricted leaflet motion was associated with high risk for late death in patients with both acute and chronic mitral regurgitation (Fig 4).

Of the 41 long-term survivors, 29 (70.7%) are in NYHA FC I, 8 (19.5%) in NYHA FC II, and 4 (9.8%) in NYHA FC III. The mean degree of postoperative mitral insufficiency was 0.6 ± 0.8 in survivors. Two patients required reoperation for failed repair. Both reoperations occurred in patients with chronic insufficiency and valve prolapse, one as a result of a dehisced Carpentier-Edwards ring and the other as a result of newly ruptured chordae. All patients undergoing reoperation were treated with valve replacement. There were no thromboembolic or other valve-related events..

Comment

As the advantages of mitral valve repair are recognized, valvuloplasty for all forms of mitral valve disease is being increasingly applied [4]. Previous surgical management of ischemic mitral regurgitation has been suboptimal. Moderate and severe mitral insufficiency precipitated by myocardial infarction is infrequent, representing the cause of valve pathology in less than 10% of the mitral valve operations [6]. After rheumatic and degenerative pathology, ischemia is the third most common cause of valve dysfunction in patients undergoing operation on their native mitral valves at The Cleveland Clinic Foundation [4]. The patients in this series undergoing mitral valve repair represent 80% of all patients with ischemic regurgitation treated surgically during the period 1985 to 1989. Nineteen patients received prosthetic valve replacement, primarily reflecting surgeon inexperience with repair techniques or preference. Occasionally, valve repair is not appropriate because of associated valve pathology or other major leaflet abnormalities that are more suitably treated with prosthetic valve replacement. Currently, the majority of patients with ischemic regurgitation are amenable to reconstructive techniques.

Table 3. Mitral Valve Replacement for Ischemic Mitral Insufficiency

Series	No. of Patients	Hospital Mortality	Late Survival
Gerbode et al [7]	41	48%	NA
Pinson et al [8]	28	38%	44% at 5 years
Lytle et al [9]	47	10%	NA
Connolly et al [10]	16	19%	85% at 5 years
Kay et al [11]	40	35%	35% at 5 years
Rankin et al [6]	42	45%	NA
Total	214	33%	

NA = not available.

Tables 3 and 4 summarize major series of patients with ischemic mitral regurgitation treated by revascularization with mitral valve replacement and repair, respectively. The hospital mortality of 9.2% compares favorably with previously published data [7–12]. A lower operative mortality with mitral valve repair for ischemic disease has been previously documented [11, 12]. A statistical comparison of results between repair and replacement was not possible in this study because of the small number of patients having replacement. When compared with previously published data, the operative mortality for repair is lower.

Early survival benefit of valvuloplasty for ischemic insufficiency has previously been most marked in patients with recent myocardial infarction [11, 12]. Early results with valve repair for acute ischemic insufficiency showed a mortality of 28% [12]. In this series, there was only one death among patients undergoing valvuloplasty for acute ischemic insufficiency, and acute disease did not emerge as a risk factor for hospital mortality. This may in part reflect improved preoperative and intraoperative management of the ischemic ventricle. However, when compared with patients with chronic ischemic insufficiency, global left ventricular function was better in patients with recent infarction. In this series, only 16.9% of patients requiring operation had acute ischemia. With improvement in the early management of ischemic regurgitation by reperfusion therapy or balloon angioplasty, fewer patients with acute insufficiency require operation [1].

Moderate to severe ischemic regurgitation has been

Table 4. Mitral Valve Repair for Ischemic Mitral Insufficiency

Series	No. of Patients	Hospital Mortality	Late Survival
Kay et al [11]	101	15%	64% at 5 years
Rankin et al [2]	23	26%	NA
Rankin et al [6]	40	18%	NA
Current series	65	9%	63% at 3 years
Total	229	17%	

NA = not available.

demonstrated to have an adverse influence on long-term survival [1]. However, repair of ischemic mitral dysfunction results in better late survival compared with valve replacement [11]. In this study, there were 18 late postoperative deaths. Even though 63% 3-year survival of patients with repair of ischemic dysfunction is poorer than the 5-year survival of 87% for all patients with mitral valvuloplasty at our institution [4], it is similar to long-term rates reported for valvuloplasty for this entity [11]. Kay and associates [11] have compiled the largest series of cases of mitral valve repair for ischemic mitral regurgitation and have demonstrated that left ventricular function is the most important determinant of both early and long-term outcome. In this study, all cardiac-related late deaths occurred in patients with restricted leaflet motion in whom ventricular function was most impaired. No late deaths occurred in patients with mild impairment. Compared with prosthetic valve replacement, better preservation of ventricular function [4] is a major advantage of valvuloplasty and makes the argument for mitral valve repair even stronger.

The mechanism of insufficiency with ischemic disease is a source of controversy and confusion. The majority of patients with ischemic regurgitation have papillary muscle dysfunction, more recently termed papillary-annular dysfunction, where regurgitation results from a combination of posterior annular dilatation, papillary muscle elongation, and loss of papillary muscle shortening [6, 11, 13]. The posterior annular dilatation is thought to be the dominant mechanism of incompetence. Reports also indicate histologic changes indistinguishable from primary myxomatous degeneration may develop in chordae and valve leaflets secondary to ischemia [14]. Patients with ruptured chordae have not been included in this study, as ruptured chordae are more likely to be secondary to primary degenerative changes, which may precede the development of ischemic disease. The high incidence of degenerative mitral valve disease and coronary artery disease makes the coexistence of the two processes likely. In this study, only mitral insufficiency resulting from leaflet prolapse or restricted leaflet motion was found. Posterior annular dilatation, in the absence of degenerative disease of the mitral valve, did not occur. Valve prolapse results from papillary muscle infarction with elongation, paresis of the papillary muscle, or papillary muscle rupture. Restriction of leaflet motion occurs with regional or global ventricular dilatation. Recognizing the functional basis for regurgitation in these valves not only dictates the surgical approach, but also provides for a useful predictor of long-term survival.

Previous anatomic studies support this classification. Pathologic examination of ischemic ventricles by Bulkley and colleagues [15] failed to demonstrate annular dilatation, with no correlation between annulus size and the presence or absence of regurgitation. Annular dilatation of any importance has been found to occur only if there is concurrent myxomatous disease. Further, mitral annular dilatation to the degree capable of causing mitral insufficiency occurs infrequently because the surface area of the mitral valve leaflets is 2½ times the area of the mitral orifice. The mitral ring has a sphincterlike action, making the mitral orifice smaller during systole than during diastole. With ischemia, the majority of dilatation occurs in the mid-portion of the ventricle at the level of origin of the papillary muscles, not the annulus [15]. This change in ventricular geometry causes mechanical displacement resulting in restriction of the leaflet excursion.

Impairment of ventricular contraction contributes substantially to the mechanism of regurgitation when papillary muscles are found to be fibrotic. Experimental studies suggest that mitral regurgitation does not occur as a consequence of fibrosis involving only the papillary muscles. If one or both papillary muscles are made fibrotic in a dog by injection of formalin or by ligating the base of the muscle, no regurgitation results. However, if the free wall beneath the papillary muscles is made fibrotic at the same time so that ventricular contraction is impaired, mitral regurgitation results secondary to restriction of motion of a portion of the leaflets [16].

Previous reports concerning ischemic mitral valve repair have demonstrated the efficacy of Kay annuloplasty for this entity [6, 11, 12]. We have found valve prolapse to be more common and believe that if valve prolapse is present, annuloplasty alone results in unsuccessful repair. A number of techniques can be used for valve repair for ischemic dysfunction. With valve prolapse, standard Carpentier techniques are successful. For restricted leaflet motion, annuloplasty reduces the size of the annulus, resulting in increased leaflet coaptation and eliminating regurgitation. These concepts can be applied when either acute or chronic disease is present. Patients with papillary muscle rupture can be treated with papillary muscle reimplantation by several methods. Some were repaired by suturing the ruptured muscle to adjacent papillary muscle, and others by suturing the papillary muscle remnant to the free wall of the ventricle.

Annuloplasty, whether performed with a prosthetic ring or suture, is an integral part of reconstruction of the valves. Early in this experience, annuloplasty with a Carpentier ring was performed. More recently, a posterior annular plication using a strip of glutaraldehyde-treated bovine pericardium has been used. Experimental fixation of the mitral annulus with a ridged annuloplasty ring has impaired left ventricular systolic function [17]. Further, a rigid ring may interfere with the saddle shape of the mitral annulus, which helps distribute the stress on the mitral valve during systole. Use of a strip of bovine pericardium resulting in multiple small posterior plications may preserve the natural shape of the annulus, and preliminary evidence supports better preservation of ventricular function by this modality.

Two in-hospital patients had failed repairs early in the experience before the routine use of transesophageal echocardiography to monitor results. Two additional failures occurred late postoperatively, reflecting the learning curve with this entity.

Transventricular repair of ischemic dysfunctions has been advocated [6]; however, we have not adapted this approach. Using cardiac mobilization and a self-retaining retractor, exposure of the mitral valve transatrially is excellent [18]. The ability to judge the extent of leaflet prolapse is facilitated by a transatrial approach, as is the ability to perform annuloplasty, which is an important component of valve repair for ischemic dysfunction.

Ischemic mitral insufficiency is an uncommon cause of mitral valve disease which is amenable to repair in the majority of both acute and chronic cases. Valvuloplasty can be performed with a low operative mortality and is associated with superior late survival in patients with valve prolapse.

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DISCUSSION

DR GREGORY L. KAY (Los Angeles, CA): I would like to thank The Society and the committee for the privilege of opening the discussion of this excellent paper.

The authors classify patients with ischemic mitral regurgitation as either having restricted or prolapsing leaflet motion and have demonstrated the apparent impact of this division on survival.

Our interest in this disease entity began in the early 1960s, and we have a series of more than 260 patients at this time who have undergone operation for ischemic mitral regurgitation. We have found that the important prognostic determinants for survival of these patients are ejection fraction and New York Heart Association classification. Compromised ventricular function (ejection fraction < 0.4) and New York Heart Association class IV were indicative of poor operative and long-term outcome. Our series also demonstrates that valve repair is superior to valve replacement, particularly for the more compromised patients.

Our long-standing interest in mitral valve repair allows us to report on its excellent durability. More than 90% of patients who are alive at 12 years have not required further operation for mitral valve disease.

In summary, mitral valve repair is durable and has the most impact on survival in patients who are most compromised. Noel Mills has often stated that, in regard to coronary artery disease, "Anatomy is destiny." The same statement can be made about this variant, ischemic mitral regurgitation, and as always, the surgeon's choice of procedures has a major impact on the immediate and long-term outcome.

I would like to ask Dr Hendren and associates two questions: As you have gained experience with mitral repair, are you now more aggressive in repairing these valves than before? Also, has your technique evolved over time?

DR JAMES H. OURY (Missoula, MT): I would like to thank Dr Hendren and Dr Cosgrove for bringing to our attention a very timely subject, namely, the surgical treatment of ischemic mitral valve disease. They have contributed a great deal to this effort in the past, and I am sure they will continue, as we have seen today. My colleagues and I would also make a plea for a classification and a systematic approach to this still rather confusing entity. I would like to make two points and pose two questions for Dr Hendren.

In reviewing a consecutive series of 161 patients with combined coronary artery disease and mitral insufficiency over a 5-year period from 1984 to 1989, we found three groups with differing valve pathology, operative mortality, and prognosis. The average age in this group of patients was, again, very similar to Dr Hendren's series, 67 years of age; the mean follow-up is 2.7 years. The classification fell into three major categories: the first, coronary artery disease with annular dilatation, which would correspond to Dr Hendren's restricted leaflet motion, we would call a functional classification; the second would be organic, that is, coronary artery disease with ischemic, dysfunctional or infarcted papillary muscle; the third, associated coronary disease with leaflet or chordal pathology. The incidence of repair versus replacement is quite different in these three categories, and the operative mortality is correspondingly different.

For instance, in the functional category, coronary artery disease with annular dilatation, 29 of the 32 patients had repair; the operative mortality was 12%, and the actuarial 5-year survival was about the same as in the series presented by Dr Hendren. The very high risk group, which we have classified as the organic group, would be coronary artery disease with ischemic, dysfunctional, or infarcted papillary muscle. The operative mortality is quite a bit higher; however, actuarial survival is about the same. The third group, coronary artery disease with leaflet or chordal pathology, including redo procedures, accounted for the largest number in this particular series, and about half of these patients underwent repair. The operative mortality, as you would expect in this series, is quite low (6%), and the late actuarial 5-year survival is higher.

Based on this series we would add two notes of caution. Given this age group, the average age being 69 years, we found that there was no significant difference in intermediate survival whether patients had repair or replacement. Again, I would stress that in the replacement category, we used chordal sparing techniques both in the anterior and posterior leaflet, and I think this is very important in this particular entity. Mitral valve repair, therefore, we thought should be reserved, in general, for younger patients in classifications I and III.

We agree with Dr Hendren's comment regarding the use of a flexible ring as being important. We favor the flexible ring in all of our annuloplasties. One further point that I would like to introduce is that, in the ongoing debate of flexibility versus the rigid ring annuloplasty, the flexible ring and its recipient do appear to maintain their flexibility at extremes of both altitude and temperature! One of the patients in our series recently trekked to 18,000 feet on Mt Everest.

I have two questions for Dr Hendren. Mitral regurgitation in the setting of the ischemic ventricle may be highly variable depending on variations in preload and afterload. Have you devised or used a method of predicting intraoperatively the need for mitral valve repair in this group? Second, did your intermediate-term results demonstrate a statistically significant difference in mortality between mitral valve repair and mitral valve replacement?

DR ROBERT W. M. FRATER (Bronx, NY): Thank you for the privilege of discussing this report. It makes an extremely important point. The normal mitral valve, when it opens, moves down into the ventricle as the posterior left ventricular wall elongates. The normal mitral valve closes by annular contraction and also by the movement of the papillary muscles and the cusps up back into the annulus. Patients with posterior infarcts who have grade III or IV mitral insufficiency in the coronary care unit needing acute operation most often have failure of shortening of the posterior left ventricular wall as their mechanism of insufficiency. It stays down there during contraction and the cusps do not meet.

You can compensate for that deficiency in posterior left ventricular wall function by performing an annuloplasty; you will notice that in this report, annuloplasty was a fundamental part of the repair and the only repair in a substantial number of cases. I think the recognition that annuloplasty will compensate for a failure of shortening of the posterior left ventricular wall transforms operation for acute mitral insufficiency and coronary disease in patients coming out of the critical care unit.

We have followed this principle now for some years, and it has made an enormous difference to our approach. It can be seen on echocardiography; you can see the failure of contraction. You can anticipate that with normal cusps and chordae, gross mitral insufficiency can be corrected by a simple annuloplasty. I congratulate Dr Hendren and associates for making this very important point.

DR HENDREN: We would like to thank the discussants for their comments. Dr Kay asked whether any changes in our technique have evolved based on our experience. We have been concerned somewhat with the use of a rigid ring, knowing that experimental fixation of the annulus with a rigid ring has impaired systolic function, and, further, that it may interfere with the saddle shape of the annulus, which helps distribute the stress during systole. In the latter part of our experience, we have used a strip of glutaraldehyde-treated bovine pericardium in lieu of a rigid ring, which essentially results in multiple small plications of the posterior annulus. We think that this may help preserve the natural shape of the annulus, and we have preliminary data that suggest the left ventricular function in the short term is better preserved.

The issue of whether we are encouraged and are more aggressive with repairing ischemic mitral valves as opposed to valve replacement was raised. The answer is yes. There are certainly rare instances of severe rheumatic disease or severe degenerative disease that may make repair not feasible. However, we believe that because a major advantage of mitral valvuloplasty, when compared with mitral valve replacement, is better preservation of left ventricular function, the more seriously left ventricular function is impaired the more compelling the argument is to proceed with valvuloplasty.

Dr Oury asked a question regarding our experience with valve replacement in the setting of ischemic mitral regurgitation. We did not have a large enough number of patients with valve replacement during this time period to compare, and so those data are not available. Our approach to the ischemic valve is that if there is intermittent ischemia, then a mitral valve procedure is not needed. If there is serious regurgitation that cannot be eliminated with medical management, then it is our belief that revascularization alone will not be adequate, and we will proceed with a valve procedure. Once we reach that decision-making point, the decision to proceed with a valve procedure is made as it is for every other cause of mitral insufficiency.

Dr Frater, we agree with your assessment of the basis of regurgitation.

Bronchopleural Fistula After Stapled Closure of Bronchus

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The incidence of bronchopleural fistula after stapling among 2,243 pulmonary resections at the Rush-Presbyterian-St. Luke's Medical Center has been reviewed. There were 35 fistulas in 1,773 stapled and in 470 sutured bronchi (segmentectomy, 2; lobectomy, 1; bilobectomy, 9; and pneumonectomy, 23). We have found that the stapler is expedient and simple to use, and that it produces a hermetic and uniform closure. The stapler is contraindicated when the bronchus is thickened, inflamed, or of insufficient length. The overall incidence of bronchopleural fistula was 1.6%. Approximately two thirds of the patients with bronchopleural fistula had preoperative radiation therapy or chemotherapy or both.

(Ann Thorac Surg 1991;52:1253–8)

Although pulmonary resections have become frequent and standard procedures, the occasional occurrence of bronchopleural fistula postoperatively continues to be an important complication that deserves further scrutiny and attention. The best method of bronchial closure is still a controversial topic that evokes many different opinions.

Data and Results

We have reviewed the records of 2,243 patients undergoing pulmonary resection at the Rush-Presbyterian-St. Luke's Medical Center from December 1, 1970, through June 30, 1989, to investigate the incidence of bronchopleural fistula after stapling. The procedures done are shown in Table 1.

The majority of the resections were done for neoplastic diseases. The TA-30 or the TA-55 (Auto Suture Co, United States Surgical Corp, Norwalk, CT) was used for bronchial stapling in 1,773 patients. For a pneumonectomy or a lobectomy, staples with a leg length of 4.8 mm and a closed height of 2.0 mm were used; for smaller bronchi, staples with a leg length of 3.5 mm and a closed height of 1.5 mm were used. In 470 patients suture of the bronchus was done because the bronchus was grossly thickened, inflammatory changes were noted, or there was insufficient length of bronchus or margin of normal tissue from the tumor to allow application of the stapler. The pulmonary artery was stapled in 443 instances and the pulmonary vein in 1,738 with no complications.

Bronchopleural fistula developed postoperatively in 35 of the 2,243 patients (1.6%). Fistulas developed in 30 of the 1,773 patients in the stapled group (1.7%) and 5 of the 470 in the sutured group (1.1%). The diagnosis of bronchopleural fistula was established in these patients by clinical and radiologic findings and usually confirmed by bronchoscopy. Thirty-three of these patients were operated on for bronchogenic carcinoma, 1 for mesothelioma (an extrapleural pneumonectomy), and 1 for an aspergilloma (a superior segmentectomy with part of the posterior basal segment in a patient who had previously had a right upper lobectomy). Of the 33 patients with carcinoma, 21 had squamous cell, 9 adenocarcinoma, 2 small cell carcinoma, and 1 large cell carcinoma.

Fourteen of the 33 patients with carcinoma received radiation therapy only preoperatively, an additional 6 were given both irradiation and chemotherapy, and 13 had no preoperative treatment. Preoperative radiation therapy or chemotherapy was given because of clinical stage III disease.

The most frequent time for development of these bronchopleural fistulas postoperatively was between 10 and 15 days, with a mean of 44 days and a range from 7 to 210 days.

There were 23 fistulas in the 506 pneumonectomy patients (4.5%), 20 in 489 (4.1%) who were stapled and 3 in 17 (17.6%) who were sutured. However, comparison is not appropriate as suturing was done in most instances when stapling was contraindicated.

There were 10 fistulas in the 965 bilobectomy and lobectomy patients, 8 of which were stapled and 2 sutured, and 2 fistulas in the 319 segmentectomy patients, both stapled (Table 2). The fistulas in the segmentectomies were bronchial, not parenchymal leaks. When the incidence of bronchopleural fistulas was analyzed according to tumor stage, history, type of resection, and whether or not preoperative radiation therapy or chemotherapy was given, no statistically significant relationships using the χ^2 test were found (Table 3).

Of the 23 bronchopleural fistulas after pneumonectomy, 17 occurred on the right and six on the left. Of the pneumonectomy patients in whom bronchopleural fistulas developed, 15 had preoperative irradiation and 5 received chemotherapy also.

In patients who received preoperative irradiation, a

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Table 1. Method of Bronchial Closure in 2,243 Pulmonary Resections

Resection	Total	Stapled	Sutured
Segmentectomy	650	319	331
Lobectomy/bilobectomy	1,087	965	122
Pneumonectomy	506	489	17
Total	2,243	1,773	470

significantly longer time (average, 48 days) elapsed before the diagnosis of bronchopleural fistula than in the other patients (average, 18 days). The data regarding bronchopleural fistula pertaining to time of diagnosis were analyzed using the Mann-Whitney U test (normal approximation) and that pertaining to the risk of fistula, by Fisher's Exact test.

Comment

The technique of bronchial stump closure and prevention of bronchopleural fistula have been of primary concern since the beginning of pulmonary resections. Many clinical efforts and experimental studies have been made regarding this subject. Meyer [1] recommended the inversion technique similar to that in appendectomy. The cartilage is first crushed or removed before placement of the inversion sutures (Fig 1).

Several years later Sweet [2] advised the use of single interrupted sutures of "medium silk," placing them over the end of the bronchus and burying the stump beneath a pleural flap; he thought the use of mattress sutures illogical. In a series of 80 lobectomies and 61 pneumonectomies using this method he found only one instance of bronchial fistula (Fig 2).

In an attempt to decrease the incidence of bronchopleural fistula and the associated mortality and to simplify lung resections, the Russians Gorkin and Strekopytov developed various mechanical stapling devices in 1957 [3]. The clinical application of bronchial stapling in several thousand patients was reported first by Amosov and Berezovsky [4], who thought it superior to conventional hand suturing because of expediency, simplicity, decreased surgical trauma, and the ability to produce a hermetic approximation of the tissues. The original design, the UKB stapler, placed a single row of metal sutures parallel to the axis of the bronchus. The subsequent instrument, the UKL, placed two staggered rows of sutures perpendicular to the bronchial axis.

Table 2. Bronchopleural Fistulas According to Type of Resection

Procedure	No.	Stapled	Sutured
Segmentectomy	2	2	. . .
Lobectomy	1	1	. . .
Bilobectomy	9	7	2
RUL and RML	4	4	. . .
RML and RLL	5	3	. . .
Pneumonectomy	23	20	3
Right	17	15	2
Left	6	5	1
Total	35	30	5

RLL = right lower lobe; RML = right middle lobe; RUL = right upper lobe.

The Russian stapler was brought to the United States by Ravitch [5, 6] and subsequently underwent several technological improvements (United States Surgical Corp, Norwalk, CT). The initial report by Ravitch and associates [5] described 139 pulmonary resections with three bronchopleural fistulas (two occurring in diabetic patients and one in a patient with active tuberculosis) using the Russian UKB stapler.

With the newly developed American staplers (TA-30 and the TA-55), Kirksey and co-workers [7] reported on 147 pulmonary resections; there was only one bronchopleural fistula and two bronchial air leaks that healed spontaneously. During the same year, Dart and colleagues [8] wrote about their experiences using both the Russian and American staplers in 493 patients. With the Russian stapler there were eight bronchopleural fistulas, but there were none with the American stapler.

Some spoke enthusiastically about the advantages of the stapling instrument for bronchial closure [9–13], whereas others resisted this new technology [14–16].

Experimentally, Scott and associates [17] studied bronchial healing after closure with silk, catgut, and the stapling device. They concluded: ". . . stumps closed with the automatic stapling device (TA-30) showed the best healing with a minimal degree of inflammation. These findings correlated well with leakage pressures." The next year Scott and associates [18] extended their studies to include nylon sutures and measured the collagen levels. Again, the bronchi that were stapled proved superior in collagen levels, had the least inflammatory reaction, and withstood the greatest pressures before disruption. Scott and associates commented: "These results have clinical relevance to the selection of suture materials for stump closures."

Lynn [19] reviewed the incidence of bronchopleural fistula after resection for tuberculosis in numerous collected series in the United States, Great Britain, Canada, and France between 1951 and 1958, and found it to be 6.7% (see also Table 4).

The results of bronchial closure vary from surgeon to surgeon [20–23], and the cause of bronchial fistula is multifactorial. Numerous studies indicate that the causes of bronchial fistula include devitalization and devascularization by excessive dissection, peribronchial infection related to nonabsorbable suture, residual bronchial disease, poor approximation of the mucosa, the length of the stump, and the surgeon's experience [24–26].

To avoid the complication of bronchopleural fistula, we consciously dissect as little of the peribronchial tissue as possible to maintain vascularity of the closed stump.

A uniform and rapid closure of the bronchus can be accomplished by use of the stapling technique, but familiarity with the instrumentation and awareness of con-

Table 3. Bronchopleural Fistulas: Type of Resection, Cell Type, Stage, Preoperative Therapy, Time of Onset, and Closure

Resection	Patient No.	Cell Type	Surgical Stage	Preop Treatment	Time of Onset (d)	Closure
Segmentectomy	1	Squamous	I	No	17	Staple
	2	Aspergilloma	. . .	. . .	14	Staple
Lobectomy	3	Squamous	II	RT	46	Staple
RUL/RML	4	Large cell	I	RT	14	Staple
	5	Squamous	I	No	20	Staple
	6	Adeno	II	No	10	Staple
	7	Squamous	I	No	27	Staple
RML/RLL	8	Adeno	IV[a]	No	10	Staple
	9	SCC	III	RT	155	Staple
	10	Squamous	I	RT	79	Suture
	11	SCC	I	RT/Chemo	70	Suture
	12	Squamous	I	No	14	Staple
Pneumonectomy						
Left	13	Squamous	III	RT	39	Staple
	14	Squamous	II	RT	7	Suture
	15	Squamous	III	No	11	Staple
	16	Adeno	III	RT/Chemo	63	Staple
	17	Squamous	0	RT	47	Staple
	18	Squamous	III	RT	11	Staple
Right	19	Squamous	0	RT	74	Staple
	20	Squamous	I	No	210	Staple
	21	Adeno	I	RT/Chemo	15	Staple
	22	Squamous	0	RT	16	Staple
	23	Adeno	I	No	14	Suture
	24	Squamous	I	RT	35	Staple
	25	Adeno	III	RT	13	Staple
	26	Adeno	III	No	21	Staple
	27	Squamous	III	RT	141	Staple
	28	Meso	. . .	No	170	Staple
	29	Adeno	IV[b]	RT	21	Staple
	30	Adeno	IV[b]	RT/Chemo	22	Suture
	31	Squamous	II	No	13	Staple
	32	Squamous	0	RT/Chemo	42	Staple
	33	Squamous	III	RT/Chemo	57	Staple
	34	Squamous	III	No	12	Staple
	35	Squamous	III	No	21	Staple

[a] Brain metastasis previously resected. [b] Pleural or distant metastases.

Adeno = adenocarcinoma; Chemo = chemotherapy; Meso = mesothelioma; RT = radiotherapy; SCC = small cell carcinoma.

traindications to its use must be present. Various stapling guns are available, and the surgeon must be familiar with their advantages and disadvantages.

Use of the proper size of staple is extremely important. Closure of the anvil to the indicated mark provides a constant space (1.5 mm or 2.0 mm) between the jaws for bronchial approximation. The leg length of the staple determines the tension of the bronchial approximation. If the bronchus is large or particularly thick, a smaller staple will pull through the bronchial wall, and the compressed closure further compromises blood supply to the cut ends of the bronchus. We use the staple with a 4.8-mm leg length for all pneumonectomies and the majority of lobectomies. In an adolescent or small female patient in whom the lobar bronchus is small, we will use the staple with a 3.5-mm leg length. The smaller segmental bronchus is routinely closed with the staple with a 3.5-mm leg length.

Proper positioning of the stapler with the jaws perpendicular to the long axis of the bronchus and parallel to the mucous membrane is obligatory. This should be done without excessive bronchial traction to avoid inclusion of the proximal airway in the stapler. The distal bronchus is

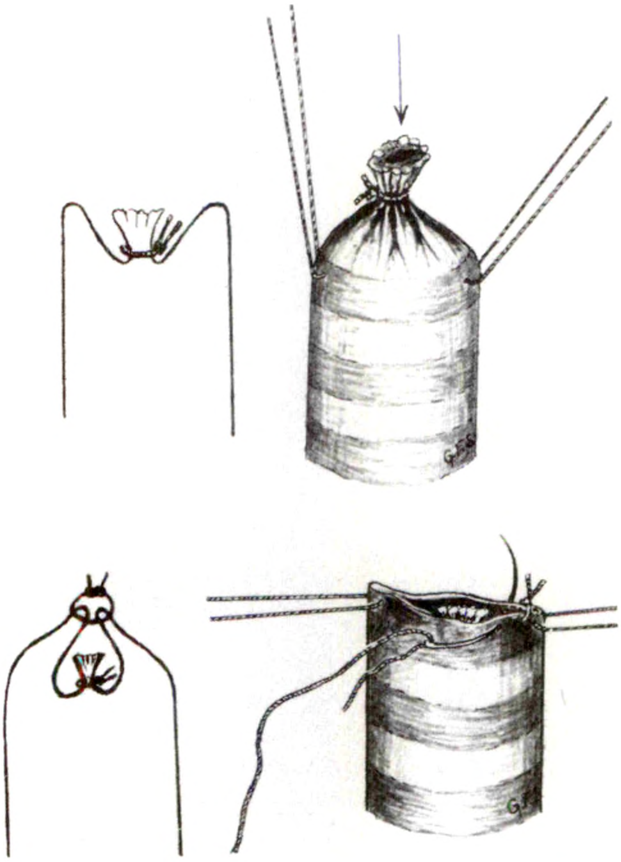

Fig 1. Method of bronchial closure as described by Meyer in 1909. He emphasized removing the cartilage, ligating the mucosa, and inverting the stump.

firmly grasped between the thumb and forefinger to flatten the cartilage rings and to avoid bending over of the cartilage wall. We believe this is important in pneumonectomy; obviously it is difficult to do when performing a lobectomy. The bronchus is transected while the jaws are still approximated and flush with the jaw on the distal bronchial side. After transection of the bronchus the specimen is examined grossly and by frozen section for evidence of pathology, particularly with a neoplasm.

After stapling, the closed bronchus is tested for air leaks at 20 cm H_2O of airway pressure. When small air leaks are identified where the staples have cut through the cartilage, these can be closed with interrupted 4-0 absorbable sutures. We make every attempt to achieve complete pneumostasis.

The right pneumonectomy stump is covered with available tissue such as pleura, mediastinal fat pad, pericardium, or an intercostal muscle flap. We do not routinely cover the left pneumonectomy stump because it retracts into the mediastinum.

The surgeon should be aware of contraindications to closing the bronchus by the stapling technique. If the cancer is close to the bronchial orifice as observed by bronchoscopy or external appearance and a larger resection cannot be done, we do not use a stapler. In this instance the bronchus is transected by knife and both the proximal and distal margins are inspected. The technique of knife transection and suture is done when there is a question as to the proximal extent of the cancer. We recommend closure with interrupted monofilament absorbable sutures. A thickened and inflamed bronchus is a relative contraindication to use of the stapler.

We do not advise two applications of the staples to close a pneumonectomy stump. This technique creates a longer length of devascularized tissue and would only enhance formation of a fistula. The use of additional absorbable

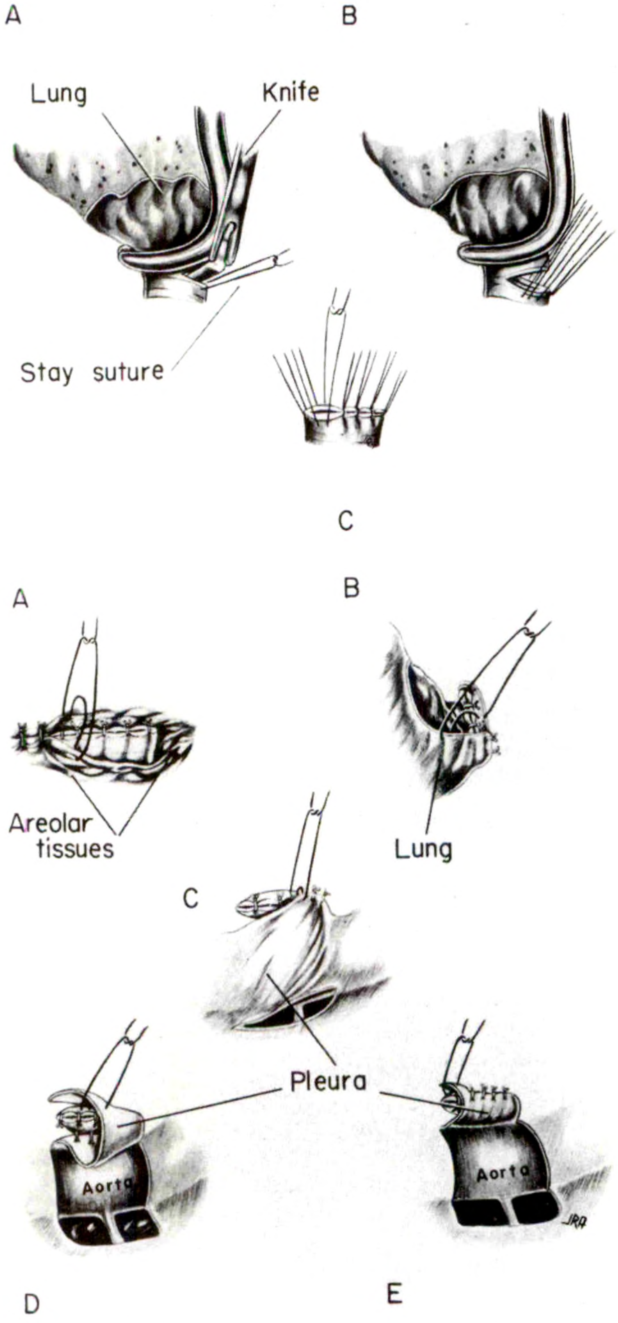

Fig 2. Method of bronchial closure advocated by Sweet [2]. He emphasized simple over-and-over sutures, a minimal amount of trauma and dissection (top), *and coverage of the stump* (bottom). *(Reprinted from Sweet RH. Thoracic surgery. Philadelphia: W.B. Saunders, 1950:148–9, by permission.)*

Table 4. Bronchopleural Fistula After Pneumonectomy[a]

Study	Sutured	Stapled
Schaefer et al, 1977 [20]	10/85 (10.6)	7/111 (6.3)
Verain et al, 1979 [21]	5/33 (15.1)	1/33 (5.3)
Forrester-Wood, 1980 [22]	25/225 (11.1)	6/225 (2.7)
Lawrence et al, 1982 [23]	3/45 (6.6)	2/37 (3.9)
Vester et al, 1991 (present study)	3/17 (18.2)	16/307 (5.2)
Total	46/405 (11.4)	36/895 (4.0)

[a] Numbers in parentheses are percentages.

sutures to reinforce an already airtight suture line requires the surgeon's judgment. The placement of these sutures will further compromise blood supply.

When performing a lobectomy, care must be taken to avoid compromise of the adjacent lobar or main bronchus. If the stapling device is placed too far proximal, narrowing of the bronchus may occur with resultant narrowing of the airway. This is most likely to occur with a left upper lobectomy.

Although not statistically significant, the incidence of bronchial fistula after pneumonectomy appears greater in those patients who have had preoperative irradiation or chemotherapy. Fifteen of the 20 pneumonectomy fistulas were in patients who had received preoperative irradiation, and 5 also received combination chemotherapy. Radiation affects the blood supply; grossly at operation there is increased perihilar fibrosis. After preoperative treatment for stage III neoplasms we make every attempt to preserve bronchial blood supply during subcarinal and mediastinal lymph node dissection.

There are disadvantages to use of the stapler, which include its occasionally cumbersome insertion and application. Closure of the stapling gun can crush the bronchial tissues, and with a thickened bronchus the stapling technique is contraindicated with the presently available staples.

Despite the occurrence of an occasional fistula after bronchial closure by the stapling technique, the stapling technique remains our choice for bronchial closure. The closure is precise, expedient, uniform, and airtight.

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DISCUSSION

DR WILLARD A. FRY (Evanston, IL): My experience in stapling bronchi for segmental resection in small people, particularly women, is that the stapled stump will sometimes leak around the 3.5-mm staples, which are the smallest of the standard bronchial closure staples. I have consequently over recent years simply sutured those small segmental bronchi; it only takes a few sutures. But I wonder if the 30 V might not be an acceptable staple closure for such small bronchi.

DR VESTER: We have rarely used the vascular staples for bronchial closure. We also have had a similar experience of a slight tear in the bronchial closure caused by the large staple, and these small defects do require the placement of interrupted sutures. Our experience with staple or suture closure of a segmental bronchus has always been individual surgeon preference and, as you have stated, it is frequently easier to close a smaller bronchus with sutures.

DR JOEL D. COOPER (St. Louis, MO): I have not been in the habit of using the stapler on the airway. I was heavily influenced by Michael Perlman, who is the head of the institute in Moscow, where they have, as you pointed out, invented the staplers. What impressed me is when I asked him 8 or 9 years ago if he used the staplers and he said, "On the vessels always, on the bronchus never, except very supple bronchus or in children." And I think the point to be made is that the stapler can be used safely, but there are some circumstances under which perhaps it cannot. I honestly believe that the incidence of leakage with stapler is every bit as high as with manual closure. People do not report their bad results with staplers; they only report their good results.

My main concern is particularly related to right pneumonectomy, where, if it is close to the trachea, you have a very strong strut on the medial aspect, which is the carina. It is one of the points that Dr Perlman made as well. You pointed out that an advantage of the stapler is the uniform application of the device, but I would argue that that is one of the disadvantages if you are working on an airway, which is nonuniform in terms of its elasticity and thickness. Under what circumstances do you think the stapler should be avoided?

DR VESTER: There are very definite contraindications to the use of the stapling technique for bronchial closure, and a surgeon must always keep these in mind. Preresection bronchoscopy by the thoracic surgeon is mandatory to permit the opportunity to visualize the mucosa of the bronchus to be transected, as well as the exact location of the cancer. We believe that serious endobronchial mucosal inflammation or severe bronchitis is a contraindication to stapling. When the cancer is in close proximity to the area of bronchial transection either by endoscopic visualization or palpation at the time of thoracotomy, the staple instrument is not used. In this instance, the bronchus is sharply transected with a knife and both the proximal and distal margins can be inspected. As you have pointed out, a substantially thickened bronchus or a bronchus in which a ring of cartilage could not be approximated satisfactorily would be a contraindication to stapling. In this situation, the bronchus is crushed by closure of the anvil and the staples tend to pull through with a resultant fistula. This requires careful selection and judgment by the surgeon as to whether to staple or hand suture the bronchus. When performing a pneumonectomy, we make every attempt to flatten the membrane against the cartilage so that we do not fold over the end of the lateral bronchial wall. Any time the surgeon believes that the bronchus is too thick for stapling or it is calcified, then stapling is not indicated.

DR THOMAS W. RICE (Cleveland, OH): Have you been able to categorize the bronchopleural fistulas into those resulting from V closure staplers versus those resulting from parallel closure staples?

DR VESTER: I am sorry, I do not know what you mean by V closure.

DR RICE: There was a stapler that had a V closure of the jaws, and the incidence of bronchopleural fistula was increased with that instrument.

DR VESTER: You are absolutely correct in stating that this type of staple cartridge was associated with a higher incidence of bronchopleural fistula. It was related to the fact that there was not a uniform application of compressive pressure on the bronchus just before the instrument was fired. We immediately recognized the defect with this type of device and never used it. We have always used the original TA-30 and TA-55 stapling instruments for their advantages of reliability and reproducible results.

DR STEVEN W. GUYTON (Seattle, WA): I am sorry I did not get here for your presentation of data, but I think the sweeping conclusions that you make are not supported by the data that I got out of the abstract. What I see in the abstract is that you have fewer leaks with segmentectomy with suture closure than with staple closure, and it seems like the staple closure should be applied selectively rather than as a sweeping statement.

DR VESTER: We have reviewed our experience with staple closure of the bronchus and are reporting the incidence of bronchial fistula after pneumonectomy, lobectomy, and segmentectomy. A true comparison of staple closure versus suture closure of a bronchus would require a randomized, prospective study, and this review is retrospective. Suture closure after pneumonectomy and lobectomy was accomplished primarily in those patients in whom the use of the stapling technique was contraindicated and incidence of fistula cannot be compared. The closure of the segmental bronchus was primarily at the discretion of the operating surgeon, and there was no statistical difference in incidence of fistula after staple or suture closure of the smaller bronchi. Selectivity is always appropriate when applying any surgical technique.

Bilateral Latissimus Dorsi Cardiomyoplasty

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This study was undertaken to test the hypothesis that a bilateral latissimus dorsi cardiomyoplasty provides greater hemodynamic augmentation than a unilateral procedure. Two types of bilateral procedure and a left posterior cardiomyoplasty were tested in each of 8 mongrel dogs. R-wave synchronous muscle pacing was achieved with a programmable burst stimulator. Hemodynamic variables of stimulated beats were compared with those of a nonstimulated baseline using paired *t* tests. The effects of a double anterior muscle wrap were equal to a right anterior/left posterior configuration. Therefore, the data on the two types of bilateral procedure were combined and compared with the left wrap. Stimulation of the bilateral cardiomyoplasty resulted in significant increases in right ventricular pressure (44 ± 3.1 versus 26 ± 1.8), first derivative of right ventricular pressure (595 ± 117 versus 196 ± 14), pulmonary artery pressure (34 ± 1.9 versus 23 ± 1.6), left ventricular pressure (90 ± 5.9 versus 69 ± 5.3), first derivative of left ventricular pressure (1454 ± 141 versus 1072 ± 107), aortic pressure (80 ± 5.4 versus 67 ± 4.9), and peak aortic flow (9.4 ± 1.1 versus 7.7 ± 0.8) ($p < 0.05$). Significant increases in all of these variables also occurred with stimulation of the left cardiomyoplasty, but the increases in right ventricular pressure, first derivative of right ventricular pressure, pulmonary artery pressure, and aortic pressure were larger for the bilateral than the left cardiomyoplasty. The bilateral and the left procedure can each augment systolic ventricular function. The bilateral procedure appears to have greater effects, especially on right ventricular function.

(Ann Thorac Surg 1991;52:1259–65)

Congestive heart failure is an important cause of disability, morbidity, and mortality throughout the world. Medical therapy has had only a small effect on the natural history of the disease. Heart transplantation has been dramatically successful in small numbers of selected patients, but the procedure has had relatively little impact on the overall problem. Cardiomyoplasty is a new surgical procedure that uses paced conditioned skeletal muscle to augment cardiac function [1–4]. Acute experimental studies have shown that left latissimus dorsi cardiomyoplasty can improve left ventricular function [5–7], but objective evidence of hemodynamic change has not been consistent in clinical trials of the procedure [8].

In theory, greater hemodynamic effects should be seen if both latissimus dorsi muscles are used for cardiomyoplasty. An acute study demonstrating that bilateral cardiomyoplasty was feasible was recently published, but no comparison with a unilateral muscle wrap was included [9]. This report describes two techniques for bilateral latissimus dorsi cardiomyoplasty and analyzes their acute hemodynamic effects in the dog, as compared with that of a unilateral left posterior cardiomyoplasty.

Material and Methods

Eight adult mongrel dogs weighing 25 to 30 kg were used. All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals," published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). Anesthesia was induced with intravenous thiopental (25 mg/kg) and pancuronium bromide (0.1 mg/kg). The animals were then intubated and ventilated with 1% enflurane to maintain anesthesia.

Operative Procedure

The cardiomyoplasty procedures were done in two stages separated by 7 to 10 days. In the first stage the muscles were mobilized from the chest wall and subcutaneous tissue through bilateral flank incisions. Each arm of a specially fabricated bifurcated bipolar pacing lead (Medtronic, Inc, Minneapolis, MN) (Fig 1) was secured to the thoracodorsal nerve on each side. The opposite end of the lead was buried in the subcutaneous tissue over the abdominal wall, where it could be retrieved in the second stage of the operation. The delay period between the two stages allowed time for the muscles to recover from interruption of collateral blood supply and ensured that they were well vascularized during the second stage of the experiment.

In the second stage the flank incisions were sequentially reopened. The origin of each muscle was divided, but the insertion on the humeral head was left intact. A 5-cm segment of the third rib was removed and each muscle was placed into the respective hemithorax. The wounds were closed and the animals were then placed in a supine position. A midline sternotomy incision was made. The pericardium was opened in the midline and suspended with sutures. The muscles were retrieved from the chest cavities. Two epicardial unipolar sensing leads (model

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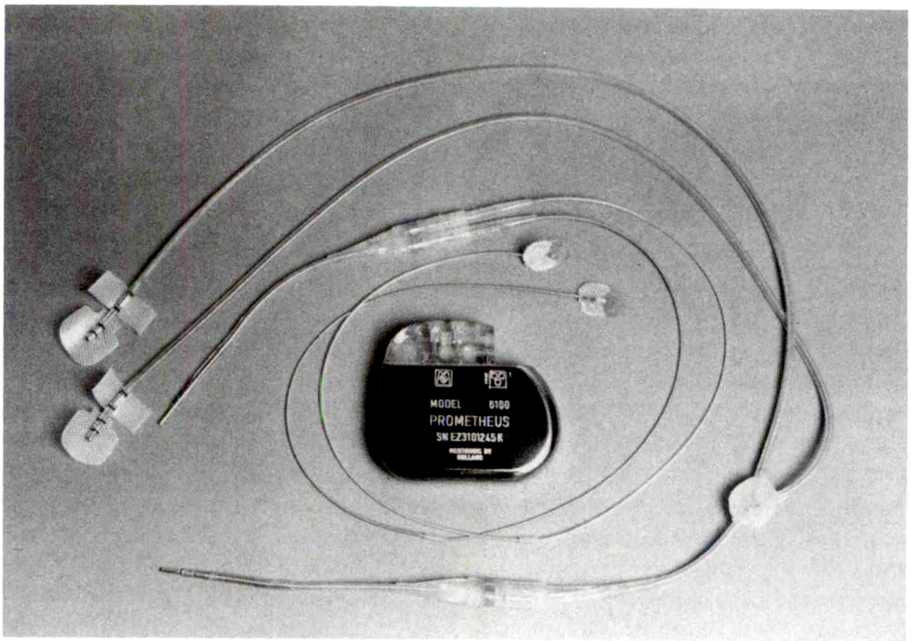

Fig 1. The Medtronic Prometheus Burst Stimulator (center), the two epicardial sensing leads (adjacent to the stimulator), and the specially fabricated bifurcated pacing lead (outside perimeter).

4951, Medtronic) were attached to the anterior wall of the right ventricle and joined using an adaptor (model 5866-24, Medtronic). The pacing and sensing leads were connected to the Prometheus (model 6100, Medtronic) pulse generator.

Two types of double muscle wrap were constructed in each animal: (1) The double anterior wrap (DAW), in which both the left and the right muscle were brought anteriorly in a criss-cross fashion, and (2) the right anterior, left posterior wrap (RALP), in which the right muscle was brought anteriorly and the left muscle was brought posteriorly. A posterior orientation of the right muscle compressed the inferior vena cava and right atrium and was therefore not technically satisfactory.

In the DAW, the right wrap was done first (Fig 2). The anterior border of the right latissimus dorsi was brought across the right and left ventricles. The distal end of the flap was sutured to the pericardium at the level of the atrioventricular groove, starting adjacent to the inferior vena cava and extending around to the vicinity of the left atrial appendage. The left muscle was then brought across and sutured to the right muscle at the level of the atrioventricular groove.

In the RALP wrap the left flap was done first (Fig 3). The anterior border of the latissimus dorsi was brought behind the heart and sutured to the pericardium at the atrioventricular groove. The scapulospinal border of the muscle was brought around the heart and sutured to itself anteriorly. The right muscle was then brought anteriorly across the heart and sutured to the left muscle at the level of the atrioventricular groove. In both types of wrap the pericardium was sutured to the adjacent latissimus dorsi muscle to minimize displacement of the heart during muscle stimulation.

Hemodynamic Measurements

After the sternotomy incision was made, but before the cardiomyoplasty was performed, the following prewrap hemodynamic variables were measured: (1) mean right and left atrial pressures; (2) systolic, diastolic, and mean pressures in the right ventricle, pulmonary artery, left ventricle, and ascending aorta; (3) peak aortic flow; and (4) first derivative of pressure with respect to time (dP/dt) of right and left ventricular pressure. Right-sided pressures were measured through a Swan-Ganz catheter and left-sided pressures were measured through polyethylene catheters filled with saline solution. Disposable transducers were used. Aortic flow was measured with an ultrasonic flowprobe (Transonic Systems, Inc, Ithaca, NY). The data were recorded on a strip-chart recorder and simulta-

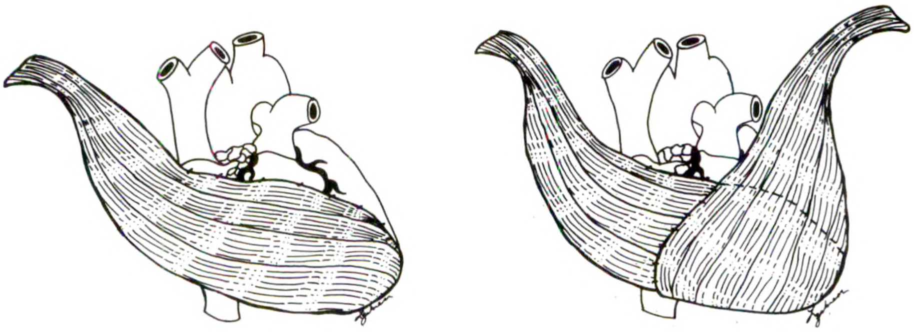

Fig 2. Construction of the double anterior muscle wrap. The right muscle is positioned first. The left muscle is brought across the right and secured to the pericardium at the level of the atrioventricular groove.

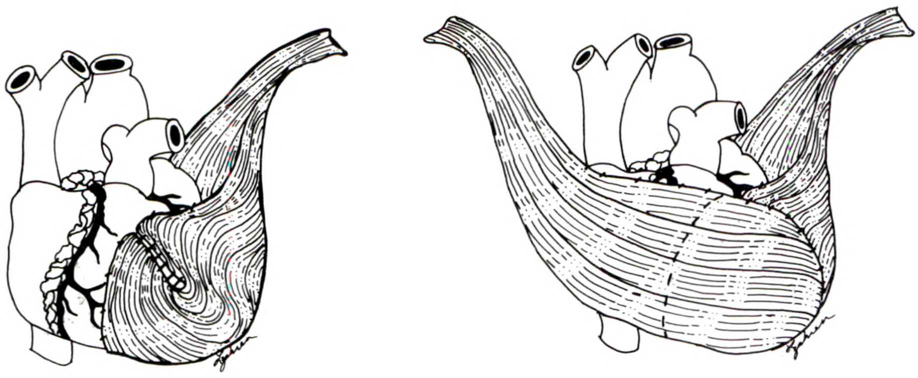

Fig 3. Construction of the right anterior, left posterior muscle wrap. The left muscle is brought behind the heart and sutured to itself anteriorly. The right muscle is brought anteriorly across the heart and secured to the left muscle.

neously entered into an IBM-AT personal computer, and processed using CODAS (Datag Instruments, Inc, Akron, OH) and ASYSTANT (Datag Instruments, Inc) software.

Hemodynamic variables were measured again after construction of the wraps, but before stimulating the muscles (postwrap baseline). The muscles were then stimulated at a rate of 1:4 with the native heart rate. Hemodynamic variables obtained with stimulated beats were compared with those of unstimulated beats. For purposes of analysis, the beat occurring just before a stimulated beat was used as the baseline for comparison with the stimulated beat. The ratio of 1:4 was chosen to ensure that the hemodynamic changes seen with stimulation were the result of augmentation of the stimulated beat rather than interference with the subsequent beat.

Experimental Sequence

After obtaining baseline hemodynamic measurements, we performed each type of bilateral cardiomyoplasty and a left posterior cardiomyoplasty in each animal. Half of the animals had the DAW done first and half had the RALP done first. After the studies on one type of double cardiomyoplasty were completed, the wrap was taken apart and the cardiac function allowed to stabilize. Once the hemodynamic state had returned to the initial baseline, the other type of double wrap was performed. The left posterior wrap was done as the first part of the RALP procedure in each animal. After the measurements were made the animal and the muscle were allowed to rest. The right muscle was then applied to complete the double wrap. The animals were killed after completing the evaluation of the different types of cardiomyoplasty.

Stimulation Parameters

All of the muscles were stimulated using a Prometheus model 6100 programmable burst stimulator (Medtronic). The unique feature of this stimulator is that the duration of the stimulus is determined as a percentage of the R-R interval of the preceding cardiac beats. We found empirically that a pulse duration of 30% of the R-R interval provided consistent augmentation with minimal impairment of the subsequent beat. The threshold for stimulation of the entire muscle using the stimulating lead was between 1 and 2 V. The muscles were stimulated at 5 V to ensure full recruitment of all fibers. The pulse frequency was 33 Hz, and the pulse width was 180 microseconds. The delay between recognition of the R-wave and pacing the muscle was 20 milliseconds.

Statistical Analysis

Each animal served as its own control. Prewrap and postwrap baseline hemodynamic variables were analyzed using paired *t* tests. Comparison of stimulated and unstimulated beats and analysis of differences between the single and double muscle wrap were also done using paired *t* tests. Significance was interpreted as p less than 0.05.

Results

Comparison of the Types of Bilateral Cardiomyoplasty

HEMODYNAMICS BEFORE AND AFTER CARDIOMYOPLASTY. Prewrap baseline hemodynamics were not different before construction of the DAW and the RALP muscle wraps. Both types of double wrap caused decreases in ventricular function, which were more marked on the left than on the right ventricle (Table 1). Nonetheless, all hemodynamic variables measured after construction of the bilateral wraps were still within a satisfactory range, and none of the animals could be considered hemodynamically unstable.

HEMODYNAMIC CHANGE DUE TO MUSCLE STIMULATION. The muscles were stimulated at a rate of 1:4 with the native heart rate. The average of five stimulated beats was compared with the average of five nonstimulated beats to determine hemodynamic changes. The data are presented in Table 2. Stimulation of each type of double wrap caused cardiac compression, which resulted in augmentation of both right and left ventricular variables. Significant increases were found with each type of bilateral cardiomyoplasty in right ventricular pressure, pulmonary artery pressure, dP/dt of the right ventricular pressure, left ventricular pressure, aortic pressure, and aortic flow. There were no significant changes in right or left atrial pressure during muscle stimulation. The increase in left ventricular dP/dt was significant for the RALP wrap but not for the DAW. Each of the two types of double wrap caused a comparable degree of hemodynamic change, and the only statistically significant differences between the two types of wraps was in left ventricular dP/dt.

Table 1. Hemodynamic Measurements Before and After Double Cardiomyoplasty With No Muscle Stimulation[a]

Variable	DAW		RALP	
	Pre	Post	Pre	Post
RA (mm Hg)	7.3 ± 1.3	9.3 ± 1.8	6.7 ± 1.3	9.2 ± 1.7
RV (mm Hg)	31.5 ± 2.8	28.8 ± 1.4	31.4 ± 2.0	25.8 ± 2.1[b]
RV dP/dt (mm Hg/s)	452 ± 75	229 ± 26[b]	372 ± 22	219 ± 28[b]
PA (mm Hg)	26.4 ± 2.7	22.1 ± 1.7	25.9 ± 2.1	25.0 ± 1.7
HR (beats/min)	116 ± 8.4	121 ± 7.7	113 ± 7.9	112 ± 5.5
LA (mm Hg)	7.9 ± 1.5	9.4 ± 2.0	10 ± 1.4	10.2 ± 1.7
LV (mm Hg)	108 ± 6.0	81 ± 3.2[b]	104 ± 6.3	72.6 ± 6.1[b]
LV dP/dt (mm Hg/s)	2086 ± 169	1299 ± 93[b]	1886 ± 132	1124 ± 109[b]
Ao (mm Hg)	98.3 ± 4.6	77.9 ± 3.2[b]	99.5 ± 3.6	71.9 ± 5.8[b]
AF (L/min)	15.1 ± 1.0	8.9 ± 0.7[b]	14.3 ± 0.8	8.2 ± 0.9[b]

[a] Data are presented as mean ± standard error of the mean. [b] $p < 0.05$, pre versus post.

AF = peak aortic flow; Ao = systolic aortic pressure; DAW = double anterior wrap; dP/dt = the first derivative of pressure with respect to time; HR = heart rate; LA = mean left atrial pressure; LV = systolic left ventricular pressure; PA = systolic pulmonary artery pressure; RA = mean right atrial pressure; RALP = right anterior, left posterior wrap; RV = systolic right ventricular pressure.

Representative tracings of the changes in right and left ventricular pressure seen with muscle contraction are shown in Figure 4. A consistent decline in early left ventricular diastolic pressure immediately after a stimulated beat was found in each animal.

Comparison of Left and Bilateral Cardiomyoplasty

Prewrap and postwrap baseline data and values measured during stimulation of the left posterior wrap are presented in Tables 3 and 4. The data for the two types of double cardiomyoplasty were combined, because both types were comparable in their hemodynamic effects. The combined double-wrap data were compared with those of the left cardiomyoplasty.

Both the left and the bilateral wraps caused some depression of normal cardiac function. The bilateral cardiomyoplasty resulted in more severe depression of left ventricular function than the left wrap, as evidenced by larger declines in left ventricular pressure, aortic pressure, and aortic flow. The left and the bilateral wraps each had small, but statistically significant effects on right ventricular variables.

Stimulation of the left and the bilateral wraps caused biventricular augmentation. Significant changes were found in all measured hemodynamic variables except atrial pressure. The bilateral cardiomyoplasty had a larger effect on right ventricular variables than did the left cardiomyoplasty, as shown by larger increases in right ventricular pressure, pulmonary artery pressure, and right ventricular dP/dt. A greater increase in aortic pressure was also found with the double wrap. However, the bilateral procedure also caused more depression of baseline aortic pressure than the left, so that the actual peak aortic pressure measured during stimulation were similar for the two procedures.

Table 2. Hemodynamic Effects of Bilateral Cardiomyoplasty[a]

Variable	DAW		RALP	
	Unstimulated	Stimulated	Unstimulated	Stimulated
RA (mm Hg)	9.3 ± 1.8	11.1 ± 1.3	9.2 ± 1.7	10.0 ± 1.7
RV (mm Hg)	27.5 ± 1.6	44.8 ± 2.7[b]	23.5 ± 2.2	43.0 ± 3.7[b]
RV dP/dt (mm Hg/s)	211.6 ± 17.5	549.0 ± 107.6[b]	180.0 ± 17.8	640.0 ± 140.6[b]
PA (mm Hg)	21.8 ± 1.7	31.3 ± 2.1[b]	23.6 ± 1.7	36.3 ± 2.6[b]
HR (beats/min)	120.6 ± 2.6	120.4 ± 1.9	111.5 ± 3.5	104.8 ± 22.6
LA (mm Hg)	9.4 ± 2.0	9.9 ± 1.9	10.2 ± 1.7	10.2 ± 1.9
LV (mm Hg)	74.8 ± 4.3	92.5 ± 6.3[b]	63.5 ± 6.5	87.1 ± 5.7[b]
LV dP/dt (mm Hg/s)	1217.8 ± 122.7	1435.8 ± 155.8	928.0 ± 116.3	1472.9 ± 168.4[b]
Ao (mm Hg)	71.4 ± 4.6	82.4 ± 5.4[b]	62.4 ± 5.6	73.5 ± 6.1[b]
AF (L/min)	8.3 ± 0.7	9.8 ± 0.7[b]	7.2 ± 0.9	8.9 ± 1.1[b]

[a] Data are presented as mean ± standard error of the mean. [b] $p < 0.05$, unstimulated versus stimulated.

AF = peak aortic flow; Ao = systolic aortic pressure; DAW = double anterior wrap; dP/dt = the first derivative of pressure with respect to time; HR = heart rate; LA = mean left atrial pressure; LV = systolic left ventricular pressure; PA = systolic pulmonary artery pressure; RA = mean right atrial pressure; RALP = right anterior, left posterior wrap; RV = systolic right ventricular pressure.

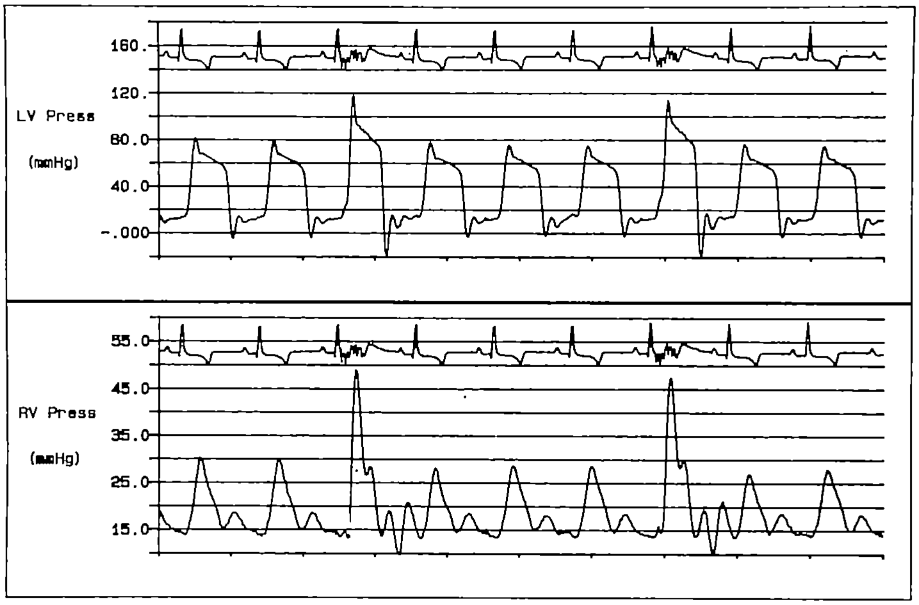

Fig 4. A representative recording of changes in ventricular pressures with stimulation of a bilateral cardiomyoplasty. Evidence of the burst stimulation is seen in the electrocardiographic tracing (top). Significant increases in left ventricular (LV) and right ventricular (RV) systolic pressure (Press) occur with stimulation. A consistent decrease in early diastolic left ventricular pressure after each stimulated beat is also evident.

Comment

We have concentrated our research efforts on using skeletal muscle for cardiac assistance because of the complications of mechanical circulatory support and the limited availability of organs for transplantation. Recent reports have indicated that cardiomyoplasty using the left latissimus dorsi can improve the hemodynamics of congestive heart failure [10–12], but these changes have not been consistent in the overall world experience with the procedure [8, 13].

The cardiomegaly that is present in humans undergoing cardiomyoplasty often prevents full coverage of both ventricles with a single left latissimus muscle, and it has been suggested that this might account for the lack of consistent hemodynamic augmentation in humans. This experimental study was undertaken to develop the technique for bilateral cardiomyoplasty and to evaluate its hemodynamic effects in comparison with the left.

The surgical technique for bilateral latissimus dorsi cardiomyoplasty is not difficult, but it is time consuming and somewhat tedious. There do not appear to be any significant differences in the hemodynamic effects achieved with the two types of bilateral cardiomyoplasty. The DAW was somewhat easier to perform than the RALP, because it can be done without lifting the heart out of the pericardium.

The major technical problem in developing the bilateral cardiomyoplasty was ensuring that the two muscles contracted synchronously. Initially, separate cardiomyostimulators were used for each muscle. This often resulted in alternate contraction of one muscle and then the other. A bifurcated sensing lead connected to two stimulators was tested, but this also resulted in alternate contraction of the muscles. A bifurcated muscular pacing lead attached to a single cardiomyostimulator solved the problem and allowed reliable synchronous contraction of both muscles.

Table 3. Hemodynamic Measurements Before and After Left and Bilateral Cardiomyoplasty With No Muscle Stimulation[a]

	Left		Bilateral	
Variable	Pre	Post	Pre	Post
RA (mm Hg)	6.5 ± 1.3	9.3 ± 1.7	6.9 ± 1.2	10.2 ± 1.5
RV (mm Hg)	32 ± 1.9	29 ± 2.2[b]	31.4 ± 2.0	27.3 ± 1.6[b]
RV dP/dt (mm Hg/s)	432 ± 69	285 ± 69[b]	412 ± 37	224 ± 25[b]
PA (mm Hg)	26 ± 1.9	25 ± 2.3	26 ± 2.2	24 ± 1.6
HR (beats/min)	115.3 ± 7.4	115.8 ± 5.4	114.3 ± 5.6	116.1 ± 4.7
LA (mm Hg)	10 ± 1.4	11.7 ± 1.4	9.5 ± 1.5	10.8 ± 1.5
LV (mm Hg)	105 ± 6.3	85 ± 6.5[b]	106 ± 5.7	77 ± 4.6[b]
LV dP/dt (mm Hg/s)	1949 ± 143	1458 ± 167[b]	1986 ± 102	1211 ± 270
Ao (mm Hg)	99 ± 3.7	82 ± 5.3[b]	99 ± 4.0	75 ± 4.4[b]
AF (L/min)	14.6 ± 0.6	10.1 ± 0.9[b]	14.7 ± 0.8	8.5 ± 0.7[b]

[a] Data are presented as mean ± standard error of the mean. [b] $p < 0.05$, pre versus post.

AF = peak aortic flow; Ao = systolic aortic pressure; dP/dt = the first derivative of pressure with respect to time; HR = heart rate; LA = mean left atrial pressure; LV = systolic left ventricular pressure; PA = systolic pulmonary artery pressure; RA = mean right atrial pressure; RV = systolic right ventricular pressure.

Table 4. Hemodynamic Effects With Stimulation of Left and Bilateral Cardiomyoplasty[a]

Variable	Left		Bilateral	
	Unstimulated	Stimulated	Unstimulated	Stimulated
RA (mm Hg)	9.3 ± 1.7	9.8 ± 1.5	10.2 ± 1.5	10.9 ± 1.4
RV (mm Hg)	27 ± 2.4	38 ± 2.6[b]	26 ± 1.8	44 ± 3.1[b]
RV dP/dt (mm Hg/s)	233 ± 16	435 ± 64[b]	196 ± 14	595 ± 117[b]
PA (mm Hg)	26 ± 2.2	32 ± 1.7[b]	23 ± 1.6	34 ± 1.9[b]
HR (beats/min)	115.8 ± 5.4	111.4 ± 6.1	116.1 ± 4.7	112.6 ± 5.5
LA (mm Hg)	11.7 ± 1.4	12.0 ± 1.3	10.8 ± 1.5	11.1 ± 1.4
LV (mm Hg)	83 ± 5.3	96 ± 4.4[b]	69 ± 5.3	90 ± 5.9[b]
LV dP/dt (mm Hg/s)	1291 ± 94	1413 ± 130	1072 ± 107	1454 ± 141[b]
Ao (mm Hg)	80 ± 3.7	86 ± 3.6[b]	67 ± 4.9	80 ± 5.4[b]
AF (L/min)	10 ± 0.7	11 ± 0.9[b]	7.7 ± 0.8	9.4 ± 1.1[b]

[a] Data are presented as mean ± standard error of the mean. [b] $p < 0.05$, unstimulated versus stimulated.

AF = peak aortic flow; Ao = systolic aortic pressure; dP/dt = the first derivative of pressure with respect to time; HR = heart rate; LA = mean left atrial pressure; LV = systolic left ventricular pressure; PA = systolic pulmonary artery pressure; RA = mean right atrial pressure; RV = systolic right ventricular pressure.

This report presents evidence that significant augmentation of right and left ventricular systolic function can be achieved with a unilateral left posterior or with a bilateral cardiomyoplasty. Increases in cardiac chamber pressures, dP/dt of ventricular pressure, and aortic flow occurred with each procedure. The bilateral cardiomyoplasty had a significantly greater effect on right ventricular function than the left cardiomyoplasty. Augmentation of left-sided hemodynamic variables was also consistently larger with the bilateral wrap, but the differences were only statistically significant for aortic pressure. However, the question of whether the bilateral procedure causes a larger effect on the left ventricle cannot be answered by this study, because the bilateral operation also resulted in a greater depression of baseline left ventricular variables.

Chronic studies will be necessary to determine if there are advantages of a bilateral cardiomyoplasty. The initial hemodynamic disturbance that occurs after wrapping the muscle is likely to be a temporary occurrence related to displacement of the heart from its normal position, which will not be a problem in long-term experiments.

Chronic studies would also resolve the question of whether equal hemodynamic effects can be achieved with a double muscle wrap that has undergone a training program. Muscle conditioning can lead to some loss in muscle mass and strength, but the degree of change might be a function of the specific protocol, rather than an inevitable consequence of stimulation. Recent work from this laboratory has shown that it is possible to condition skeletal muscle without a significant loss in size or power [14].

At this time, we do not plan to do bilateral cardiomyoplasty in humans. It is not certain that the bilateral procedure offers enough additional hemodynamic benefit to justify prolonging what is already a long operation. Another concern is whether both muscles would fit in the mediastinum of a human with cardiomegaly. There is ample room in the dog chest, but the human chest has a different configuration. A final concern is whether the disturbance in function seen in this model after construction of the double wrap would also occur in humans. In the dog much of the disturbance is related to displacement of the heart from its normal position. This is less likely to occur in humans, because the heart is more firmly rooted in the mediastinum in humans than dogs. However, any cardiac depression, even if it is temporary, would be detrimental in the clinical setting.

Despite these concerns, further investigation of bilateral cardiomyoplasty is warranted. The limitations of medical therapy, transplantation, and mechanical pumps are well known. There is a need for alternative approaches to the problem of heart failure. Our initial clinical experience with cardiomyoplasty indicates that patients with isolated left ventricular failure can be helped with the procedure, but that those with associated right ventricular failure have a high early and late mortality after the operation. Perhaps after additional research, a bilateral cardiomyoplasty could provide an approach to biventricular failure, as the major difference with the bilateral procedures is a greater effect on right ventricular function.

This work was supported in part by Public Health Service grant HL 38078 and the Allegheny-Singer Research Institute.

We are grateful to Dennis Trumble and Kathleen Simpson for their help in collecting and analyzing the data, to Leslie Arelt for running an efficient animal care facility, and to Dinean Samuel for preparing the manuscript.

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DISCUSSION

DR JONAH ODIM (Montreal, Que, Canada): Dr Ray C.-J. Chiu sends his regrets that he cannot be here today to discuss this report. We enjoyed reading the manuscript kindly provided by the authors regarding the use of both right and left latissimus dorsi muscle as a double wrap for cardiac assist.

Their findings support work done by Dr Larry Stephenson and his group using a similar double cardiomyoplasty technique. Laboratory studies using predominantly unilateral or single latissimus dorsi muscle have led to clinical application of this particular surgical configuration in patients with end-stage heart failure.

This operation—pioneered by Dr Carpentier as dynamic cardiomyoplasty—is normally conducted in one stage and usually takes 4 to 6 hours to accomplish. Naturally, one of our concerns relates to the logistics of a bilateral (double-wrap) latissimus dorsi cardiomyoplasty in terms of the duration and conduct of this operation in a high-risk group of patients, some of whom may ultimately require cardiopulmonary bypass to safely accomplish this goal. Furthermore, we understand that your group currently performs the unilateral (single-wrap) latissimus dorsi cardiomyoplasty in two stages. Clinical application of this double-wrap technique may add further complexities to a multistage operative approach. For starters, how would one position the patient on the operating table?

The preoperative chest roentgenogram of 1 of our recent patients demonstrated the bulky heart of end-stage dilated cardiomyopathy. The left ventricular ejection fraction was 0.08 to 0.09. This man was turned down for cardiac transplantation south of the border because of age. Four months after dynamic cardiomyoplasty, his exercise tolerance had improved quite dramatically. In fact, he has recently resumed his love of tennis. This patient is unique in our series of cardiomyoplasty. Despite his huge heart, his body habitus permitted a complete 360-degree biventricular muscular wrap using the left latissimus dorsi muscle alone.

An important concept that is emerging regarding the salutory effects of cardiomyoplasty may rest with complete muscular coverage of both ventricles rather than any added hemodynamic benefit from a double-wrap technique per se. In this regard, patient selection becomes important.

It may not be valid to compare the efficacy of this double-wrap technique with historical controls using a single muscle wrap. Technology and techniques in this field have advanced rapidly. The methods and orientation of muscle wrapping, stimulating parameters, and cardiomyostimulators are constantly improving.

I have only one question of Dr Magovern. Did you compare your present results with bilateral latissimus dorsi cardiomyoplasty with a control group of animals undergoing unilateral cardiomyoplasty (and complete biventricular muscular coverage) under identical experimental conditions with your current Prometheus cardiomyostimulator?

I commend Dr Magovern and his associates for this contribution to the continuing evolution of a new surgical operation designed to assist the management of patients with intractable heart failure.

DR MAGOVERN: We agree that a double muscle flap clearly doubles the complexity of the operation. Whether this will evolve into a clinical reality is not yet clear. At this point the operation is a laboratory phenomenon.

I think if you compare the logistics of doing a double muscle wrap to doing a cardiac transplantation, a transplantation is more complicated because it involves two teams and flying around the country in a jet. If one did get into doing this in patients, it would be best to do it in two stages. It would be an awfully long operation to do in one day.

In terms of comparing a double muscle wrap with a control group, when we compared these data with historical controls that we had done in the laboratory, these effects were much more marked than anything we had seen before. We have compared them recently with single flaps using the left muscle. We have been able to show similar changes with just one flap, but the magnitude of the change has been more with the double flap than with the single flap.

Results of the Fontan Procedure for Patients With Univentricular Heart

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One hundred twenty-four consecutive patients with univentricular heart undergoing the Fontan operation were reviewed. Patients with tricuspid atresia or biventricular heart with hypoplasia of one ventricle were excluded. Eighty-four patients had left ventricular morphology. Atrioventricular connection was double-inlet (n = 76), common (n = 29), absent left atrioventricular connection (n = 14), and absent right atrioventricular connection (n = 5). Actuarial survival was 77% (70% confidence limits, 73% to 81%) at 1 year, 66% (70% confidence limits, 60% to 72%) at 5 years, and 49% (70% confidence limits, 36% to 61%) at 10 years, indicating a continuing risk for premature death. Multivariate analysis identified preoperative ventricular function and hypertrophy as risk factors for survival. High postrepair right atrial pressure (>15 mm Hg) emerged as a strong intraoperative predictor of survival. Logistic regression analysis of these factors predicts high probability of death for certain subgroups of patients after the Fontan operation. Forty-four percent (n = 53) of these original 124 patients are alive and in New York Heart Association class I at follow-up. Thirty-eight percent (n = 33) of survivors have worse ventricular function than preoperative. Long-term survival is disappointing. Certain identifiable subgroups of patients with univentricular heart have unacceptable risks for the Fontan operation and should have alternate management. High postrepair right atrial pressure is an ominous sign, and if it persists the Fontan should be fenestrated or taken down.

(Ann Thorac Surg 1991;52:1266–71)

Since Fontan and Bandet [1] and Kreutzer and colleagues [2] independently described atriopulmonary connection for treatment of tricuspid atresia, the Fontan operation has been extended to other complex congenital anomalies. Survival rates are lower when the Fontan operation is used for univentricular heart lesions as compared with tricuspid atresia [3–6]. Late survival after Fontan operation for univentricular heart is not defined. Presently we manage most patients with univentricular heart with the expectation of eventually constructing a Fontan circulation. To determine factors that predict survival, we reviewed our entire experience with the Fontan procedure for univentricular heart.

Patients and Methods

From November 1978 to June 1989 there were 306 Fontan procedures done at The Hospital for Sick Children and The Toronto General Hospital. Of this group 124 were for univentricular heart. Specifically excluded from this group were patients with tricuspid atresia or biventricular hearts. Restrictive atrial septal defects—so-called fenestrated Fontan—were not made in any of these patients. The hospital and clinic records of these 124 patients were reviewed. Preoperative angiograms and postoperative echocardiograms were interpreted by blinded observers. Follow-up of the majority of patients was done at routine cardiology clinic. When a recent visit was not recorded, patients were asked to return for clinical evaluation. Follow-up was completed on 120 patients. Of the remaining patients, 1 had an uneventful recovery, returned to Spain, and was lost to follow-up. The others were followed up for 2, 4, and 6 years, respectively. Average follow-up was 6.3 years.

Seventy-five patients were male and 49 were female. Average age of patients at Fontan operation was 10.3 years (range, 1 to 36 years). Eighty-eight percent of patients (n = 54) were palliated before their Fontan operation. Forty-four percent of patients had a systemic to pulmonary artery shunt, 10% (n = 13) had a Glenn procedure, and 31% (n = 39) had pulmonary artery banding as their initial palliation. Forty-nine patients required a second palliative procedure and 9 required a third.

Cardiac morphology was analyzed and classified by previously published criteria [7]. Ventricular and atrioventricular valve morphology are detailed in Figure 1. Discordant ventriculoarterial connections were present in 63 patients, whereas only 13 patients had concordant connections. Double outlet was present in another 33 patients, with 30 of these being double-outlet right ventricle. Fifteen patients had pulmonary atresia.

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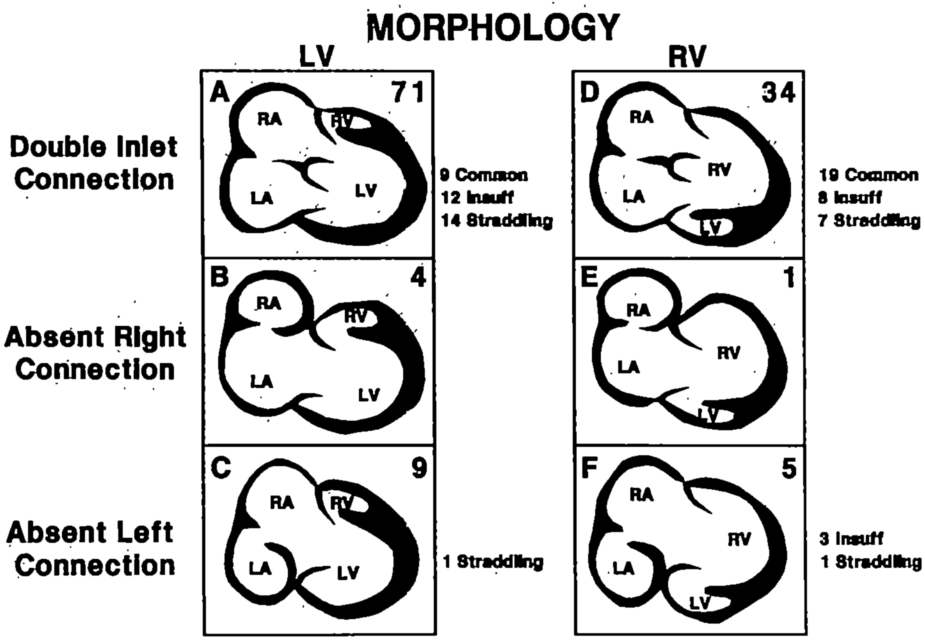

Fig 1. Eighty-four patients had a univentricular heart of left ventricular type. Forty patients had univentricular heart of right ventricular type. Double-inlet connection was present in 105 patients, 71 with univentricular heart of left ventricular type and 34 with univentricular heart of right ventricular type. Twenty-three patients had some atrioventricular valve insufficiency, 11 of whom had common atrioventricular valves as their mode of connection. (Insuff = *insufficiency;* LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

Associated lesions were documented in each patient. Particular attention was directed to diagnosing and grading subaortic stenosis, pulmonary artery stenosis, and atrioventricular valve incompetence. Subaortic stenosis was identified in 40 patients. It was diagnosed by angiography and defined as the outlet ventricular septal defect or immediate subaortic area smaller than aortic annulus. It was treated surgically by direct ventricular septal defect enlargement in 16 patients and by Damus-Kaye-Stansel operation in 15. Most patients were treated for their subaortic stenosis at the time of the Fontan operation (n = 18), but 6 were treated before and 6 subsequent to the Fontan operation.

Pulmonary arteries were measured at their maximal diameter and at sites of maximal stenosis. Atrioventricular valve incompetence was diagnosed in 23 patients and graded as mild, moderate, or severe by echocardiogram. Atrioventricular valves were managed by repair or exclusion at time of Fontan operation.

Preoperative ventricular function and hypertrophy were evaluated and graded by angiograms and echocardiograms. Ventricular function was graded as normal, moderately depressed, or severely depressed. Ventricular hypertrophy was graded as none, mild, moderate, or severe. The grade of ventricular hypertrophy could be determined in 119 patients. Postoperative ventricular function was determined by echocardiogram. It was compared with the preoperative echocardiogram and judged as to whether the function was the same, better, or worse.

Postrepair atrial pressures are mean pressures taken in the operating room after cardiopulmonary bypass was discontinued. Each surviving patient's late functional status was determined from the most recent follow-up visit.

Overall survival analysis was performed by nonparametric methods using the Cox proportional hazards method. Univariate analysis identified possible risk factors, and stepwise multivariate analysis then determined significant and probably significant variables. Predicted probability of death was determined by logistic regression analysis of these variables.

Results

Overall Survival

Twenty-five patients died in the hospital and 10 died during follow-up. Most patients died of acute or chronic cardiac failure. Actuarial survival (Fig 2) at 5 years and 10 years respectively was 66% (70% confidence limits [CL], 60% to 72%) and 48% (70% CL, 36% to 61%). Multivariate

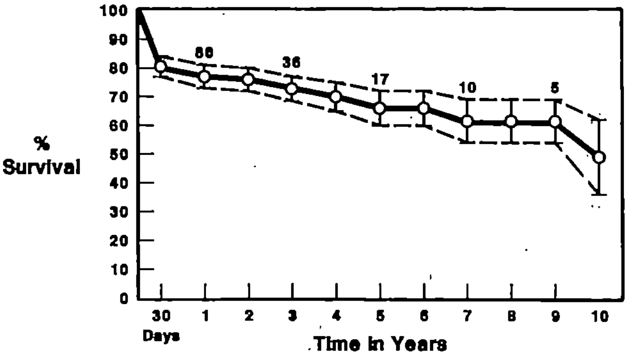

Fig 2. One-year actuarial survival after Fontan operation for univentricular heart is 77% (70% confidence limits, 73% to 81%), 5-year survival is 66% (70% confidence limits, 60% to 72%), and 10-year survival is 49% (70% CL, 36% to 61%). The numbers indicate the number of patients at risk entering each time period.

analysis demonstrated the following variables to be predictive of survival:

- Significant ($p < 0.05$)
  - Preoperative ventricular function
  - Preoperative ventricular hypertrophy
  - Postrepair right atrial pressure
- Probably significant ($0.05 < p < 0.10$)
  - Cardiopulmonary bypass time
  - Pulmonary artery band

Ventricular Function

Preoperative ventricular function emerged from multivariate analysis as a significant predictor of survival. Only 14 patients had moderately depressed ventricular function and no patient had severely depressed function. Of 14 patients with depressed function only 6 are long-term survivors. Actuarial survival of patients with normal preoperative ventricular function is compared with those with depressed function in Figure 3.

Ventricular Hypertrophy

The second preoperative factor identified by multivariate analysis to predict survival is ventricular hypertrophy. Fifty-nine patients had some degree of hypertrophy. Eight of 32 patients with mild hypertrophy died whereas 11 of 24 patients with moderate hypertrophy died. No patient with severe ventricular hypertrophy survived (n = 3). Comparison of the actuarial survival for patients with no or mild hypertrophy with patients with moderate or severe hypertrophy is dramatic (Fig 4). Analysis of 40 patients with subaortic stenosis and 40 patients with pulmonary artery banding indicates that if they are associated with ventricular hypertrophy, survival is decreased. If, however, these factors are present without ventricular hypertrophy, survival is not different (Table 1).

Postrepair Right Atrial Pressure

High postrepair right atrial pressure (RAP) is associated with decreased survival. As RAP increases, the probability of survival decreases exponentially. Logistic regression analysis demonstrates a significant relationship between postrepair RAP, preoperation ventricular function, and ventricular hypertrophy (Fig 5). Predicted probability of death for any measured postrepair RAP is greater when depressed function or moderate to severe ventricular hypertrophy is present.

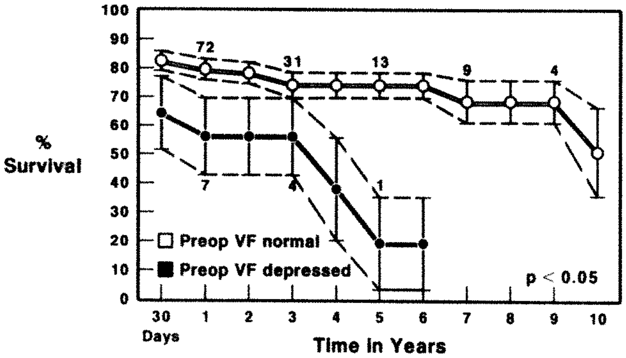

Fig 3. Actuarial survival for patients with moderately depressed ventricular function (VF) versus patients with normal ventricular function. Observed survival is significantly different. Survival for the 14 patients with preoperative depressed ventricular function is 19% (70% confidence limits, 3% to 24%) at 5 years.

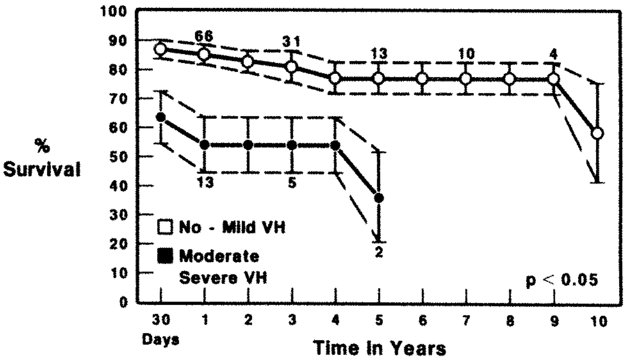

Fig 4. Actuarial survival comparing patients with no or mild ventricular hypertrophy (VH) to patients with moderate or severe VH. Survival at 5 years is 77% (70% confidence limits, 71% to 83%) for patients with no or mild VH and 36% (70% confidence limits, 20% to 52%) for patients with moderate or severe VH.

Late Results

Sixty-one percent (n = 53) of surviving patients are in New York Heart Association (NYHA) functional class I at latest follow-up. Twenty-eight percent (n = 24) are in NYHA class II and 11% (n = 10), NYHA class III. Overall, 61% of the surviving patients have normal ventricular function compared with 87% before the Fontan procedure. Thirty-nine percent of survivors have mild (n = 18), moderate (n = 13), or severe (n = 3) depression of ventricular function. Even among those 53 patients in NYHA class I at most recent follow-up, only 70% have normal ventricular function whereas 30% have reduced (mild, 10; moderate, 6) ventricular function. Thirty-eight percent (n = 33) of the surviving patients have worse

Table 1. Mortality of Subaortic Stenosis and Pulmonary Artery Banding Appear Related to the Degree of Ventricular Hypertrophy They Produce

	SAS			PAB		
Hypertrophy	N	M	%M	N	M	%M
None	21	2	9.5	14	4	28
Mild	8	2	25	11	3	27
Moderate	10	5	50	12	6	50
Severe	1	1	100	2	2	100

M = mortality; PAB = pulmonary artery banding; SAS = subaortic stenosis.

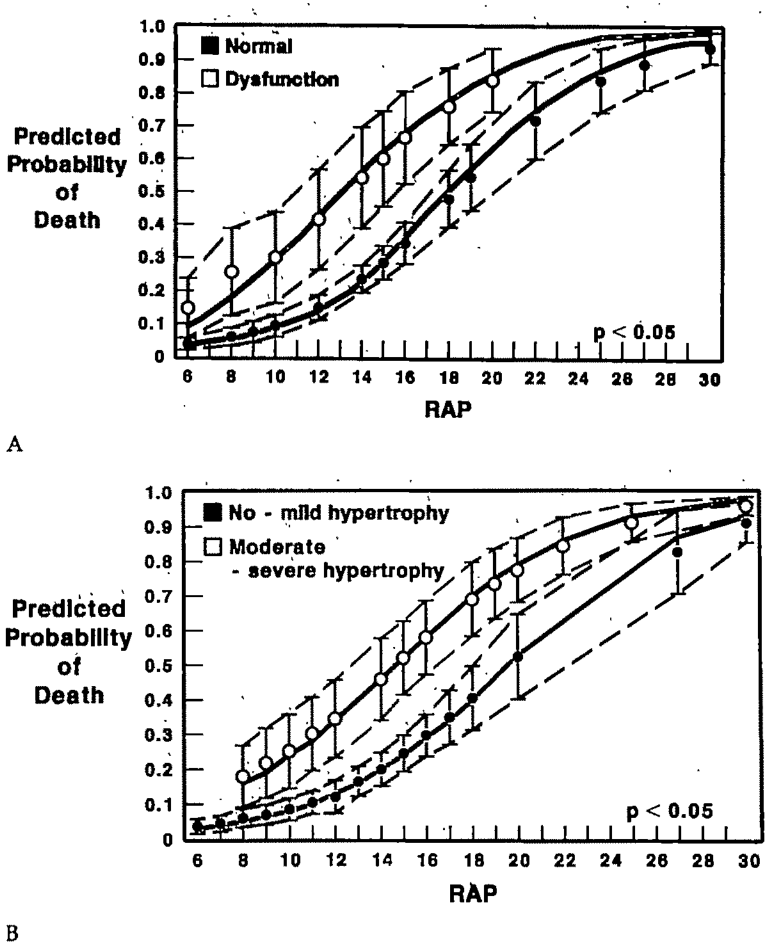

Fig 5. (A) Logistic regression analysis of ventricular function (normal versus dysfunction) and postrepair right atrial pressure (RAP) predicts mortality of 23% (70% confidence limits, 15% to 33%) for patients with normal ventricular function and a postrepair RAP of 14 mm Hg. A patient with moderately depressed ventricular function has a predicted mortality of 54% (70% confidence limits, 26% to 81%) with a postrepair RAP of 14 mm Hg. (B) Logistic regression analysis of ventricular hypertrophy (no or mild versus moderate or severe) and a postrepair RAP of 14 mm Hg and no or mild ventricular hypertrophy is 20% (70% confidence limits, 15% to 25%). For a patient with moderate or severe ventricular hypertrophy and a postrepair RAP of 14 mm Hg, predicted mortality is 46% (70% confidence limits, 34% to 58%).

ventricular function at the date of most recent follow-up compared with their preoperative echocardiogram.

Comment

The anticipated benefits of the Fontan procedure for univentricular heart are prolonged survival and improved quality of life. By the establishment of a circulation in series, ventricular demand is normalized; one assumes that ventricular function will be preserved and the patient will experience improved long-term functional capacity and survival.

Actuarial survival of 66% at 5 years (70% CL, 60% to 72%) and 49% at 10 years (70% CL, 36% to 61%) is similar to that of other large series [5–8]. The risk of death in this series is high during the early postoperative period and stabilizes in the intermediate period, but as follow-up lengthens there appears to be an acceleration of risk. This observation is consistent with that of Fontan and associates [9], who noted appreciable late increased risk of death, which begins gradually about 6 years after operation. If these observations are substantiated, serious questions about the long-term palliative benefits of the Fontan operation arise.

The functional results of survivors are also of concern. Only 53 of the original 124 patients in this study are asymptomatic at follow-up, and even in these patients 30% have mild to moderate depression of ventricular function by postoperative echocardiogram. Overall, 38% of survivors have worse ventricular function than before operation. These observations suggest that the Fontan operation is not achieving long-term preservation of ventricular function. Fontan and associates [9] have reported similar findings and pointed out that the longer the interval between operation and follow-up examination, the smaller the proportion of patients who were in NYHA functional class I.

To improve results with the Fontan operation, better patient selection is required. Ventricular function and hypertrophy should be evaluated and graded for each patient. Of 14 patients with moderately depressed ventricular function in this study, 8 died during the follow-up period of 6 years (actuarial 5 year survival, 19%; 70% CL, 3% to 34%). For this group other forms of palliation may be more appropriate, with the expectation of later cardiac transplantation when indicated.

The decision about patients with ventricular hypertrophy is more difficult. In spite of the fact that our methods for determining the grade of ventricular hypertrophy are crude, the grade of hypertrophy accurately predicted survival. These findings agree with the analysis of Kirklin and co-workers [5] for all their patients undergoing the Fontan procedure. If ventricular hypertrophy is mild, it is probably reasonable to proceed with the Fontan operation. In this study, 8 of 32 patients with mild hypertrophy died at operation or during follow-up (mortality, 25%). However, if ventricular hypertrophy is moderate or severe the Fontan risk is high and should be abandoned. Of 27 patients with moderate or severe hypertrophy, 14 died in the hospital or during follow-up. Patients with pulmonary artery banding or subaortic stenosis who have moderate to severe ventricular hypertrophy respond to pulmonary artery debanding or subaortic stenosis resection in a separate operation. If ventricular hypertrophy regresses, they can be reconsidered for a subsequent Fontan operation. In this study, 6 patients had subaortic stenosis resected before Fontan procedure, with 5 long-term survivors.

Improved intraoperative and early postoperative decision making may also increase overall survival. This study and others demonstrate that high postrepair RAP is a strong predictor of death [4, 5, 10]. With normal ventricular function and no ventricular hypertrophy there is rapid acceleration of risk when postrepair RAP is greater than 15 mm Hg. When high RAP persists in the early postoperative period, serious consideration should be given to taking down the Fontan. Another approach we have used recently (not in any patient in this series) is to fenestrate the Fontan as described by Laks and associates [11]. Later the patient can undergo transcatheter closure of this residual atrial septal defect [12]. The deleterious effect of high postrepair RAP on survival is much more dramatic in patients with decreased ventricular function or in the presence of ventricular hypertrophy. We would consider Fontan takedown or baffle fenestration at a somewhat lower postrepair RAP in these situations.

In summary, our data do not support expanding the indications for the Fontan operation in univentricular heart to poor-risk patients. Patients with depressed preoperative ventricular function or more than mild ventricular hypertrophy have unacceptable survival after Fontan operation, and alternate forms of management, such as bidirectional Glenn shunt and cardiac transplantation, should be considered. In preparing patients for Fontan operation, factors that result in myocardial hypertrophy, such as pulmonary artery banding and subaortic stenosis, should be identified and alleviated. High postrepair RAP is an ominous sign, and early takedown or baffle fenestration should be considered.

We thank Henriette Sherret, PhD, and Robyne Campbell, BA, for their efforts during the preparation of this report.

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DISCUSSION

DR GORDON K. DANIELSON (Rochester, MN): I thank The Society for the invitation to discuss this well-presented and challenging paper. Dr Cohen and associates have addressed an important issue: "Does the modified Fontan procedure provide improved palliation and increased longevity for patients with complex congenital heart disease?" or, phrased another way, "Does the dismal natural history of univentricular heart continue unabated even though the ventricular volume overload has been corrected and the blood made fully saturated by the modified Fontan procedure?"

Our results are somewhat at variance with those quoted in the abstract and lead us to be more optimistic with what can be accomplished by this operation. We calculated the Kaplan-Meier survival of 352 patients who underwent operation more than 6 years ago, between 1973 and 1984. When 30-day operative mortality is included, the 5-year survival is 70% and the 10-year survival is 60%. When operative survivors only are included, the 5-year survival is 80% and the 10-year survival is 70%. When this latter curve is extended to 15 years, for the 38 patients in that group, survival remains 70%.

When one considers the 30-day operative survivors, the survivors at 10 years in the double-inlet group and the more complex group, which includes polysplenia and asplenia patients, are similar and only slightly lower than survival of the tricuspid atresia group. As evaluated by our cardiologists, 80% of patients are in class I or II, and 50% are receiving no cardiac medications.

Only 13% of the patients met all of Choussat and Fontan's ten criteria. When correlated with the six most important risk factors in our group, survival is clearly related to the number of risk factors present in a given patient. We agree with Cohen and associates that for patients in this last category (24% 10-year survival), at least, alternative options such as heart or heart-lung transplantation should be considered.

I have a question. In view of the currently improved understanding of which patients are candidates for operation and the improved operative mortality (less than 3% to less than 10% for all diagnostic categories), do you believe that the 10-year results of patients now undergoing operation will not be significantly improved compared with these earliest results?

DR WILLIAM G. WILLIAMS (Toronto, Ont, Canada): I would like to apologize to the Chairman and perhaps to the audience as well for the confusion in this presentation. Dr Amram Cohen did the major ground work for this paper, and shortly after we submitted the abstract he was called to active service and is now stationed in Saudi Arabia. In his absence, Dr Cleveland has been instrumental in finishing this work, and I am grateful for his effort and this thoughtful input into the paper. I thank Dr Guyton of the Program Committee for allowing Dr Cleveland to present this paper.

In response to Dr Danielson's comments, and specifically his question about today's selection of candidates for the Fontan operation, our data show that the traditional risk factors for the Fontan operation exert their deleterious effects through the mechanism of ventricular hypertrophy and ventricular dysfunction. Specifically, my response to selection of today's candidates would be to eliminate those patients with more than mild depression of ventricular function.

Will today's results be better? I think they will for three reasons. The first reason is improved timing in the selection of patients. This would include earlier intervention in patients prone to ventricular hypertrophy and ventricular dysfunction. The second reason is improved selection of patients by staging those patients with ventricular hypertrophy. We recommend, in patients with moderate or severe ventricular hypertrophy, that the subaortic stenosis be dealt with as a separate procedure, perhaps in conjunction with a bidirectional Glenn shunt. After an interval, their suitability for a Fontan operation may be reconsidered. Third, none of the patients in this series had the so-called fenestrated Fontan. By deliberately creating a restrictive atrial septal defect as part of the Fontan operation, the preloading of the systemic ventricle is improved. The postoperative hemodynamics after a fenestrated Fontan are more stable, and there is a decrease in postoperative mortality and morbidity. The late impact of the fenestrated Fontan on ventricular function is as yet unknown, but I anticipate that it would have a beneficial effect.

Main Bronchial Sleeve Resection With Pulmonary Conservation

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Twenty-seven main bronchial resections (19 left, 8 right) were performed without pulmonary resection between 1975 and 1991. The patients were 17 men and 9 women with an average age of 35 years (range, 20 to 65 years). Tumors comprised 55% of the lesions, including 9 carcinoid tumors (33%), 2 mucoepidermoid tumors, 2 fibrous histiocytomas, 1 hemangiopericytoma, and 1 large cell carcinoma. Scarring and stenosis secondary to multiple causes occurred in 10 patients (37%). Two patients had miscellaneous lesions. Presenting symptoms included dyspnea (52%), wheezing or stridor (44%), cough (41%), hemoptysis (37%), and pneumonia (18%). Preoperative chest roentgenogram was abnormal in 60% of patients, whereas tomograms delineated the lesion in 94%. All patients had bronchoscopy for lesion evaluation. Anesthesia was accomplished through a long single-lumen endotracheal tube in 19 cases and a double-lumen tube in 8 cases. Mobilization and exposure techniques to create a tension-free anastomosis were critical for left main bronchial resections and included pretracheal mobilization (100%), neck flexion (100%), tracheal and main bronchial retraction (85%), aortic and pulmonary artery retraction (44%), and intrapericardial hilar release (33%). All resections were for cure; there was no operative mortality. Morbidity in 4 patients (15%) included an anastomotic stenosis (successfully reresected), prolonged air leak and pneumonia, transient recurrent nerve palsy, and atelectasis. Median 5-year follow-up revealed 92% of patients alive, with only one of two late deaths being disease-related. Main bronchial resection is an ideal technique for selected benign and malignant lesions, allowing complete pulmonary parenchymal preservation.

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Tracheal, carinal, and pulmonary sleeve resections have been described in various reports [1–3], but isolated main bronchial lesions are unusual, and scant information is available about their clinical course. Over the past 16 years, 26 patients at the Massachusetts General Hospital underwent 27 main bronchial resections with complete pulmonary preservation. The presenting signs and symptoms, preoperative evaluation, resectional technique, pathology, perioperative management, and follow-up are presented. The specific operative techniques that allow main bronchial resection and avoidance of pneumonectomy are described in detail. Furthermore, pathologic comparison is made with lesions in other segments of the airway.

Material and Methods

Patients and Pathology

Twenty-six patients comprise this 16-year series (1975 to 1991) of 27 main bronchial resections. Seventeen patients were male and 9 female. Data were collected from hospital and office records with follow-up data obtained from questionnaires or by direct contact.

Pathology is shown in Table 1. Tumors represented the single largest category of main bronchial lesions (55%). Nine patients (33%; 7 men and 2 women) had typical carcinoid tumors. Six were in the left main bronchus and three were in the right main bronchus. No patient had carcinoid syndrome. Two patients (7.5%) had left main bronchial mucoepidermoid tumors. Fibrous histiocytoma was present in 2 patients (7.5%)—one right and one left. One patient had a left main bronchial hemangiopericytoma. An additional patient had biopsy performed on a discrete right main bronchial nodule at the time of right upper lobe sleeve resection for a peripheral large cell carcinoma. Despite negative intraoperative frozen section, the nodule proved to be a separate focus of large cell carcinoma on permanent pathological review. One month after lobectomy the patient underwent right main bronchial resection. All tumors resected had negative histological margins and negative lymph nodes.

Ten lesions (37%) were benign stenoses of varying cause and occurred in 5 male and 5 female patients. Stenosis developed in 4 of these patients after a previous chest operation. Two patients had undergone right upper lobectomy for carcinoma and 1 patient, left lower lobectomy for carcinoid before referral. Main bronchial stenosis developed in these patients at the site of closure of the lobar bronchus. Another patient in this series was operated on twice owing to anastomotic distraction and stenosis after resection of a left main bronchial mucoepidermoid tumor. Five months after initial resection, her stenosis was repaired, and she remains well 6 years later.

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Table 1. Pathology of Main Bronchial Lesions

Pathology	Left	Right
Tumors (55%)		
Carcinoid (33%)	6	3
Mucoepidermoid	2	0
Fibrous histiocytoma	1	1
Hemangiopericytoma	1	0
Large cell carcinoma	0	1
Stenosis (37%)		
Postoperative (15%)	2	2
Posttraumatic rupture	1	1
Secondary to laser use	1	0
Idiopathic	2	0
Tuberculosis	1	0
Other (8%)		
Inflammatory pseudotumor	1	0
Lymphoid aggregate	1	0
Total	19	8

Stenosis developed in 2 patients after blunt traumatic rupture of the bronchus. One was seen 4 months after rupture of the right main bronchus without having undergone definitive initial treatment, and one 8 months after failed primary repair of the left main bronchus. Left main bronchial stenosis developed in another patient after ill-advised multiple photodynamic laser treatments for bronchial metaplasia found on bronchoscopy done to evaluate a chronic cough. After resection, the specimen showed chronic scarring and lymph nodes consistent with sarcoid. Two patients with benign stenosis were women, both of whom had histories of asthma and bronchitis since childhood and were found subsequently to have discrete fibrotic left main bronchial lesions. Although both recovered well initially after left main bronchial resections, additional fibrotic stenoses later appeared in the trachea and right main bronchus. These 2 patients were categorized as having idiopathic stenosis, as no other cause was identified (ie, mediastinal fibrosis, histiocytosis, tuberculosis or other infection, specific inflammatory disease, trauma, chemical or thermal exposure). One final patient with severe left main stenosis secondary to tuberculosis underwent total left main resection.

Two other patients had miscellaneous pathology. One patient had a lymphoid aggregate (endobronchial lymph node) in the left main bronchus. Another patient had an inflammatory pseudotumor.

Operative Technique

Initially, bronchoscopy (rigid or flexible) was performed to precisely identify the location and extent of the lesion. A biopsy with frozen section examination was performed when it appeared the operative strategy would be affected by the results. Biopsy was avoided in vascular-appearing carcinoid tumors. Occasionally, in patients with obstructive pneumonia it was necessary to suction away secretions in the distal airway.

After bronchoscopy, continued intraoperative anesthesia was provided by reintubating the patient with either a Wilson tube into the opposite bronchus (19 patients) or a double-lumen tube (8 patients). The Wilson tube (Fig 1) is a long, flexible, single-lumen tube constructed by attaching an extension onto a flexible armored tube. There is also an extender on the balloon cuff inflation port. The end is square, not beveled, for better fit into the main bronchi, and the balloon cuff is smaller to minimize herniation. The tube is advanced into the opposite bronchus before thoracotomy. Tube position is verified by examination or by use of a flexible pediatric bronchoscope. When double-lumen tubes were used, right double-lumen tubes were placed for left main lesions (5 patients) and left double-lumen tubes were placed for right main lesions (3 patients). The relative rigidity of the double-lumen tube compared with the Wilson tube can

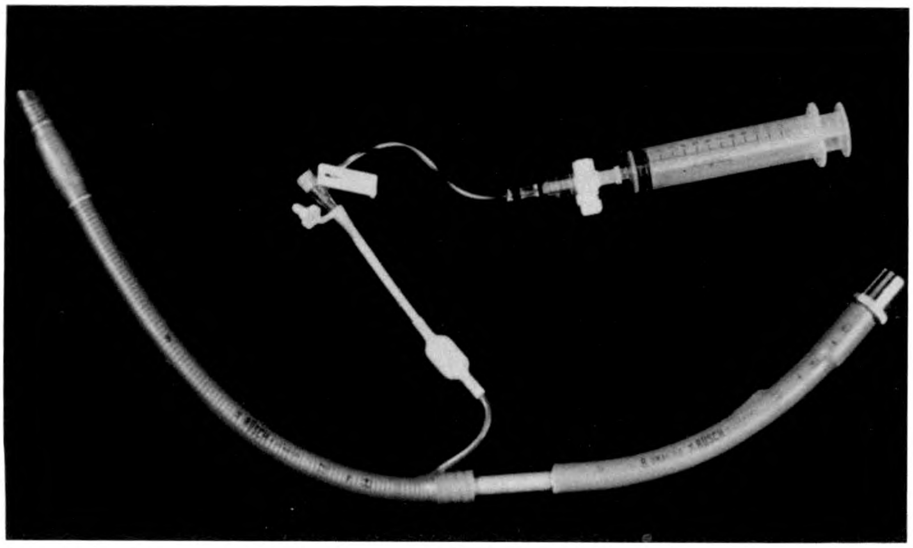

Fig 1. The Wilson tube constructed by attaching an extension tube onto a square-ended, flexible armored tube.

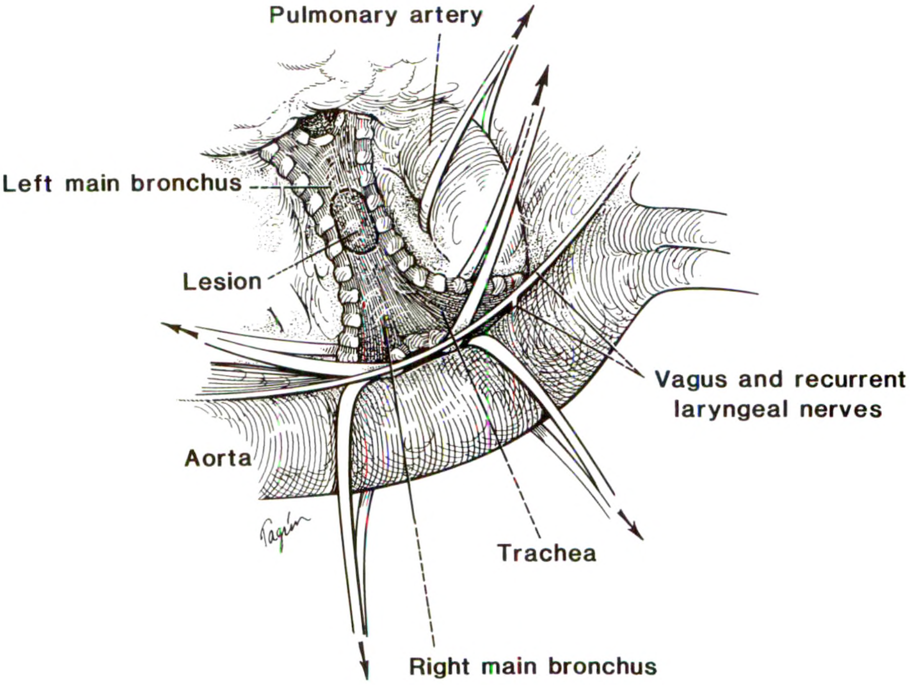

Fig 2. Exposure for left main bronchial resection. Note the tracheal, right main bronchial, pulmonary artery, and aortic retraction that provides improved exposure.

limit mobility of the carina and is a disadvantage of the double-lumen tube in extensive left main bronchial resection.

The operative approach was through the bed of the resected fifth rib or in the fourth interspace on the appropriate side. Intraoperative exploration revealed lobar collapse in 3 patients and hyperinflation in 1. The lesion could usually be palpated, and nodes were occasionally enlarged by inflammation. After assessment of resectability, mobilization techniques were initiated and varied with extent of resection and whether right or left main bronchial resection was done. The inferior pulmonary ligament was freed in all cases. For right main lesions, tension was not a problem during reconstruction, given the short length of the right bronchus; therefore, additional mobilization and exposure techniques were not required. For left main bronchial resections, the length of the left main bronchus resected, the prominent aortic arch, and left heart border made the use of tension-reducing maneuvers important. Pretracheal mobilization from the carina cephalad was initiated by sharp dissection and extended by blunt finger dissection. This dissection can be done in patients after prior mediastinoscopy although less mobility may be gained. Temporary neck flexion was used in all left-sided cases after resection to facilitate approximation of the anastomosis. A guardian suture from the underside of the chin to the premanubrial skin was placed after the thoracotomy was closed but before extubation to maintain the neck in 45-degree flexion for 7 postoperative days only when extensive left main resection was carried out. Both pretracheal dissection and neck flexion provide added proximal main bronchial length by allowing the trachea, carina, and proximal main bronchus to descend.

In most patients (85%) before resection, traction tapes (6.4-mm [¼-inch] Penrose drains) were placed encircling the trachea for right main bronchial resections and encircling the trachea and right main bronchus for left main bronchial resections. Tracheal and bronchial traction is a key exposure technique in the successful completion of the operation. Additional dissection to retract the aortic arch (without division of intercostal vessels) and pulmonary artery was required for exposure in 8 of the 19 left main bronchial resections (44%). Smith and Nigam [4] have previously described aortic mobilization with intercostal vessel division to expose the left main bronchus. An example of the extent of exposure obtained by careful dissection for left main resection is shown in Figure 2. The trachea and right main bronchus are pulled up into the field by the traction tapes to expose the origin of the left main bronchus. The recurrent laryngeal nerve must be carefully identified and preserved for left main bronchial resections.

If needed, added distal airway mobility can be obtained by the intrapericardial hilar release technique, which allows the distal end of the bronchus and hilum to ascend approximately 1.0 to 2.0 cm. In 6 of the 19 patients undergoing left main bronchial resection (33%), intrapericardial hilar release was performed to assist in providing a tension-free anastomosis. It was especially helpful for proximal, subtotal, or total left main resections. Hilar release (Fig 3) involved a U-shaped incision in the pericardium beneath the inferior pulmonary vein to allow upward movement of the hilum. The pericardial incision may be extended superiorly or completely circumscribe the hilar vessels if additional mobility is required for reconstruction, but this is rarely needed. If the extended pericardial incision is used, the subvenous frenulum

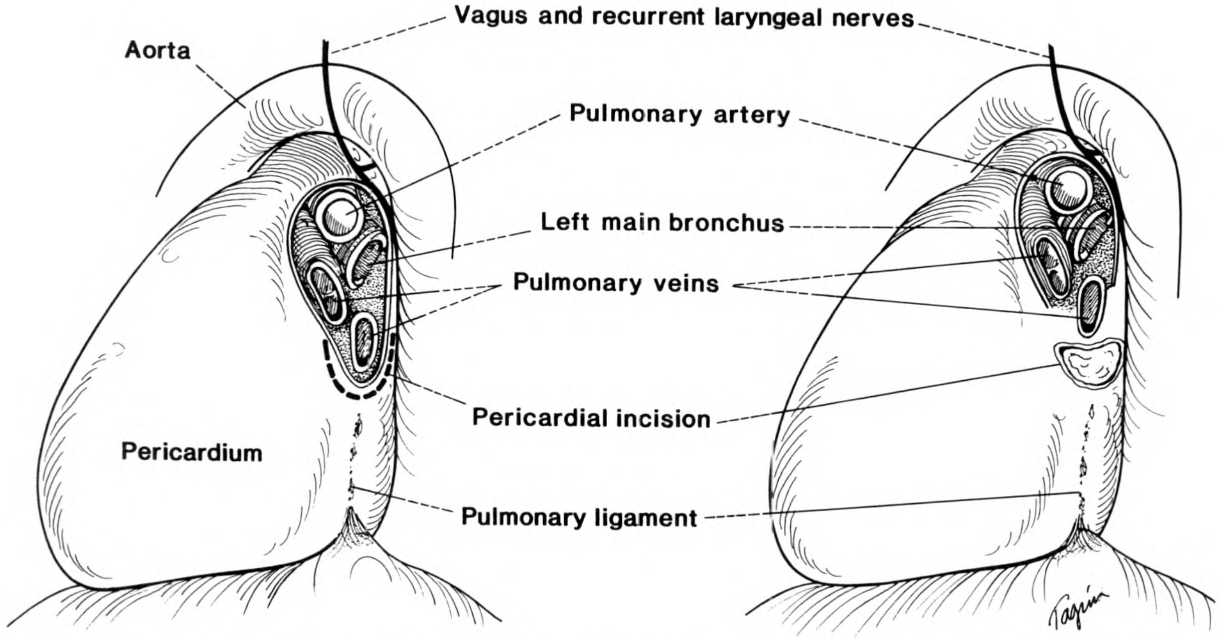

Fig 3. The left side intrapericardial hilar release technique, showing the U *shaped pericardial incision allowing 1 to 2 cm of upward hilar mobility to facilitate the creation of a tension-free anastomosis.*

should be divided and the posterior pedicle of lymph nodes preserved to avoid lymphatic obstruction in the early postoperative days.

Resection was performed by circumferential complete division of the airway proximal and distal to the lesion. A typical pathology specimen is shown in Figure 4. Resection margins were negative by frozen section for malignant lesions and at grossly normal mucosa for benign stenoses. Routine airway anastomosis reconstruction was carried out as described elsewhere [5] using two lateral traction sutures (2-0 Vicryl; Ethicon, Somerville, NJ) for orientation control and assistance in creating a tension-free anastomosis. Interrupted sutures (4-0 Vicryl) placed 3 to 4 mm apart and 3 to 4 mm from the cut edge with knots tied outside the airway completed the reconstruction. All patients had their anastomosis tested for an airtight seal. In 96% the anastomosis was buttressed with a flap of viable pleura or pericardial fat pad. Pulmonary artery kinking was not observed.

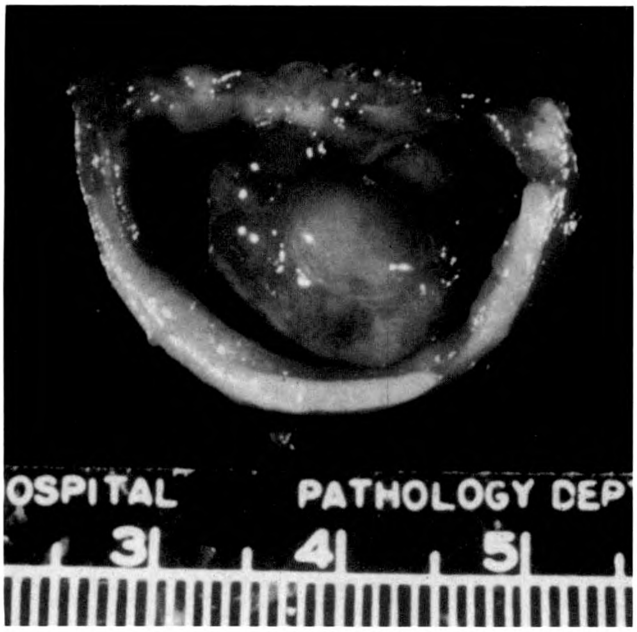

Fig 4. The pathology specimen of a right main bronchial carcinoid tumor. In this patient, only a short segment of the airway needed to be removed.

The average length of resected right main bronchus was 1.5 cm (range, 1.0 to 2.1 cm), and for the left main bronchus the average resected specimen was 2.8 cm (range, 1.0 to 4.0 cm). Right main bronchial resections tended to be the total length of the bronchus, as it is naturally short, whereas left main bronchial resections were more variable. The entire left main bronchus was removed in 3 patients (2 for benign stenosis [1 idiopathic, 1 postoperative] and 1 for stenosis secondary to tuberculosis). The intrapericardial hilar release was an especially critical tension-reducing maneuver during reconstruction in these patients. An additional patient with a carcinoid tumor had the carina between the left upper and lower lobes resected with anastomosis of the upper and lower lobe bronchi together before anastomosis to the proximal left main bronchus.

Perioperative management began with immediate extubation in the operating room and intensive care observation, usually for 24 hours and occasionally 48 hours. All patients received aggressive chest physiotherapy and perioperative antibiotics, and 4 patients required postoperative bronchoscopy for clearance of secretions. The

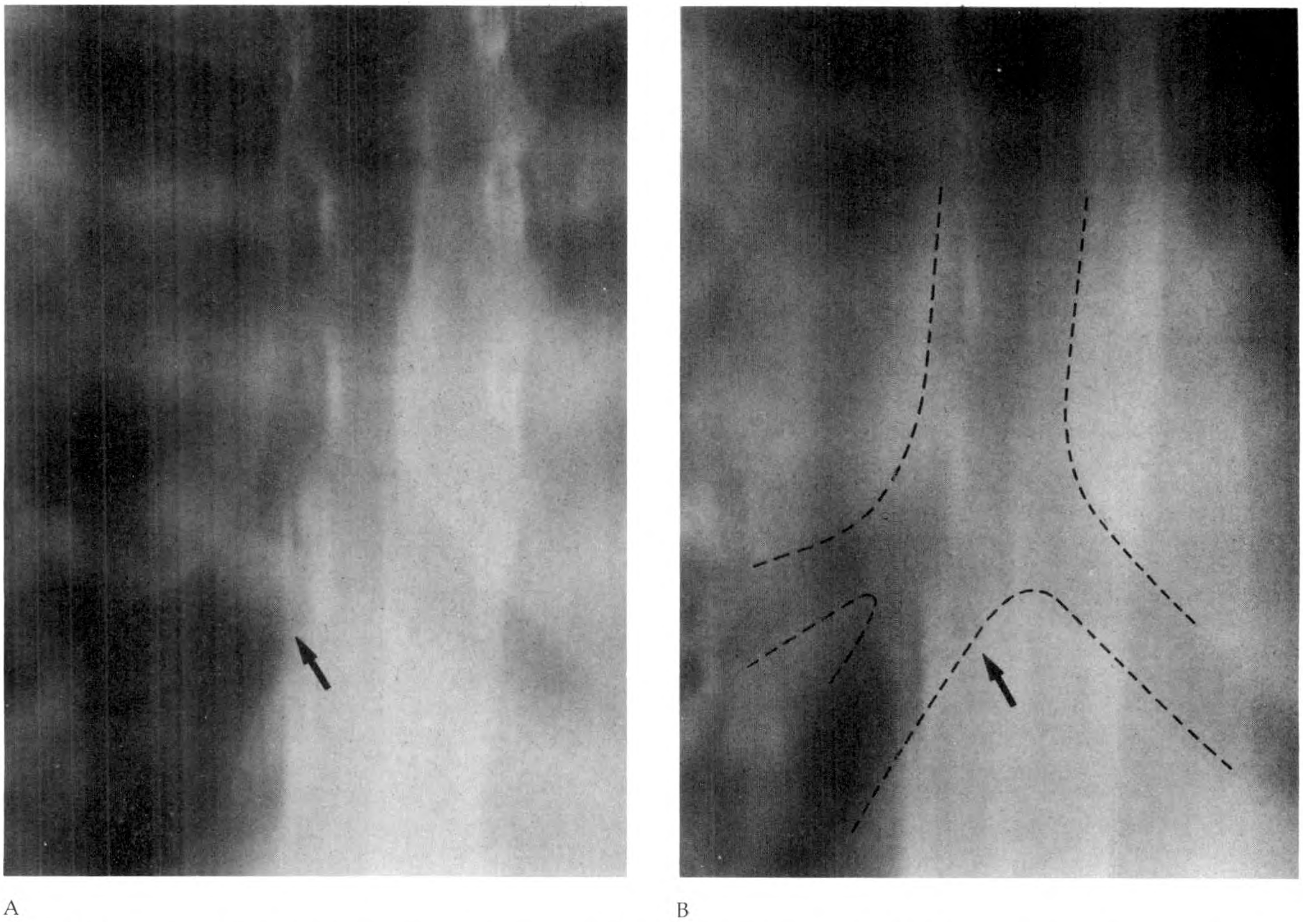

Fig 5. (A) Preoperative tracheobronchial tomograms demonstrating a right carcinoid tumor, identified by the arrow. *(B) Postoperative tracheobronchial tomograms demonstrating a patent airway with an* arrow *marking the anastomosis.*

average total hospital stay was 9 days (range, 6 to 36 days). Adjuvant therapy was not required for any patient.

Results

Symptoms and Preoperative Evaluation

All patients were symptomatic and had an average of 2.5 symptoms. The presenting signs and symptoms included dyspnea (52%), wheeze or stridor (44%), cough (41%), hemoptysis (37%), pneumonia (18%), fever (11%), and chest pain (7%). Left or right main bronchial location or pathology of the lesion produced no difference in symptoms. The average age at presentation was 35.0 years (range, 20 to 65 years), although generally patients with left main bronchial lesions were younger (33.1 years) than those with right-sided lesions (42.6 years). Associated medical problems were uncommon (23%) and included coronary artery disease in 2 patients (1 of whom also had diabetes and diverticulosis) and chronic obstructive pulmonary disease, hypertension, gout, and Crohn's disease in 1 patient each. Fifteen of the 26 patients (58%) had a history of smoking.

Preoperative evaluation included a chest roentgenogram in all patients, which was normal in 41%. A specific lesion was visible in the main bronchus in 18%, and secondary effects of atelectasis, pneumonia, or air trapping were noted in 41%. Standard tracheobronchial tomograms (Figs 5, 6) were especially useful in delineating the lesion in 16 of 17 patients (94%). Twelve patients had computed axial tomography before referral, detecting the lesion in 9 (Fig 7). One patient, before referral, had a magnetic resonance scan which detected the lesion. Pulmonary function tests revealed an average forced expiratory volume in 1 second of 2.5 L (range, 0.8 to 5.4 L). Ventilation and perfusion scanning usually showed decreased function on the affected side, although depending on the size of the lesion, values were occasionally near normal. No patient had bone or brain scans, pulmonary angiography, or mediastinoscopy. Nineteen patients had undergone bronchoscopy before referral, which occasionally established the correct diagnosis, and all patients underwent bronchoscopy immediately before thoracotomy. The respective bronchus was on average stenosed to approximately 80% of normal caliber by visual inspection, with 15 patients showing nearly total obstruction.

Despite this degree of obstruction, only 2 patients required preoperative therapeutic bronchoscopy to clear secretions of a postobstructive pneumonia. One patient

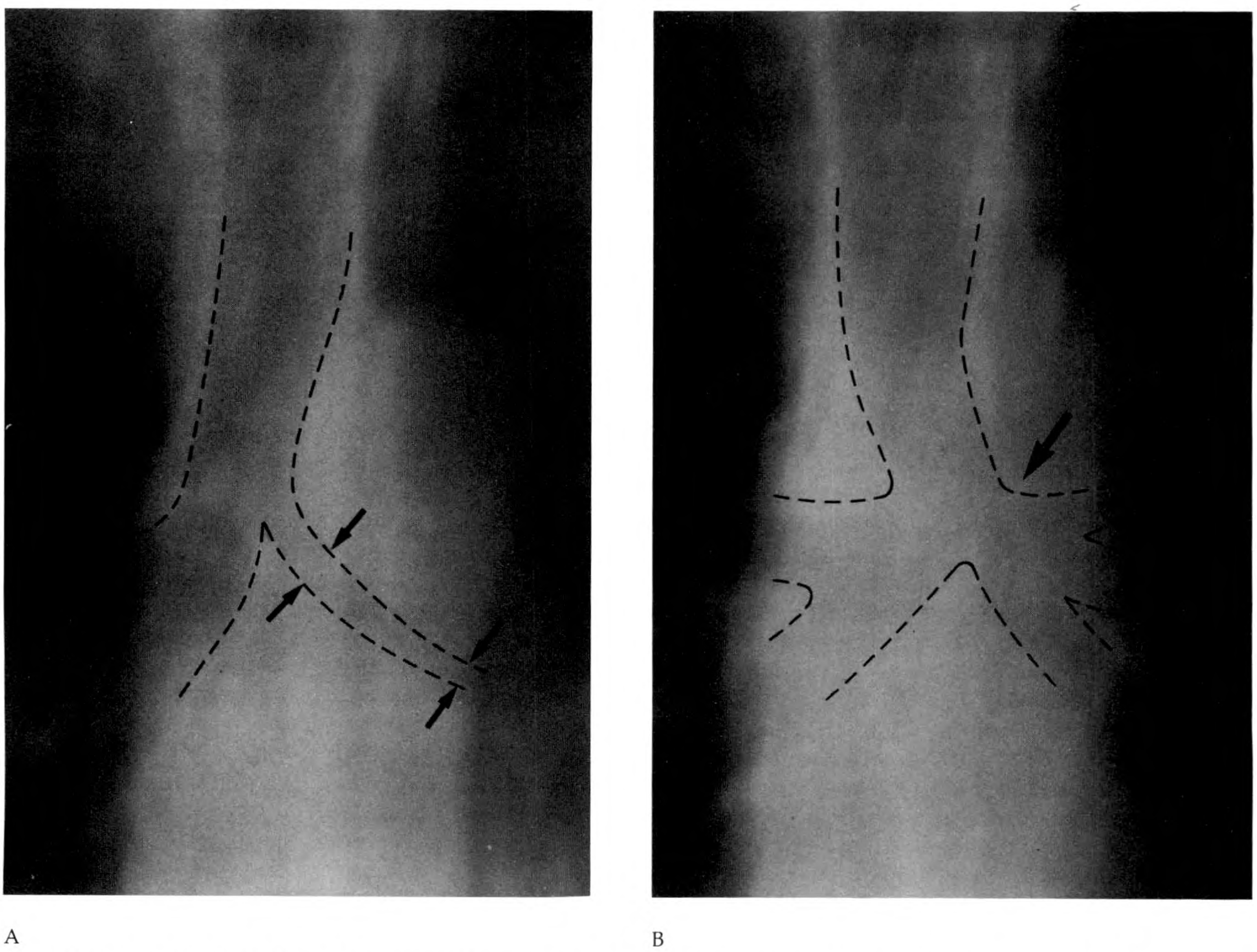

Fig 6. (A) Preoperative tracheobronchial tomograms demonstrating total left main stenosis secondary to tuberculosis as outlined by the arrows. *(B) Postoperative tracheobronchial tomograms demonstrating left upper and lower division bronchi anastomosed to the lateral trachea at the site of the* arrow.

with a left main bronchial fibrous histiocytoma underwent a therapeutic bronchoscopy 48 hours before thoracotomy, and 1 patient with a right main bronchial carcinoid underwent therapeutic bronchoscopy 24 hours before thoracotomy. Both needed prompt relief of obstruction and drainage of postobstructive pneumonia in conjunction with appropriate preoperative antibiotics and chest physiotherapy to reduce sepsis in the involved lung.

Morbidity and Mortality

Complications (Table 2) occurred in 4 of the 26 patients (15%). A 29-year-old woman with a 1-cm mucoepidermoid tumor of the left main bronchus had an initial 2.2-cm resection performed. Postoperatively, shortness of breath and wheezing prompted bronchoscopic examination, which revealed partial dehiscence due to anastomotic tension, which progressed to stenosis. She was managed with five rigid bronchoscopic dilations over the ensuing 5 months before undergoing subtotal resection of the remainder of her left main bronchus with intrapericardial hilar release. She recovered without difficulty; 6 years postoperatively she is working without respiratory symptoms. Transient hoarseness and temporary left vocal cord

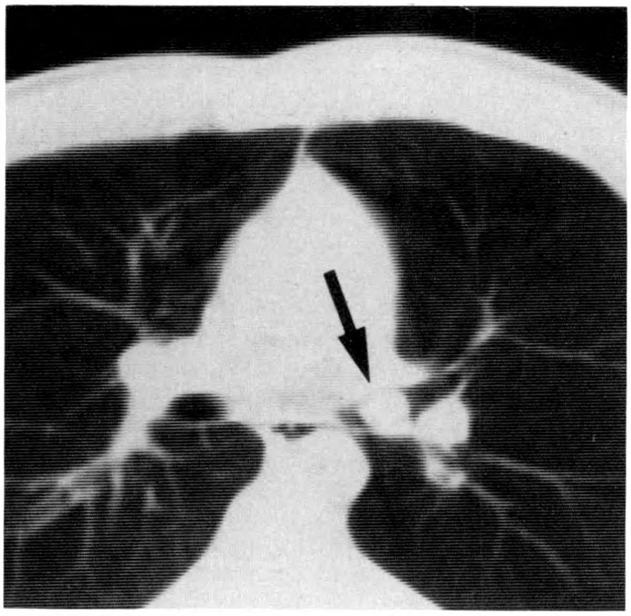

Fig 7. Computed tomographic scan of a left main bronchial carcinoid tumor (arrow).

Table 2. Morbidity of Main Bronchial Resections[a]

Morbidity	Right	Left
Anastomotic stenosis	0	1
Transient recurrent nerve palsy	0	1
Pneumonia	1	0
Prolonged air leak	1	0
Atelectasis	0	1

[a] There was no operative mortality.

palsy developed in 1 patient with left main bronchial resection for a lymphoid aggregate but resolved without treatment after 6 months. One patient undergoing resection for stenosis after right upper lobectomy for adenocarcinoma had development of a prolonged (30-day) air leak which healed spontaneously. He also had a postobstructive *Hemophilus* pneumonia on presentation; postoperatively a new left lower lobe infiltrate developed despite clearing of the right lung. This responded to appropriate antibiotics. Minimal left lower lobe atelectasis, which responded to chest physiotherapy and did not prolong hospital stay, occurred in 1 patient after left main bronchial resection. There were no problems with anastomotic granulations, pulmonary torsion, or fistula.

No patient died before hospital discharge or within 30 days of operation. Long-term follow-up showed that 92% of the patients (24 of 26) were alive a median of 5.0 years after operation (range, 1 month to 13 years). One patient, a 55-year-old woman who underwent left main resection for carcinoid, died 22 months postoperatively of a severe head injury after a motor vehicle accident. The second death occurred in a 35-year-old woman who underwent total left main bronchial resection for idiopathic stenosis. She originally was seen 5 years before operation with diffuse tracheobronchial inflammation. She required periodic dilations. During a period of decreased inflammation the persistent isolated left main bronchial stenosis was resected. Initially her postoperative course was uncomplicated, but recurrent dyspnea and wheezing 4 months postoperatively mandated four bronchoscopic dilations over a 14.5-month period before her death. Diffuse inflammation and stenosis of the distal trachea and both bronchi were noted. She suffered a respiratory arrest at home and died subsequently of anoxic cerebral injury at another hospital. One other patient also has suffered from diffuse idiopathic tracheobronchial stenosis. She is alive 12 years after left main bronchial resection with a patent anastomosis but requires dilation of her distal trachea and right main bronchus every 4 months.

Postoperative Evaluation

Twenty-four patients underwent postoperative bronchoscopy (usually approximately 3 months after resection), and 21 (86%) were normal. Two of the abnormal examinations revealed diffuse inflammatory stenosis in the trachea and right main bronchus, both in patients with idiopathic stenosis and left main bronchial resection. One abnormal examination was in the patient requiring reoperation for anastomotic stenosis. Her bronchoscopic examination is now normal. Eleven patients had postoperative tomograms; 10 were normal (see Figs 5, 6). The abnormal examination was in 1 of the patients with idiopathic stenosis. Twenty-three of the 24 living patients (96%) currently report no symptoms whatsoever and have returned to work. One patient with idiopathic stenosis has dyspnea.

One patient who underwent resection of a right main bronchial stenosis after right upper lobectomy and irradiation (3,500 cGy) for lung carcinoma had a second primary lung tumor resected from his left lung 3 years after his main bronchial operation. He is the only patient in the series other than the 2 with idiopathic stenosis in whom any other chest pathology developed. Early postoperative pulmonary function testing was formally performed on 3 patients who also had such studies preoperatively. Performed an average of 7 days postoperatively (just before discharge), the studies had essentially the same results as preoperative studies. The lack of demonstrated improvement probably is due to persistent thoracotomy discomfort at 7 days.

Comment

Main bronchial circumferential resection with end-to-end anastomosis has been described previously, mostly for traumatic disruptions and resultant stenosis and occasionally for primary tumors or tuberculosis. Although main bronchial traumatic injuries were described in 1873 [6] and 1947 [7], it was not until 1949 that Griffith [8] successfully performed a near circumferential resection of a posttraumatic stenosis of the left main bronchus with end-to-end reconstruction. Previous experimental work by Jackson and associates in 1948 [9] showed that main bronchial resection and primary anastomosis could be carried out successfully in the dog model. A variety of other authors have reported resection and reconstruction of main bronchial posttraumatic stenosis, with Deslaurier and colleagues' series of 7 in 1981 [10] and Hood and Sloan's series of 4 in 1959 [11] representing the largest experiences. Few reports of main bronchial resection and reconstruction for tumor, which form the bulk of this report, have appeared. Isolated cases of successful surgical treatment by circumferential resection and end-to-end anastomosis for carcinoid [12–14], fibrous histiocytoma (15), tuberculosis [16], and carcinoma [17] have been described. Frist and co-workers [3] noted some of the present cases in a report that dealt mostly with sleeve pulmonary resections.

Other authors have reported bronchoplastic techniques either with lateral closure or with interposition or augmentation of the airway diameter with artificial or autologous materials [17–25]. These techniques may result in narrowing of the airway due to either the initial repair or subsequent scarring. Avoidance of any narrowing is a primary concern in airway reconstruction. Circumferential sleeve resection with precise primary end-to-end anastomosis as described here usually avoids this complication.

The pathology of main bronchial lesions differs somewhat from lesions found in the remainder of the airway and generally permits their isolated resection. At the Massachusetts General Hospital, tracheal resection and reconstruction is undertaken most commonly (75% of cases) for postintubation stenosis [1–3]. Stenosis was a common main bronchial lesion as well (37%), although none were secondary to intubation. Prior surgical manipulation, trauma, laser injury, and idiopathic stenosis were the pathologic lesions responsible for main bronchial stenosis. Previous surgical manipulation was usually pulmonary resection with tangential lobar bronchial division and resultant narrowing. Such stenoses would probably have been avoided if sleeve lobectomy had been carried out primarily.

The most common tumors of the main bronchi were carcinoid (33%), mucoepidermoid (7.5%), and fibrous histiocytoma (7.5%). In the trachea, the most common tumors are adenoid cystic (approximately 40%) and squamous carcinoma (approximately 35%) [2], neither of which were found as isolated main bronchial lesions. Isolated carinal lesions, perhaps representing a transition zone between the trachea and main bronchus, reflect a broad mixture of lesions including adenoid cystic carcinoma (approximately 40%), carcinoid, stenosis, squamous cell carcinoma, and mucoepidermoid tumor [1]. Distal airway resections (sleeve lobectomy) were done at the Massachusetts General Hospital mainly for bronchogenic carcinoma (70%) and less commonly for carcinoid (21%) [3].

Based on the data presented, preoperative evaluation of patients with main bronchial lesions should include standard chest roentgenogram and tomography. Bronchoscopic examination of patients should be performed along with pulmonary function testing. If there is concern regarding potential pneumonectomy, ventilation-perfusion scanning should be performed. If the clinical course suggests an obstructive pneumonia, preoperative culture-specific antibiotics and bronchoscopic decompression should be used, followed in 24 to 48 hours by resection. Metastatic work-up and mediastinoscopic staging generally are not indicated as the majority of these lesions are benign or of low-grade malignancy (96% of cases). Patients with persistent tracheobronchial inflammation and focal areas of stenosis without identifiable causative factors should be handled cautiously as further airway problems may develop in these patients. This was exemplified by the 2 patients with idiopathic stenosis.

Operative management is facilitated by use of the long, flexible Wilson tube. Tension-reducing maneuvers are critical for left main bronchial resection. Initially all patients should have the inferior pulmonary ligament divided and should have pretracheal dissection and neck flexion performed. If, upon approximation of the lateral traction sutures, tension still exists, then intrapericardial hilar release may yield 1 to 2 cm of added length, especially for subtotal or total left main bronchial resections. Tracheal and bronchial traction tapes, pulmonary artery retraction, and aortic mobilization are critical maneuvers for exposure. Clear resection margins and a precise anastomosis with absorbable sutures complete the operation. Postoperative tomograms and follow-up bronchoscopy either before discharge or several months postoperatively or both assist in ensuring an excellent result. A failed anastomosis can be successfully resected again as long as maximal tension reduction can be achieved. Postoperative stenosis, granulations, fistula, or other complications should be watched for but are rare.

Main bronchial resection with pulmonary preservation can be carried out with minimal morbidity and mortality. The usually benign or low-grade malignant pathology permit such resections without pneumonectomy. Successful preservation of the normal lung allows virtually all patients to return to unrestricted activity.

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DISCUSSION

DR HAROLD C. URSCHEL, JR (Dallas, TX): I congratulate Dr Newton and his colleagues on a landmark paper. I want to make two technical points. I talked to Hermes Grillo about a recent patient of mine. This 26-year-old patient had a totally occluded left main bronchus and underwent laser removal of a mucoepidermoid carcinoma of the left main bronchus. A stent was placed to support the bronchus, allowing time for drainage of the infected lung before resection. This is a valuable technique for total occlusions if an attempt is to be made to save the whole lung.

The second point is that he described the inferior pericardial release, which we use. But in this case we performed a total circular pericardial release, which we use for lung transplants. The whole lung comes up easily without any tension at all on the tracheal suture line. I think the principles that you have illustrated technically are very valuable to all of us.

DR NEWTON: Thank you very much.

DR ROBERT J. GINSBERG (New York, NY): I want to emphasize one technical point that Newton and associates mentioned briefly, and that is the type of tube used. Those who use a right-sided double-lumen tube will find that they cannot always mobilize the carina the way Newton and associates have demonstrated so nicely. It is very important to use something like the Roger Wilson adaptation, or a bronchial blocker [1], to allow the carina to be pulled down through the aortic arch. A double-lumen tube will fix the carina on the other side of the arch, making the anastomosis extremely difficult. I think this is a very important point in performing left main bronchial anastomoses.

DR NEWTON: I agree with that point. We found the flexibility of the Wilson tube to be very important.

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Treatment of Aortic Coarctation by Axillofemoral Bypass Grafting in the High-Risk Patient

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Operative correction of coarctation of the aorta has been performed for 45 years. Reoperation for recurrent coarctation is necessary in as many as 5% to 10% of patients. Repair of recurrent coarctation carries an operative mortality of between 5% and 10%. Coarctation repair involves an increased risk in patients with advanced age, recurrent coarctation, congestive heart failure, and pulmonary disease. We report 3 cases where axillofemoral bypass has been used to treat high-risk patients with aortic coarctation. Two patients had had previous coarctation repair in addition to serious medical problems. Another patient had suffered three myocardial infarctions and had disabling congestive heart failure. All patients had an immediate marked decrease in their preoperative peak systolic pressure gradient across the coarctation. Systemic hypertension and symptoms of congestive heart failure were improved in all patients. The length of follow-up was 15 months, 8.5 years, and 10.5 years. Reassessment with noninvasive vascular segmental pressure studies with and without an exercise component showed no recurrence of the pressure gradient. This procedure should be considered when treating coarctation of the aorta in the high-risk adult.

(Ann Thorac Surg 1991;52:1281–4)

The first successful operative correction of coarctation of the thoracic aorta was reported by Crafoord and Nylin [1], who performed resection and end-to-end anastomosis of coarctation in 2 patients in 1944. Gross [2] applied the techniques learned from his extensive animal research on thoracic coarctation when he performed a successful coarctation repair in 1945. Since then, other techniques for the treatment of coarctation have been described including interposition grafting, subclavian flap angioplasty, and intraluminal angioplasty.

Although these procedures are more commonly performed in childhood and early adult life, there are a substantial number of older patients who require surgical repair of aortic coarctation, usually for a recurrence. A number of series have reported recurrent coarctation in from 5% to 50% of patients [3]. In general, recurrent coarctation repair can be performed effectively with acceptable mortality rates quoted at between 5% and 10%.

We believe, however, that there is a subset of patients with either aortic coarctation or recurrent aortic coarctation who would fall into a much higher risk category for repair by an intrathoracic approach owing to advanced age and associated medical disorders such as severe congestive heart failure, pulmonary disease, and cerebrovascular disease.

In 3 older patients with coarctation of the aorta and serious risk factors for perioperative mortality or complication, an axillofemoral bypass was performed to minimize operative risk while at the same time providing correction of the gradient from the upper to the lower extremities. This technique has been used once in the repair of a 14-year-old boy with tandem coarctation of the thoracic and abdominal aorta with an intervening hypoplastic thoracic aorta [4].

Case Reports

Patient 1

The patient is a 70-year-old woman who had undergone coarctation repair at another hospital in 1949 at age 30 years. She initially did well, but in 1965 she required treatment for hypertension and diabetes. Subsequently she required treatment for supraventricular arrhythmias. In 1975 she had a cerebrovascular accident involving the vertebrobasilar system. In 1978 she was hospitalized with congestive heart failure. Cardiac catheterization revealed an 80-mm Hg gradient across her coarctation. Her symptoms of congestive failure progressed to New York Heart Association class III. Her age of 59 years and her comorbidity (cerebrovascular disease, congestive failure, and diabetes) made her risk for thoracotomy prohibitive. Therefore a right axillofemoral bypass was performed in March 1979 using a preclotted 12-mm-diameter knitted Dacron graft. Her mean pressures intraoperatively were 80 mm Hg proximally and 77 mm Hg distally after the bypass.

The patient did well. Her systolic blood pressure decreased from between 200 and 210 mm Hg on admission to between 150 and 180 mm Hg postoperatively on the same antihypertensive regimen. Over the next several years she required outpatient management of exertional

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Table 1. Noninvasive Vascular Studies

Patient No.	Time (mo)	Pressure[a] Brachial	Pressure[a] Thigh	Gradient[b]	PE Gradient[b]
1	108	102	111	0	0
2	104	170	169	1	0
3	15	149	161	0	0

[a] Peak systolic pressure in millimeters of mercury. [b] Gradient from arm to thigh in millimeters of mercury.

PE = postexercise.

angina and mild congestive heart failure. Over the last 2 years she has suffered several myocardial infarctions and has required hospitalization on multiple occasions. At 10.5 years' follow-up her graft was patent. Her most recent arm peak systolic pressure was 102 mm Hg with a systolic thigh pressure of 104 mm Hg (Table 1).

Patient 2

The patient is a 76-year-old man who was first found to have coarctation of the aorta in 1978 at the age of 65 years. Angiography at that time showed a lesion 2 cm distal to the left subclavian artery with a 100-mm Hg gradient across the aorta. He has a long history of hypertension and had his first of three myocardial infarctions in 1970. Between 1978 and 1980 the patient remained in a borderline compensated state with recurrent angina, dyspnea on exertion, and cardiomegaly. Ventricular ectopy developed, necessitating antiarrhythmic therapy, and the patient also had a conduction disturbance characterized by a left bundle-branch block and an intermittent high-grade atrioventricular block.

In late 1980 the patient was admitted to our institution for further evaluation, which included noninvasive vascular studies. These demonstrated a right brachial pressure of 180 mm Hg and a right thigh peak systolic pressure of 88 mm Hg. Consideration was given to repairing his coarctation; however, it was believed that the patient's age, history of multiple myocardial infarctions, and arrhythmias placed him at substantially increased risk for thoracotomy and repair of his coarctation. A right axillo-bifemoral bypass was performed using a preclotted 12-mm-diameter knitted Dacron graft. Postoperative angiography demonstrated preferential shunting of blood through the graft and a decrease in the gradient across the coarctation to 40 mm Hg. The patient's upper extremity blood pressure subsequently decreased to 130/70 mm Hg. One month later there was no segmental pressure gradient from the arm to the thigh. At the time of his most recent arterial examination in October 1989, there was no pressure gradient from upper to lower extremities at rest or after exercising on a treadmill for 5 minutes at 1½ mph with a 10-degree grade.

During the 9 years of follow-up the patient has done extremely well with well controlled hypertension and an improvement in his later noninvasive vascular studies (see Table 1).

Patient 3

This male patient had undergone an operation for his coarctation at age 16 years in 1949; the operation consisted of a division of the distal subclavian artery, which was turned down and anastomosed end-to-side to the aorta distal to the coarctation. In 1965 he had hypertension and stenosis of the anastomosis. He underwent thoracotomy, resection of the coarctation distal to the transverse arch, and replacement with an end-to-end Dacron graft.

The patient was lost to follow-up until he returned in 1988 at age 55 years with hypertension and congestive heart failure. Despite aggressive medical management his symptoms progressed from New York Heart Association class III to class IV. Noninvasive vascular studies revealed a right brachial peak systolic pressure of 166 mm Hg and thigh peak systolic pressure of 114 mm Hg. Echocardiography revealed moderately to severely reduced left ventricular function. An aortogram demonstrated severe stenosis of the graft, with a 50-mm Hg gradient between the transverse arch and the descending thoracic aorta (Fig 1). It was recognized that repeat thoracic operation posed a serious risk in this patient. A right axillofemoral bypass graft was performed to decompress the upper extremities using a 10-mm-diameter Gore-Tex conduit. Intraoperative direct pressure measurement recorded 105 mm Hg proximally and 103 mm Hg peak systolic distally. His postoperative course was unremarkable.

At follow-up 7 months later, the patient had returned to work full time and his hypertension was controlled by medication. A noninvasive postexercise arterial examination demonstrated no gradient from upper to lower extremities 15 months after operation (see Table 1).

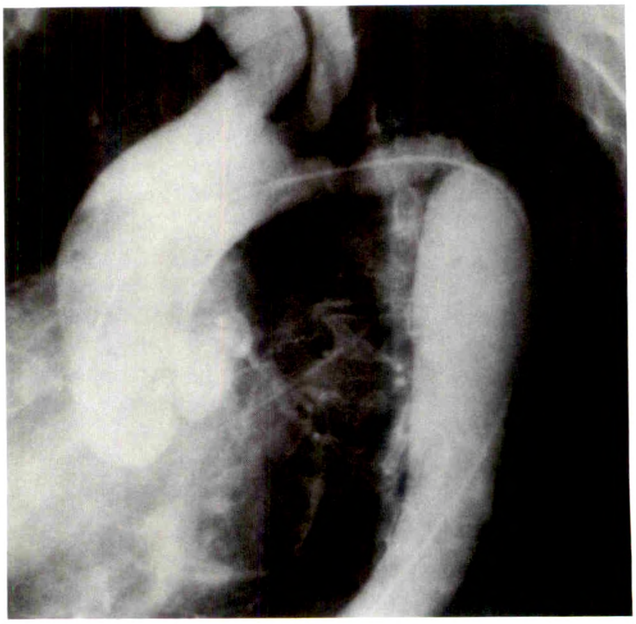

Fig 1. Aortogram of patient 3 demonstrating severe stenosis of previous graft and recurrent coarctation.

Comment

The older population can present difficult problems when planning operation for aortic coarctation. Reoperation for restenosis of the aorta is a fairly common occurrence, as seen in 2 of the 3 patients described here. A recent review reported the incidence of reoperation for aortic coarctation to be from 5% to 10.2%. These rates vary with the type of repair, age at time of first procedure, and the duration of follow-up [3, 5, 6].

Reoperation is generally believed to be indicated when there is a documented gradient of greater than 30 to 35 mm Hg at rest or exercise with evidence of recoarctation on angiography [3, 5, 7]. Noninvasive vascular studies, particularly with an exercise component, are a sensitive indicator of recurrent or persistent coarctation [8]. Symptoms of recurrent coarctation include hypertension, exertional fatigue, severe headaches, epistaxis, congestive heart failure, and lower extremity claudication [5, 6].

Findings at the time of recurrent coarctation repair may include dense mediastinal scarring, friability of the aorta, and less extensive collateral flow around the coarctation due to the previous surgical manipulation. With these findings at the time of operation it is not surprising that there are serious complications described after reoperation. These include anastomotic bleeding, recurrent laryngeal nerve paralysis, chylothorax [5, 6], phrenic nerve injury, and intrathoracic sepsis [3]. Presumably the risk of paraplegia secondary to spinal cord ischemia would be increased in the group of patients requiring reoperation; however, this complication is reported as rare [3].

Though some series report reoperation with no mortality [5, 9], operative mortality is generally believed to be between 5% and 10% [3]. A study by Sweeney and associates [5] described 53 patients who underwent reoperation over 23 years with no mortality and only 3 recorded complications. It should be noted, however, that the average age at reoperation in their study was only 18 years and congestive heart failure was present in only 3 of 53 patients preoperatively. Pollack and co-workers [9] reported a series demonstrating no mortality at reoperation, but their oldest patient was 21 years old.

A variety of techniques for reoperation have been described to try to limit the above-noted morbidities. These methods include Dacron patch angioplasty, intrathoracic bypass around the coarctation [5, 10], resection with interposition graft, and resection with end-to-end anastomosis. Balloon dilatation angioplasty is described in the pediatric population [11].

Late results after reoperation for aortic coarctation have shown that from 30% to 35% of patients have an upper extremity to lower extremity pressure gradient of more than 20 mm Hg [3]. Persistent hypertension has been documented in 20% of younger patients [9].

Axillofemoral grafts were first described by Blaisdell and Hall [12] in 1962 when they bypassed an abdominal aortic graft occlusion in a patient who had suffered an arrest upon induction of anesthesia. The patient survived the hospitalization and was ambulatory. As shown in our patients, axillofemoral bypass as a method of palliating recurrent coarctation avoids the potential intrathoracic complications described previously (Fig 2). In addition, the risk of perioperative death is presumably lessened in the patient with serious pulmonary disease and ventricular dysfunction by avoiding an intrathoracic approach. The procedure was well tolerated by the patients discussed, and follow-up data revealed excellent results concerning equalization of pressures in the upper and lower extremities and improved control of hypertension during the interval from 1 to 10.5 years postoperatively.

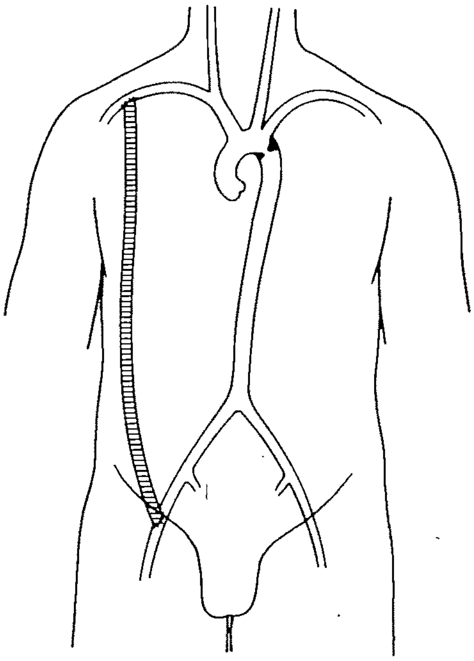

Fig 2. Use of axillofemoral bypass grafting for coarctation.

Operation for coarctation has been performed now for 45 years. One can reasonably expect that the percentage of older patients with serious risk factors requiring consideration for operative correction will increase in the near future. Axillofemoral bypass is another technique that should be in the surgeon's armamentarium when planning therapy in these patients at increased risk.

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Anomalous Origin of the Right Pulmonary Artery From the Ascending Aorta

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Five cases of anomalous origin of the right pulmonary artery from the ascending aorta were seen at our hospital. Patients 1 and 2 had an intact right pulmonary artery originating from a right posterior aspect (proximal form), and primary anastomosis of the right pulmonary artery and main pulmonary trunk was performed. Patients 3 and 4 showed a narrowing right pulmonary artery originating from near the base of the innominate artery (distal form). The stenotic right pulmonary artery was reconstructed with an 8-mm graft in patient 3, whereas patient 4 became inoperable because complete obstruction had developed in the right pulmonary artery during the 3 months that the patient was waiting to undergo operation. In patient 5, primary anastomosis was undertaken, but morphologically the anomalous origin was of the distal form, so the occurrence of stenosis in the reconstructed right pulmonary artery was a matter of concern. The morphological type was found to be related to the surgical options in this anomaly. Therefore, primary anastomosis was considered the best choice for a correction of the proximal form, whereas a graft interposition with a resection of the stenotic portion on the right pulmonary artery was deemed preferable in the distal form.

(Ann Thorac Surg 1991;52:1285–91)

Anomalous origin of the right pulmonary artery from the ascending aorta (AORPA) is a rare cardiac malformation. We operated on 5 cases of AORPA between 1983 and 1989. These could be divided into two morphological types (2 patients had the proximal form and 3, the distal form) [1], and 2 of the patients with the distal form had narrowing of the right pulmonary artery (PA). It seemed that an adequate evaluation of its pathogenesis was required for appropriate correction of the AORPA. Our purpose in the present report is to comment on the various surgical considerations of this disease.

Summary of Cases

The clinical conditions and data at admission of the 5 patients are summarized in Table 1. Two were neonates and 3 were infants at first admission. Patients 1, 2, and 4 were admitted with moderate to severe heart failure, whereas patients 3 and 5 had no prominent cardiac symptoms except for poor weight gain. In patient 4, the operation was postponed until the patient could achieve an adequate weight gain, because the patient's condition dramatically improved after admission. Two-dimensional echocardiography was able to detect the AORPA in 3 of the 5 patients.

Preoperative Angiography and Catheterization

All patients underwent preoperative angiography. Typical features of the proximal form of AORPA were observed in patients 1 (Fig 1A) and 2 (Fig 1B), that is, the right PA originating from the posterior aspect of the ascending aorta near the aortic valve. No narrowing site on the right PA was found in either case. Patients 3 (Fig 1C) and 4 (Fig 1D) had a narrowing site on the middle portion of the right PA, which originated from the right lateral aspect of the ascending aorta. Those features were compatible with the distal form of AORPA. Patient 5 (Fig 1E) showed no evidence of narrowing of the right PA, but the takeoff from the aorta was near the bifurcation of the innominate artery, so this case of AORPA seemed to belong to the distal form. Patients 1, 2, 3, and 5 also had patent ductus arteriosus.

The pressure values at the first catheterization are given in Table 2. Severe pulmonary hypertension was demonstrated in all patients. Even in the patients with stenotic right PA (patients 3 and 4), pulmonary hypertension was found.

Operative Findings and Surgical Procedures

Surgical repair was performed several days after the first catheterization in patients 1, 2, 3, and 5. However, in patient 4, the operation was undertaken about 3 months after the first catheterization. In all patients, the correction was performed through a median sternotomy. Cardiopulmonary bypass was not used in patients 1, 3, 4, or 5 but was on standby. In patient 2, the most recent case, cardiopulmonary bypass was used.

PATIENT 1. The main pulmonary artery was enlarged (11 mm) and the right PA (5 mm) originated from the right posterior aspect of the ascending aorta. A partial occluding clamp was placed across the right side of the aorta to

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Table 1. Clinical Features at Admission

Patient No.	Age	Sex	Weight (g)	History	Murmur	CTR[a]	ECG
1	16 d	F	2,530	Tachypnea, mild cyanosis	Grade III SM, 3LSB	0.69, Atelectasis	RAD, BVH
2	2 mo	M	3,900	Tachypnea	Grade II SM, 2LSB; grade II MDM, Apex	0.64	NAD, RVH
3	1 y 5 mo	F	7,400	Poor weight gain	Grade III continuous, 2RSB	0.59, PV (right < left)	RAD, RVH
4	22 d (4 mo)[b]	F	4,900	Tachypnea, mild cyanosis	Grade III SM, 3-4LSB	0.64	NAD, BVH
5	7 mo	F	4,200	Poor weight gain	Grade I-II SM, 2LSB	0.60	NAD, RVH

[a] By chest roentgenogram. [b] In patient 4, operation was postponed for 3 months after the first admission.

BVH = biventricular hypertrophy; CTR = cardiothoracic ratio; ECG = electrocardiogram; LSB = left sternal border; MDM = middiastolic murmur; NAD = normal axis deviation; PV= pulmonary vascularity; RAD = right axis deviation; RSB = right sternal border; RVH = right ventricular hypertrophy; SM = systolic murmur.

include the origin of the right PA. The right PA was divided, and the aorta was sutured with 5-0 polypropylene. The right PA was passed behind the ascending aorta, and the stump was anastomosed to the right lateral aspect of the side-clamped main pulmonary trunk in an end-to-side fashion with 6-0 polypropylene continuous suture posteriorly and with an interrupted suture anteriorly. The patent ductus arteriosus was then doubly ligated.

PATIENT 2. The right PA (6 mm in diameter) originated from the ascending aorta in the same fashion as in patient 1. A pansystolic thrill was palpable on the right atrial wall due to tricuspid regurgitation. Cardiopulmonary bypass was performed using aortic cannulation, which was done 5 mm distal to the takeoff of the innominate artery using a 2.4-mm metal cannula, and venous cannulation, which was selectively inserted in the superior and inferior venae cavae. Moderate hypothermia with high-flow cardiopulmonary bypass (2.5 L/min/BSA) was performed. After ligation of the patent ductus arteriosus, cardioplegic arrest was induced by crystalloid cardioplegia (glucose, 2.5 g/L; sodium chloride, 75 mmol/L; sodium bicarbonate, 10 mmol/L; potassium chloride, 20 mmol/L; calcium gluconate, 0.6 mmol/L; xylocaine, 100 mg/L; albumin, 10 g/L; osmolarity, 340 mOsm/L) combined with topical cooling using ice slush. After aortic cross-clamping and occlusion of the right PA, the right PA was divided at its origin and an end-to-side anastomosis was performed with absorbable sutures. The aortic cross-clamp time was 46 minutes.

PATIENT 3. A right PA originated from the right lateral aspect of the ascending aorta near the takeoff of the innominate artery. The middle portion of the right PA was stenotic, with a luminal diameter of 1 mm. After division of the right PA, a longitudinal incision was made in the peripheral side through the stenotic portion. A shelf was found in the stenotic portion. Graft interposition was performed using an 8-mm polytetrafluoroethylene graft. The new right PA was reconstructed through the anterior side of the aorta.

PATIENT 4 (INOPERABLE CASE). The right PA originated from the ascending aorta in the same fashion as in patient 3. However, at 10 mm distal to its origin, the right PA showed a complete obstruction. We dissected the obstructed peripheral right PA beyond its trifurcation, but atrophic change of the right PA extended to the intrapulmonary artery, which prevented any surgical repair. The stenotic lesion on the right PA was considered to have developed during the 3 months while waiting for the operation. The magnetic flowmeter indicated a flow of 0 mL/min in the right PA. Fortunately, the main pulmonary arterial pressure showed a normal value (33/8 mm Hg).

PATIENT 5. The right PA (8 mm in diameter) originated from right lateral aspect of the ascending aorta just proximal to the takeoff of the innominate artery. No stenotic segment was observed in the right PA. Primary anastomosis was performed with a double ligation of the patent ductus arteriosus. We encountered severe hypotension and bradycardia during the anastomosis. This episode discouraged us from reconstructing the AORPA routinely without cardiopulmonary bypass.

Postoperative Angiography, Catheterization, and Courses

Postoperative angiographic and catheterization data are shown in Figure 2 and Table 3. Postoperatively, all patients had uneventful courses, and in the patients who were operated on, the symptoms of heart failure dramatically improved. Cardiac catheterization at 1 month after operation showed the tendency of residual moderate pulmonary hypertension (pulmonary to systemic pressure ratio, 0.5). In patients 1 and 2, neither evidence of stenosis nor progress of pulmonary hypertension had appeared at 6 years and 1 year after operation, respectively. In patient 1, a lung perfusion scintigram 18 months after operation showed no perfusion defect in the right lung (Fig 3). In the patient who received a graft (patient 3),

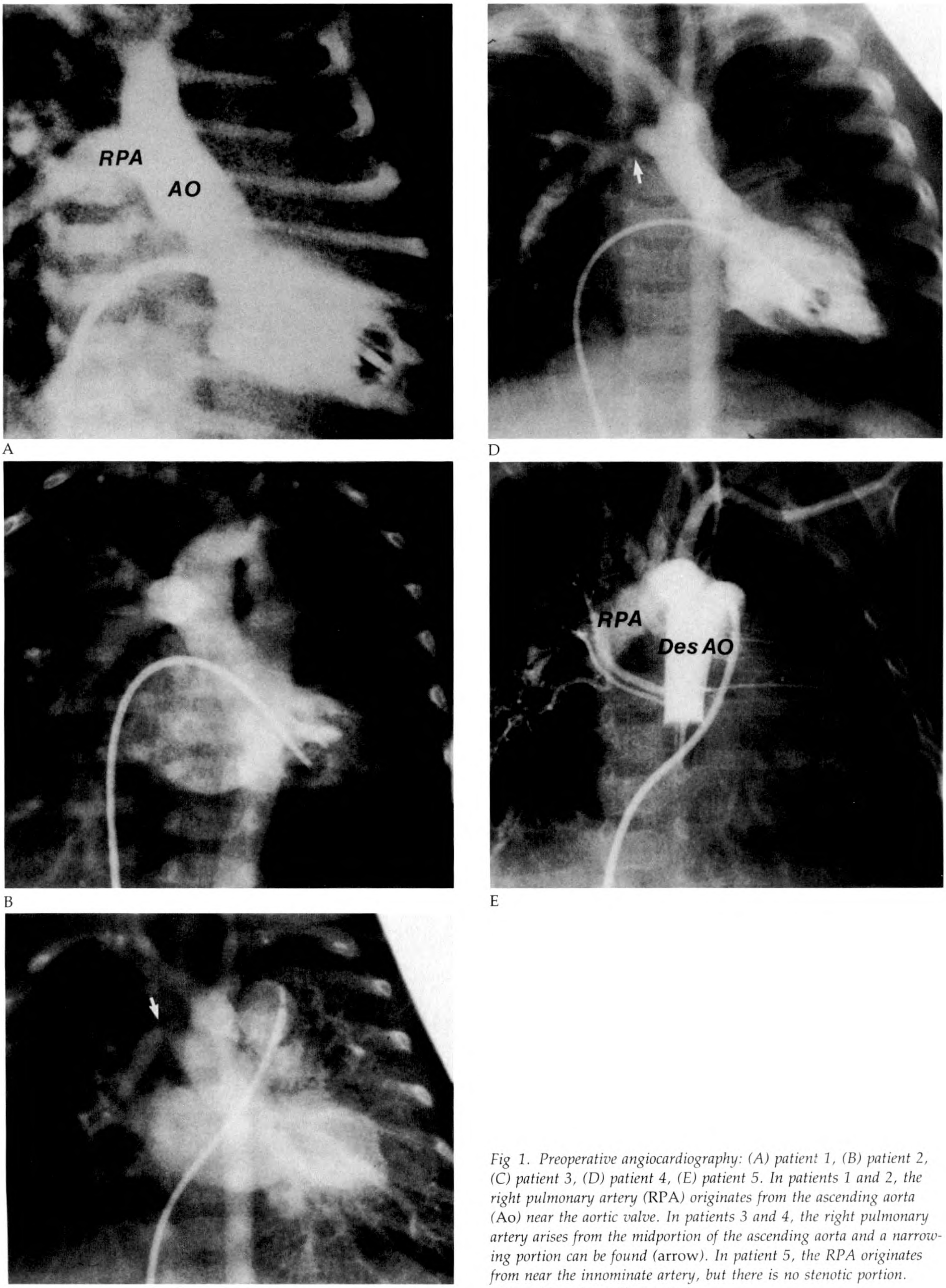

Fig 1. Preoperative angiocardiography: (A) patient 1, (B) patient 2, (C) patient 3, (D) patient 4, (E) patient 5. In patients 1 and 2, the right pulmonary artery (RPA) *originates from the ascending aorta* (Ao) *near the aortic valve. In patients 3 and 4, the right pulmonary artery arises from the midportion of the ascending aorta and a narrowing portion can be found* (arrow)*. In patient 5, the RPA originates from near the innominate artery, but there is no stenotic portion.* (Des AO = *descending aorta.)*

Table 2. Preoperative Cardiac Catheterization Data

Patient	Pressure (mm Hg)					
No.	Mean RA	RV	MPA	Mean LA	LV (Ao)	Pp/Ps
1	2	78/6	67/36	9	66/15	1.02
2	4	64/0	63/32	13	69/23	0.91
3	1	50/0	47/27	. . .	(61/30)	0.77
4	5	108/8	104/49	3	91/6	1.14
5	4	97/0	80/50	. . .	(75/50)	1.06

LA = left atrial; LV (Ao) = left ventricular (aortic); MPA = main pulmonary artery; Pp/Ps = pulmonary to systemic peak pressure ratio; RA = right atrial; RV = right ventricular.

anticoagulant therapy (aspirin, 10 mg · kg^{-1} · day^{-1}, and dipyridamole, 5 mg · kg^{-1} · day^{-1}) has been continued. Although a chest roentgenogram showed no laterality of pulmonary vascularity, the systolic murmur increased at the right upper sternal border 2 years after operation. In the inoperable patient (patient 4), no pulmonary hypertension had developed by 3 years after operation. The patient has no particular complaints, but the laterality of the pulmonary vascularity is unchanged. We carefully followed up patient 5 in regard to the stenosis in the right PA. Neither laterality of pulmonary vascularity on chest roentgenograms nor a heart murmur has appeared in the recent outpatient check-ups.

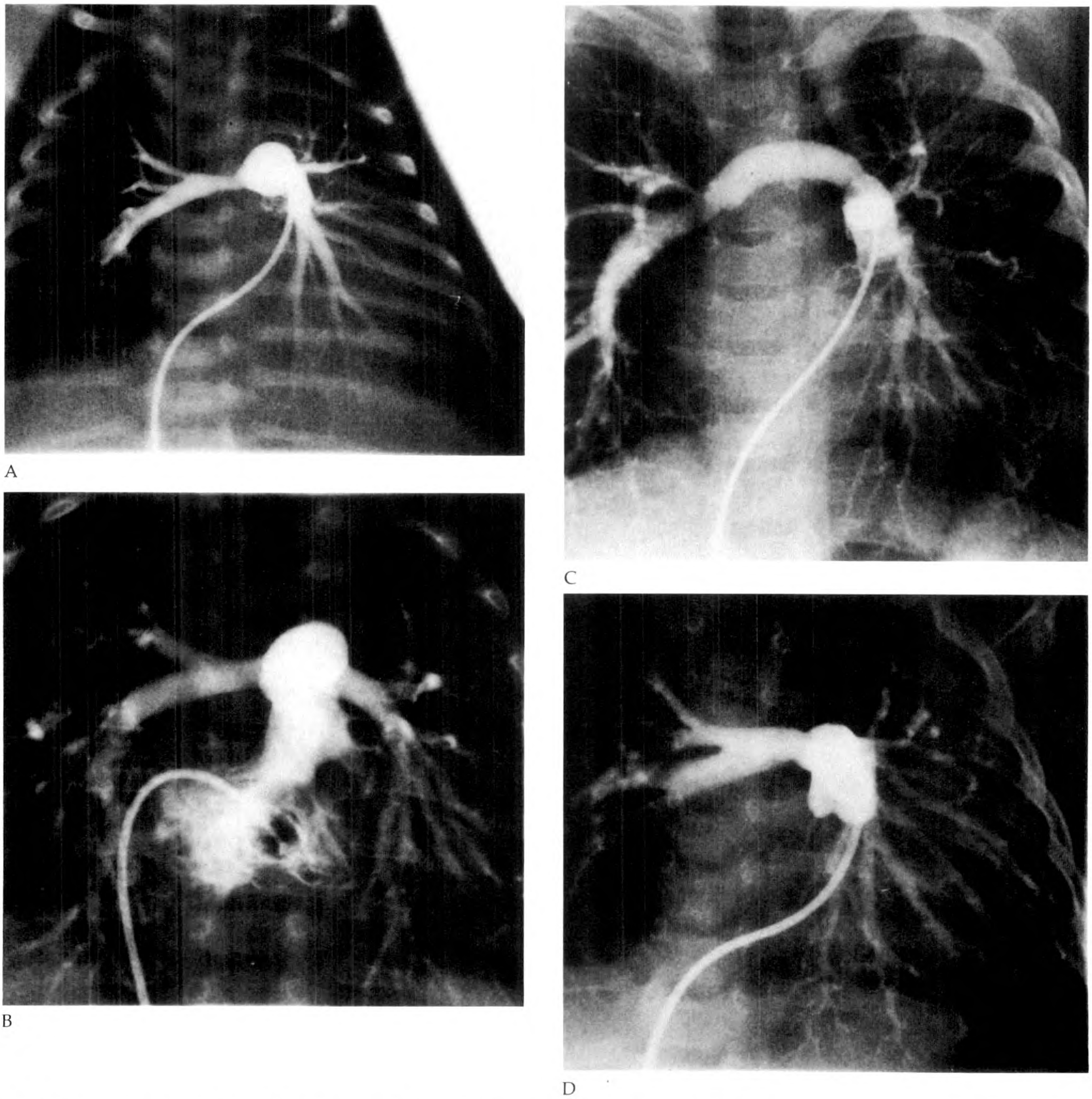

Fig 2. Postoperative angiocardiography: (A) patient 1, (B) patient 2, (C) patient 3, (D) patient 5. Direct anastomosis was performed in patients 1, 2, and 5, and a graft interposition was undertaken in patient 3.

Comment

Anomalous origin of the right pulmonary artery from the ascending aorta is a rare malformation. Since it was first reported by Fraentzel [2] in 1868, more than 80 cases have been documented in terms of its interesting embryological and physiological features. It is generally recognized that AORPA has two different formal types of genesis [1, 3, 4], the proximal and distal form.

In the proximal form, the anomalous right PA originates from the right posterior aspect of the proximal ascending aorta close to the aortic valve. The cumulative study by Kutsche and Van Mierop [1] showed that 85% of cases of AORPA were classified as this type. Schneiderman [5] and others [6] consider it to be the result of a lack of leftward migration of the right sixth aortic arch. Cucci and associates [7] and Richardson and co-workers [8] believe it to be due to an abnormal truncal septation. The abnormal right PA develops from the proximal portion of the right sixth arch in the same way a normal one does, and there is no narrowing site in this type.

On the other hand, in the distal form, the anomalous right PA originates from the ascending aorta near the base of the innominate artery. Odell and Smith [9] explained that a fifth arch developed and the proximal and the distal part of the sixth arch disappeared. The dorsal portion of the sixth arch (which involved patent ductus arteriosus tissue) thus remains connected to the fifth arch and pulmonary artery. This is a plausible explanation for the fact that narrowing of the PA often occurs in this type of AORPA. The difference in the pathogenesis between the two types could also be suggested by the associated anomalies. Aorticopulmonary septal defect (15%) and the interruption of aortic arch type A (9%) are commonly associated anomalies in the proximal type [1, 10], whereas they are not common in the distal type. Patients 1 and 2 could be classified as having the proximal form, and the others, the distal form. Patients 3 and 4 had narrowing of the PA, whereas patient 5 had none.

We want to comment on the surgical considerations for this anomaly. There is no doubt that radical correction is life saving for this anomaly. Penkoske and associates [6] report that the mortality of this malformation with palliative operation including ligation of the anomalous RPA, ligation of the patent ductus arteriosus, or PA banding reached 82% (18/22), whereas it is 26% (6/23) in radical correction. Table 4 shows the reported radical correction and its results. Those include 28 cases of the proximal form and 3 of the distal form. In the cases of proximal form, 70% had primary anastomosis performed either with or without cardiopulmonary bypass. The origins of the right PA (posterior aspect of ascending aorta) and the main PA are not distant in this form, so the right PA could easily be reanastomosed through the posterior side of the ascending aorta. Technically, adequate dissection of both PAs beyond their first bifurcation may be a point for providing an easy anastomosis.

Table 3. Postoperative Cardiac Catheterization Data

Patient No.	Pressure (mm Hg) Mean RA	RV	MPA	Mean LA	LV (Ao)	Pp/Ps
1	0	43/2	37/8	0	66/3	0.56
2	4	26/0	27/9	. . .	58/35	0.47
3	1	40/−4	43/4	1	84/−6	0.51
5	1	42/−1	38/14	. . .	(73/36)	0.52

LA = left atrial; LV (Ao) = left ventricular (aortic); MPA = main pulmonary artery; Pp/Ps = pulmonary to systemic peak pressure ratio; RA = right atrial; RV = right ventricular.

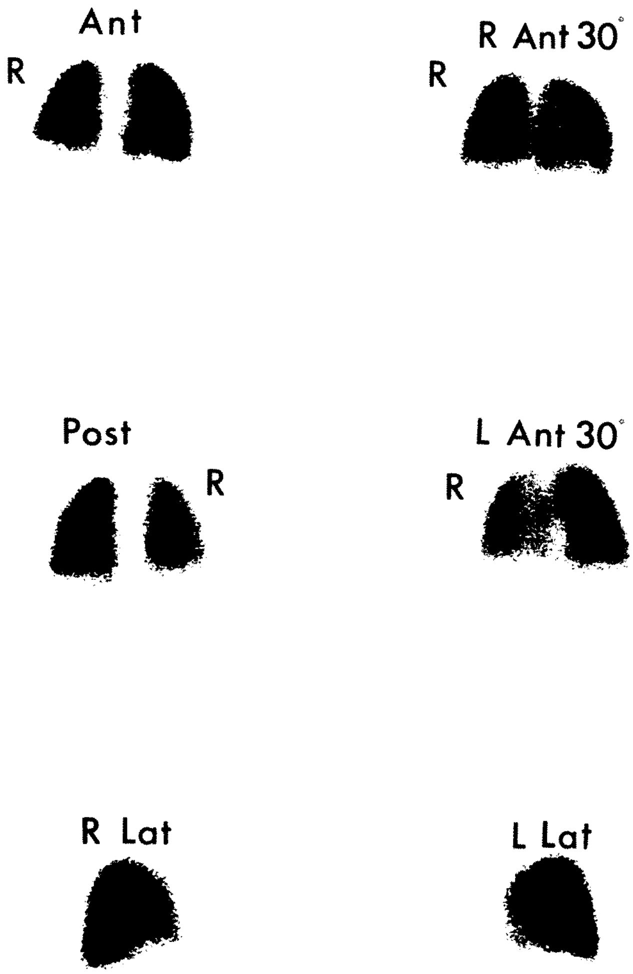

Fig 3. Lung perfusion scintigram in patient 1 at 18 months after operation shows no perfusion defect in the right lung. (Ant = *anterior;* L = *left;* lat = *lateral;* post = *posterior;* R = *right.)*

Few reports of the reconstruction of right PA with a stenotic portion can be found [11], but we encountered 2 cases of this type. Complete resection of the narrowing portion and graft interposition would be a preferable means of reconstruction in this form. The stenosis in the right PA seems to be sometimes progressive [4], so surgical intervention should not be delayed. In patient 4, complete obstruction of the stenotic right PA, which interfered with the radical correction, had developed during the 3-month waiting period. Fortunately, the pulmonary hypertension and the clinical symptoms were relieved by the disappearance of shunt flow. However, there is no doubt that two lungs are superior to one lung from the standpoint of pulmonary function. Hemoptysis

Table 4. Reported Cases of Radical Correction

Morphological Type	No. of Cases	Procedure: Primary Anastomosis	Procedure: Graft	Use of CPB or Circulatory Arrest
Proximal form	28 (5)[a]	20 +VSD closure 2 +ASD closure 2 +repair of cor triatriatum 1	8	13
Distal form	3 (0)	2	1	2
With stenosis	1	. . .	1	
Without stenosis	2	2 +TOF correction 1	. . .	
Unclear	4 (0)	3	1	No description
Total	35 (5)	25	10	15

[a] Numbers in parentheses are operative deaths.

ASD = atrial septal defect; CPB = cardiopulmonary bypass; TOF = tetralogy of Fallot; VSD = ventricular septal defect.

due to a hemorrhage into the disused lung supplied by the anomalous systemic vessels [12] is a serious complication. These disadvantages would not permit us any delay in surgical intervention in the case of a distal form of AORPA.

Although patient 5 had no evidence of narrowing lesions in the right PA, she was considered to have a distal form. The surgical problems must focus on the distal form of AORPA without a narrowing right PA. Descriptions of the microscopic structure of the right PA have seldom been given, so it is not possible to find out how often tissue of ductal origin is present in this form of AORPA. If patent ductus arteriosus tissue is contained in the right PA, then a resection of the midportion of the AORPA and reconstruction using a synthetic graft may be the best choice. A different pathogenesis might be taken into consideration for patient 5, however, such as explained by Ambrus [13]; a right fifth arch developed whereas a sixth did not. If this hypothesis is true in this case, then primary anastomosis may be the best choice. We are now carefully following up this patient.

We did not routinely use cardiopulmonary bypass to avoid its several postoperative adverse effects. Now, however, its use might be taken into consideration depending on the details of each case, because anastomosis with a side clamp would not be so easy and safe in all cases. The development of a cardiopulmonary bypass technique for neonates or small infants would be one reason for using it [14].

There are some explanations for the cause of the bilateral pulmonary hypertension caused by different blood supply to each lung [12, 15]. In the right lung, the increased pulmonary blood flow at systemic levels of pressure would affect the pulmonary vasculature like a large patent ductus arteriosus or ventricular septal defect. The left lung is perfused with the entire right ventricular output, but it is unlikely that the flow alone is responsible for the changes in this lung. The vasoconstrictive reaction due to the abnormal state of the vascular bed on the right lung is considered to be responsible for the development of pulmonary hypertension. The differential development of pulmonary obstructive disease in each lung has been also reported. Yamaki and colleagues [16] hypothesized that the increased blood flow with highly saturated blood is a cause of highly progressive vascular disease in the right lung. In the present study, all 4 patients who were operated on, which included 1 neonate and 3 infants, showed moderate pulmonary hypertension (pulmonary to systemic pressure ratio >0.5) after reconstruction of the right PA. These facts would support early surgical intervention in this anomaly.

Recently, some reports have emphasized the usefulness of echocardiography to determine the diagnosis and operative indications in this anomaly [17]. Echocardiography, however, seems unable to define the morphological type and presence of stenosis. We believe that cineangiography is necessary for determining the morphological diagnosis and surgical method. Therefore, an early definitive diagnosis by angiocardiography and prompt surgical intervention is our recommended surgical strategy for this anomaly.

In summary, 5 cases of AORPA were operated on in our hospital over a 7-year period. Two were of the proximal form and the others were of the distal form. A narrowing right PA was seen in 2 of the 3 cases of distal form of AORPA. In 1 of them, complete obstruction developed during the 3-month period before surgical intervention. The morphological type is considered to be related to the surgical therapy in this anomaly. Primary anastomosis is the choice for the proximal form of AORPA, whereas a graft interposition would be preferable for the distal form with a narrowing right PA.

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Extrapleural Takedown of the Internal Mammary Artery as a Pedicle

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Four hundred thirty-three patients underwent coronary bypass grafting using both internal mammary artery (IMA) and saphenous vein graft. In 233 patients the IMA was exposed with a conventional technique (group 1), whereas in the remaining patients the IMA was harvested leaving the pleura intact and performing an associated lysis of the endothoracic fascia (group 2). Postoperative course and complications of these groups were studied and compared in a period of time ranging from 8 to 28 months. Postoperatively, the blood transfused was significantly less in group 2 patients than in group 1; group 2 also had a reduction of surgical reexploration. Pleural effusion and hemidiaphragm impairment were infrequent or absent in group 2 patients, whereas pericarditis, severe postoperative chest pain, and respiratory insufficiency were noted only in group 1 patients. Our data suggest that extrapleural access without pleurotomy may be preferred owing to its low rate of chest wall complications.

(Ann Thorac Surg 1991;52:1292–4)

The internal mammary artery (IMA) has been shown to be the conduit with the greatest longevity among those currently available for coronary artery bypass grafting [1]. Pleurotomy during IMA graft mobilization, however, may produce some postoperative complications as reported by several surgeons [2, 3].

This study was designed to compare the postoperative course of two randomized groups of patients who underwent IMA graft mobilization with or without pleurotomy for myocardial revascularization.

Material and Methods

Since 1987, a total of 439 patients at Casa di Cura Villa Maria have been treated for coronary occlusive disease with coronary artery bypass using at least one IMA as a conduit plus saphenous vein grafts for additional vessels requiring bypass. From this group, 433 survivors were entered into the study. It is of note that 427 of the 433 left IMA grafts were placed to the left anterior descending coronary artery, 4 to the diagonal, and the remaining 2 to the intermediate. The mean of number of grafts per patient was 3.4 in group 1 and 3.6 in group 2.

Two methods of harvesting the IMA were randomly used: (1) mobilization of the IMA as a wide pedicle including a 1- to 2-cm width of the endothoracic fascia with an open-pleura access (group 1, n = 233) and (2) extrapleural takedown of the IMA with lysis of the endothoracic fascia (group 2, n = 200).

The average age of the patients was 61 years for group 1 and 59 for group 2, with a range of 34 to 73 years in each group. Eighty-five percent of the patients were male and 15% were female. All patients were in New York Heart Association functional class III or IV.

Surgical Technique

All patients were operated on by the same surgeon. Myocardial protection was provided with both systemic cooling (26°C), multidose blood cardioplegia, and topical cooling with 4° to 5° C saline solution.

In all methods, the dissection of the IMA was performed in large part with electrocautery, and the side branches were occluded with hemoclips. The IMA was mobilized from near its origin proximally to its bifurcation distally at the level of the xiphoid process. The IMA of group 1 patients was harvested with a "conventional technique "as described by Sewell and Davalos in 1962.

Extrapleural Access

The endothoracic fascia was dissected along the chondrosternal cartilages without opening the pleura. The initial incision for the dissection of the endothoracic fascia and pleura from the chest wall was performed at the level of the fourth rib. The anterior part of the IMA was freed throughout its length, and the surrounding fat was trimmed. The side arterial branches were divided between clips using gentle traction on the fascia. In this manner the IMA was separated from the chest wall, taking down both fascia and pleura.

In a second stage the IMA with surrounding fat and veins was mobilized from the endothoracic fascia, clipping the pleuropericardial arterial branches. From the first intercostal space upward, the artery was freed without the use of cautery to avoid injury to the phrenic nerve.

Postoperative Protocols

Postoperative course and complications were studied in a follow-up period ranging between 8 and 28 months. The

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Table 1. Postoperative Complications

Variable	Group 1 (n = 233)	Group 2 (n = 200)
Blood given (mean units ± standard deviation)	3.4 ± 0.30	1.2 ± 0.70[a]
Surgical reexploration	9 (3.86%)	5 (2.5%)
Pleural effusion		
Mild	52 (22.32%)	19 (9.5%)
Marked	8 (3.43%)	0
Hemidiaphragm impairment[b]		
Moderate (>2 cm)	50 (21.46%)	17 (8.5%)
Marked (<2 cm)	9 (3.86%)	0
Infection of the sternum	3 (1.29%)	0
Pericarditis	11 (4.72%)	0
Severe postop chest pain	10 (4.31%)	0
Respiratory insufficiency	3 (1.29%)	0

[a] $p < 0.05$ versus group 1. [b] Moderate elevation of the left hemidiaphragm is movement of at least 2 cm during deep inspiration; marked elevation is less than 2-cm excursion during deep inspiration.

x-ray films were used to determine the incidence and size of postoperative pleural effusion, to determine the position of the left hemidiaphragm in relationship to the right hemidiaphragm, and to observe the movement of the left diaphragm during deep inspiration. A grading system was used to define roentgenographic images as reported by Landymore and Howell [3]. Postoperative exercise testing has been carried out in 149 group 1 patients and 132 group 2 patients from 6 to 8 months after operation.

Statistical Analysis

Results have been reported as the arithmetic mean ± the standard error of the mean. Two-way analysis of variance was used for statistical comparisons. A p value of less than 0.05 was considered significant.

Results

The rate of perioperative myocardial infarction was equal in both groups, with an average of 4.5%. The intraortic balloon pump was required in 2% of each series of patients. The length of intubation and hospitalization tended to be slightly longer in patients of group 1, but the difference did not reach statistical significance. Data concerning postoperative complications of both groups are shown in Table 1.

Conventional IMA Graft Preparation (group 1)

Postoperative bleeding that required surgical reexploration occurred in 9 patients. The amount of blood given was approximately 3 units per patient (mean, 3.4 ± 0.3 units).

The follow-up of this group ranged from 10 to 28 months. A pleural effusion requiring at least one thoracentesis for resolution developed in 8 patients, whereas a moderate effusion was present in 52 patients. Marked left hemidiaphragm impairment occurred in 9 patients; moderate impairment was present in 50 patients. The elevation of the diaphragm appeared to be secondary to the presence of pleural fluid and left lower lobe atelectasis rather than phrenic nerve paralysis.

Three months after operation, 96.49% of these patients had normal chest roentgenograms. There were only 2 patients who had persistent elevated hemidiaphragm without clinical signs.

The incidence of respiratory insufficiency was 1.28%. A wound infection developed in 3 patients; none of them were diabetics. Surgical debridement was required in 1 patient, but the infection involved the mediastinum and the patient died 35 days after the second operation.

Of the 232 surviving patients, all have improved at least two New York Heart Association functional classes. Postoperatively 150 (64.38%) of these patients are in class I, 73 (31.33%) are in class II, and 9 (3.86%) are in class III.

Postoperative treadmill data were negative in all tested patients. Severe left chest wall pain with persistent discomfort occurred in 10 patients. The incidence of postoperative pericarditis within the follow-up period was 4.72%.

Extrapleural Access (group 2)

Surgical reexploration for postoperative bleeding was performed in 5 patients. A mean of 1.2 ± 0.7 units of blood was given per patient. The follow-up period ranged between 8 and 20 months.

The patients in this group had small or moderate pleural effusion, and 7 patients had evidence of diaphragmatic elevation. Although some of these effusions (2.5%) were moderate in size, none required thoracentesis. None of these patients had a diaphragmatic excursion that was less than 2 cm.

Infection of the sternum, phrenic nerve injury, pericarditis, and severe postoperative chest pain did not occur in this group of patients.

Postoperatively 160 (80%) of these patients are in class I; the remaining patients are in class II.

The anginal syndrome has been relieved in all patients, and postoperative exercise testing was negative in 130 patients. ST segment depression, but no angina, developed in 2 patients after 450 and 479 seconds.

Comment

Expanded use of IMA for myocardial revascularization is based on accumulating data of superior late patency of the IMA compared with venous conduits [4]. From a practical standpoint, there are two ways in which IMA may be harvested and used for coronary artery bypass grafting: (1) techniques that routinely open the pleura during the mobilization of IMA and (2) techniques that leave the pleura intact using extrapleural access. Several surgeons consider the second technique a problem because of the final position that IMA adopts in the mediastinum. The major criticism is that a closed pleura might push the pedicle back into its bed, decreasing the available length of the graft with subsequent torsion and tension of the pedicle [3].

A technical answer to this particular problem may be provided by dissecting the pleura off the mediastinal structures and maximizing the length of the pedicle with a fasciotomy [5]. Lysis of the endothoracic fascia may be used safely, and this maneuver may favor the final position of IMA under the left upper lobe [6, 7].

The focus of the present study was to examine two randomized groups of patients in whom the pleura was or was not maintained intact during IMA mobilization. In group 2 patients we used concomitant lysis of the endothoracic fascia without the need of a left chest drainage tube.

Postoperative bleeding decreased considerably in group 2 patients, with a significant reduction in transfused blood and a minor incidence of surgical reexploration. These results may be related to minor surgical trauma and injury to the chest wall with a limited area of dissection. It is our opinion that the closed pleura favors a decrease in the surgical time for hemostasis. In this way the surgical field may be well visualized, avoiding an oversight of blood loss into the left hemithorax.

Postoperatively, pleural effusion and elevation of the left hemidiaphragm were considerably less common and less severe in group 2 patients, who had undergone IMA dissection without pleurotomy. Postoperative pleural effusions and atelectasis of the lower lobe, which contributed to loss of left lung volume, pulmonary insufficiency, and elevation of the left hemidiaphragm, developed in almost all group 1 patients. Although we were uncertain as to the cause of these pulmonary complications, in accordance to Landymore and Howell [3] we believe that the pleura should not be opened during dissection of the IMA.

Another minor problem to be considered concerns the quality of comfort after operation; discomfort in a sternotomy wound is usually mild, but some patients who underwent the conventional IMA graft preparation had persisting discomfort and severe chest wall pain for several weeks after their discharge from the hospital. In contrast, we did not observe any important discomfort in group 2 patients.

In summary, the minor postoperative bleeding and low incidence of pulmonary complications confirm the validity of extrapleural access and preparation of IMA graft without pleurotomy. In addition, this approach has no apparent effect on long-term patency and may be preferred for its low rate of chest wall complications.

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Denervation of Transplanted Porcine Lung Causes Airway Obstruction

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Lung transplantation can be complicated by a form of small airway obstruction known as bronchiolitis obliterans. We tested the hypothesis that lung denervation causes small airway obstruction in young pigs (10 ± 1 weeks). Control pigs had an innervated native lobe, and study pigs had either a denervated native lobe or a denervated transplant lobe. Transplanted pigs received standard immunosuppression. At 10 weeks we measured isolated left lobe pulmonary mechanics. Dynamic resistance in both study groups was significantly higher than in the lobectomy group, whereas dynamic compliance in both study groups was significantly lower than in the lobectomy group. No significant difference in resistance or compliance was noted between the transplant and reimplant groups. Histologic changes consistent with rejection were noted in the transplant lobes. We conclude that the small airway obstruction noted in this model is due to operative denervation rather than to immunosuppression or rejection.

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Heart-lung and unilateral or bilateral lung transplantation are now established therapeutic options for patients with end-stage pulmonary disease. Unfortunately, the long-term survival of graft recipients may be reduced significantly by progressive small airway obstruction [1]. This complication, which has the histopathologic features of bronchiolitis obliterans, is of unknown etiology but is thought to result from chronic immune-mediated injury to the bronchiolar epithelium [2]. Pulmonary denervation has also been identified as a potential cause of small airway obstruction in the transplanted lung through bronchial hyperreactivity to cholinergic stimulation [3–6]. In previous studies, denervation of a single porcine lobe through excision and reimplantation led to small airway obstruction in the absence of immune rejection [7]. We hypothesized that neither immunosuppression nor rejection would potentiate the small airway obstruction seen in denervated porcine allograft lobes. To test this hypothesis we studied respiratory system mechanics and pulmonary histology in immunosuppressed pigs with denervated left lower lobe allografts, in pigs with denervated left lower lobe autografts, and in pigs receiving only a left upper lobectomy leaving an innervated native left lower lobe.

Material and Methods

Surgical Preparation

Twenty-two pigs, aged 10 ± 1 weeks, underwent surgical preparation and survived to complete the study protocol. Animals were not littermates and were not matched for blood type. All animals were sedated with intramuscular ketamine (20 mg/kg) and xylazine (2 mg/kg), intubated with a No. 5 endotracheal tube, and ventilated with oxygen and halothane (1% to 1.5%). Each pig underwent a left lateral thoracotomy through the fifth intercostal space.

A control group of 8 pigs had a left upper lobectomy alone. The reimplant group of 9 pigs had a left pneumonectomy after administration of sodium heparin for anticoagulation (200 U/kg). The excised lung was flushed through the pulmonary artery with heparinized normal saline solution at 4° C until the venous effluent was clear. The upper lobe was then dissected free and removed. The lower lobe was reimplanted by anastomosing first the pulmonary vein with running 6-0 absorbable monofilament suture, then the bronchus with running 4-0 absorbable monofilament suture, and finally the pulmonary artery with running 6-0 absorbable monofilament suture. A pedicle of greater omentum was brought through the diaphragm and wrapped around the bronchial anastomosis to protect the bronchus during healing. The transplant group of 5 pigs was operated on in pairs. Two animals simultaneously had a left pneumonectomy and isolation of the left lower lobe. Each animal then underwent transplantation of the allograft left lower lobe using a technique identical to that of the reimplant group. In all animals the chest was closed in layers. A chest tube was placed and was removed the following day.

Immunosuppression

All animals in the transplant group received cyclosporine (15 $\mathrm{mg \cdot kg^{-1} \cdot day^{-1}}$) orally starting the day before operation. These animals also received azathioprine (2 $\mathrm{mg \cdot kg^{-1} \cdot day^{-1}}$) orally starting immediately postoperatively and prednisone (1 $\mathrm{mg \cdot kg^{-1} \cdot day^{-1}}$) orally starting

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on the 15th postoperative day [8]. Acute rejection was diagnosed on the basis of an elevated rectal temperature of greater than 39.4°C and symptoms of respiratory distress. Episodes of acute rejection were treated with intramuscular methylprednisolone (500 mg/day for 3 days).

Study Protocol

Ten weeks after operation each pig was sedated with intramuscular ketamine (20 mg/kg) and xylazine (2 mg/kg), intubated with a No. 8 endotracheal tube, and ventilated with 100% oxygen at a tidal volume of 12 mg/kg and a rate of 15 breaths/min. Intravenous pentobarbital (10 mg/kg) and metocurine (0.4 mg/kg) were given for anesthesia and paralysis as needed. A Teflon catheter was inserted into the right carotid artery, and a balloon-tipped thermodilution catheter was inserted into the right internal jugular vein and advanced into the pulmonary artery. A tracheostomy with placement of a No. 8 cuffed tube was then performed. To isolate the left lower lobe for measurement of respiratory mechanics the right main bronchus and eparterial accessory bronchus to the right upper lobe were occluded with 6F balloon catheters placed under direct vision using a flexible bronchoscope. Each pig recovered for 15 minutes after isolation of the left lower lobe.

Measurements

Electrocardiogram, heart rate, carotid artery pressure, and pulmonary artery pressure were monitored continuously throughout each experiment. Arterial blood gas levels, pulmonary capillary wedge pressures, cardiac output, and end-tidal CO_2 level were recorded before and after every experiment. Airflow and transrespiratory pressure were measured at the tracheostomy tube using a Fleisch pneumotachometer (size No. 2, linear to 300 mL/s with a dead space of 40 mL; OEM Corp, Richmond, VA) and two differential pressure transducers (MP45-1; Validyne Engineering Corp, Northridge, CA). The signals were digitized at a rate of 15 ms with a data acquisition system (AII Devices, San Rafael, CA) and stored on diskettes (Apple IIE; Apple Computers, Cupertino, CA). These instruments were calibrated immediately before every experiment.

A standard volume history of 1,000 mL was administered with a calibrated syringe before each set of measurements. Gas flow and transrespiratory pressure were then recorded over at least five ventilated breaths. Pressure–volume loops were constructed later for each inflation by hand editing the three points of zero flow with a manual cursor and text editor program. Using the method of multiple linear regression the dynamic compliance was calculated as the slope of the line of best fit through the origin and apex of the pressure–volume loop, and the dynamic resistance was calculated from the total pressure change at 50% tidal volume divided by the flow at that volume. Functional residual capacity of the left lower lobe was then measured using the helium dilution method as previously described [7].

Histology

After measurement of left lobe mechanics the chest was opened and the heart and lungs dissected free. The pig then received a lethal dose of pentobarbital. The heart-lung block was then removed and the trachea instilled with 10% formalin at 25 cm H_2O pressure for 1 to 2 hours. Tissue slices in a sagittal plane were prepared for paraffin embedding and staining with hematoxylin and eosin.

Statistics

Measurements are reported as the mean ± standard error of the mean. Student's *t* test for unpaired samples was used to compare the two study groups, and to compare each study group with the control group. A p value of 0.05 or less was used to indicate a significant difference between measurements.

Humane Animal Care

All animals received care in facilities accredited by the American Association for Accreditation of Laboratory Animal Care and in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

Two pigs in the transplant group had an episode of acute rejection within the first 10 days after operation that responded to intramuscular methylprednisolone. All pigs appeared healthy at the time of study. At bronchoscopy no evidence of bronchial anastomotic strictures or pneumonia was evident in any of the animals. At autopsy the only apparent difference between the lobectomy lobes, the reimplanted lobes, and the transplanted lobes was the finding of somewhat denser pleural-parenchymal adhesions in the transplanted animals.

Isolated Left Lower Lobe Mechanics

Dynamic resistance in the isolated left lower lobe was significantly higher in both the transplant and reimplant groups than that observed in the control lobectomy group. No significant difference in resistance was noted between the transplant and reimplant groups (Fig 1). Dynamic compliance in the isolated left lower lobe was significantly lower in both the transplant and reimplant groups than that observed in the lobectomy group. No difference in compliance was noted between the transplant and reimplant groups (Fig 2). No significant differences were noted in the functional residual capacity of the left lower lobes in the three groups (Fig 3).

Histology

Grossly all lobes appeared normal. All vascular and bronchial anastomoses were widely patent. On hematoxylin and eosin stained sections the reimplant and lobectomy lobes had occasional areas of very mild peribronchiolar lymphoid aggregates, but there were no acute or chronic inflammatory changes, and these lobes were indistinguishable from normal porcine lung. Microscopic exami-

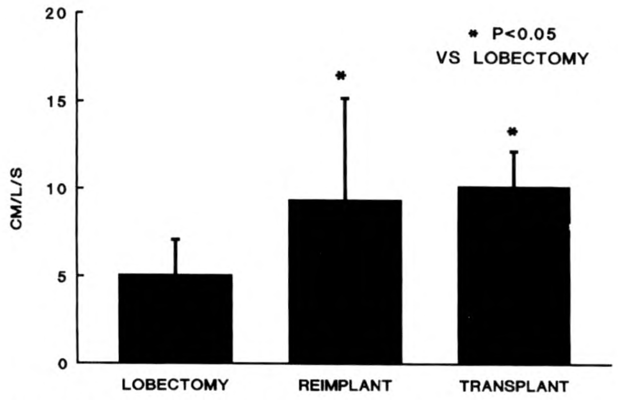

Fig 1. Dynamic resistance of the isolated left lower lobe.

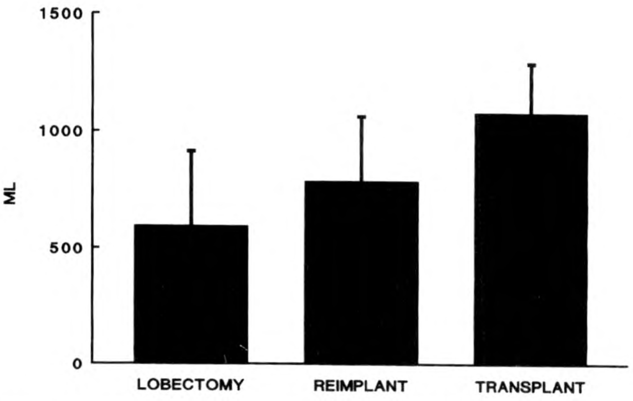

Fig 3. Functional residual capacity of the isolated left lower lobe.

nation of all left lower lobes from the transplant group showed focal, patchy, mild to moderate peribronchial and bronchiolar lymphoid infiltrates (Fig 4). These infiltrates would occasionally focally elevate the bronchial mucosa but were not associated with epithelial erosion or necrosis. The lymphoid infiltrate was mitotically active with immunoblasts and plasma cells noted, and occasionally formed nodules resembling germinal centers (Fig 5). One animal in the transplant group had, in addition to the previously mentioned changes, multiple areas of severe mixed peribronchial and peribronchiolar inflammation with mucosal erosion and necrosis (Fig 6). In this animal there were occasional areas of frank small airway destruction with replacement by chronic inflammatory cells consistent with the histologic picture of bronchiolitis obliterans (Fig 7) [9]. Despite these histologic findings the pulmonary mechanics for this animal were better than the mean in all measured categories.

Comment

The salient finding in this study is that reimplantation of an autologous lobe and transplantation of an allograft lobe result in similar changes in respiratory resistance and compliance. No significant difference in isolated left lower lobe mechanics was noted between the reimplant and transplant groups, even though histologic changes consistent with rejection were noted in the transplant group. We conclude that the changes in pulmonary mechanics noted in both groups are due to denervation and not to postoperative factors such as chronic immunosuppression or rejection.

Operative factors that could conceivably cause airway obstruction in a transplanted lung include bronchial anastomotic stricture, pulmonary edema secondary to lymphatic disruption, bronchial and bronchiolar smooth muscle hypertrophy, and increased bronchial and bronchiolar muscle tone. In this study all vascular and bronchial anastomoses were sewn with absorbable suture and remained intact and widely patent even given the somatic growth of the animal. Edema has not been evident in the reimplanted porcine lobe at 10 weeks, and in fact other investigators have demonstrated lymphatic regeneration at 3 weeks in the canine reimplanted lobe [10]. Bronchial smooth muscle hypertrophy in the reimplanted porcine lobe has not been demonstrated by immunostaining of the smooth muscle actin of the lobe [11]. We believe that operative denervation of the lung results in decreased airway diameter secondary to increased smooth muscle tone in the bronchi and bronchioles.

Respiratory resistance is a direct measure of airflow

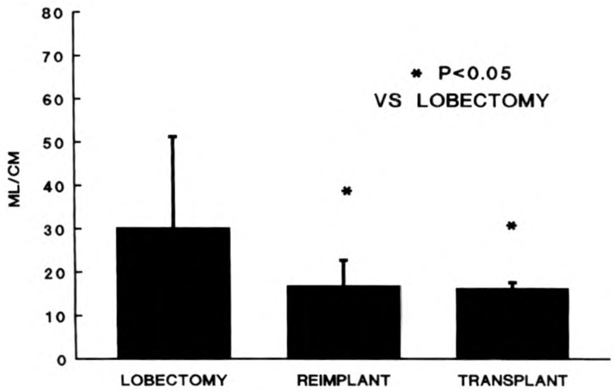

Fig 2. Dynamic compliance of the isolated left lower lobe.

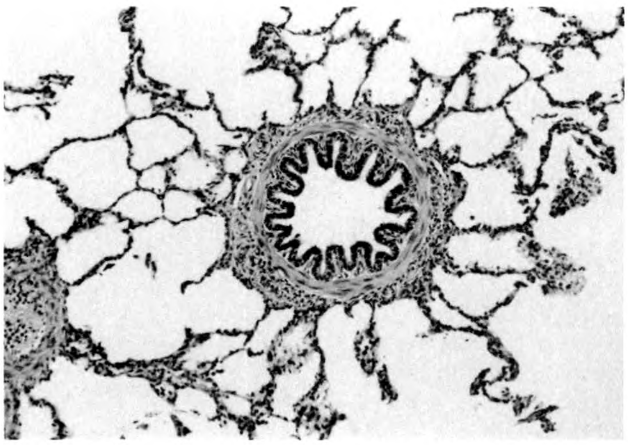

Fig 4. Mild peribronchial infiltrate in transplanted pig left lower lobe. (Hematoxylin and eosin; ×100 before 50% reduction.)

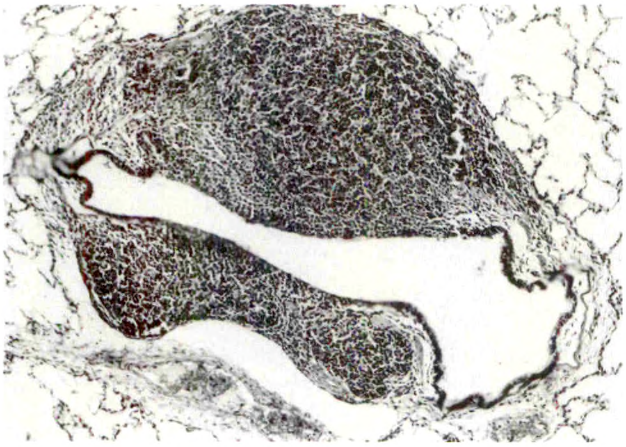

Fig 5. Lymphoid infiltrate in transplanted pig left lower lobe. (Hematoxylin and eosin; ×100 before 50% reduction.)

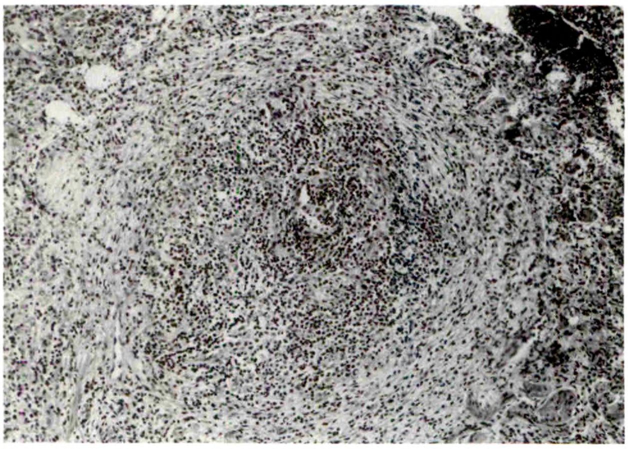

Fig 7. Small airway destruction in transplanted pig left lower lobe. (Hematoxylin and eosin; ×100 before 50% reduction.)

obstruction and is largely determined by the diameter of the lobar, segmental, and subsegmental airways. An increase in bronchial smooth muscle tone would decrease the caliber of these airways and result in increased resistance as noted in our animals. Similarly, respiratory compliance is a measure of the elastic recoil properties of the lung and depends on the physical properties of the lung tissues, the surface tension of the alveoli, and the mechanical properties of the chest wall. An increase in bronchial and bronchiolar smooth muscle tone would stiffen the lung and result in the decreased compliance noted in our animals.

In humans, nervous control of airway smooth muscle is mediated through cholinergic parasympathetic pathways that promote smooth muscle contraction and through β-adrenergic sympathetic pathways that promote smooth muscle relaxation. Stimulation of α-adrenergic receptors also results in smooth muscle contraction, but α-receptors are numerically insignificant compared with β-receptors. Possible causes of denervation-induced increased smooth muscle tone in humans include up-regulation of acetylcholine receptors, up-regulation of α-adrenergic receptors, or loss of β-stimulation.

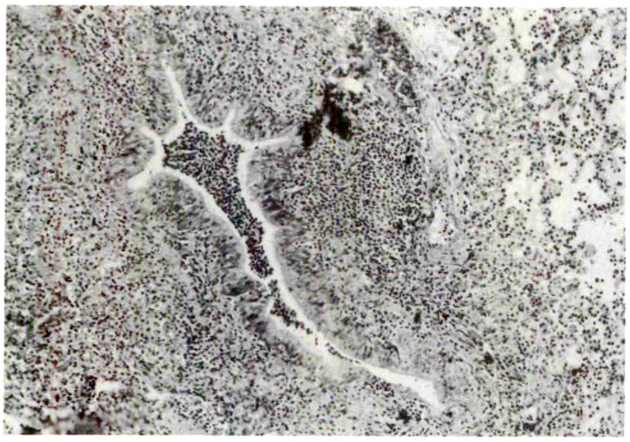

Fig 6. Severe peribronchial inflammation in transplanted pig left lower lobe. (Hematoxylin and eosin; ×100 before 50% reduction.)

Clinically the most important manifestation of small airway obstruction in human lung transplant recipients is the progressive lesion of bronchiolitis obliterans. Although bronchiolitis obliterans in transplant recipients is of unknown etiology, there are some data to suggest that it is related to rejection. The reported decrease in the incidence of bronchiolitis obliterans in an era of improved monitoring for rejection with transbronchial biopsies is thought to support the theory that rejection is the cause [12]. Furthermore, the predictive value of the primed lymphocyte test for bronchiolitis obliterans and the presence of Leu-7 positive T lymphocytes in 2 patients with lung allograft rejection and bronchiolitis obliterans support an immunologic cause for this process [13, 14]. However, the failure of most patients with biopsy-proven bronchiolitis obliterans to respond to augmented immunosuppression argues against rejection as a primary factor.

Regardless of the contribution that rejection may play in the development of progressive airway obstruction, this study may have implications for the care of lung transplant patients. Most importantly, the airway dysfunction seen in patients with bronchiolitis obliterans may be partly due to the denervation of the lung and not solely to a rejection phenomenon. The practice of using pulmonary mechanics measurements, especially forced expiratory volume in 1 second, to assess for rejection may be complicated by the patient's underlying fixed airway changes. Finally, although current bronchodilator therapy has been almost uniformly ineffective in treating lung transplant patients with established bronchiolitis obliterans, pharmacologic manipulation of airway tone may be possible to prevent the development or progression of bronchiolitis obliterans in these patients.

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Cardioprotective Effects of Nucleoside Transport Inhibition in Rabbit Hearts

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The cardioprotective effects of a nucleoside transport inhibitor, R75231, were investigated in the isolated rabbit heart. The hearts were subjected to 20 minutes of global normothermic ischemia followed by reperfusion. Before ischemia either solvent (group 1), 5 μmol/L of adenosine (group 2), or 0.64 mg/L R75231 (group 3) was added to the perfusate. Preischemic hemodynamics were not changed by treatment, except for an increase in coronary flow in the adenosine group (126% of control; $p < 0.05$). Upon reperfusion, coronary flow was depressed in the controls (72% of the preischemic control values), increased in the adenosine group (113%) and unchanged in the R75231 group (89%). Functional recovery was significantly better in the adenosine group as well as in the R75231 group as compared with the controls ($p < 0.05$). Cardiac output was 74% of the preischemic control value in the R75231 group, 67% in the adenosine group, and only 38% in the controls. Analysis of the coronary effluent after reperfusion showed a significant inhibition of rapid release of purines and a reverse of the adenosine/inosine ratio in the R75231 group as compared with the others. We conclude that R75231 has a cardioprotective effect that is probably related to accumulation of endogenous adenosine.

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Adenosine is reported to have cardioprotective properties against ischemia-reperfusion injury of the myocardium [1–3]. Although adenosine is a natural catabolite in the ischemic myocardium, it is difficult to keep adenosine in the myocardium because of its fast transport to the endothelium, its fast degradation to inosine, and its washout upon reperfusion [3, 4]. Many researchers have tried to increase the adenosine level in the myocardium by infusion of exogenous adenosine with [5] or without [6, 7] the use of an adenosine deaminase inhibitor. However these trials are far from clinical use, because of critical side effects of adenosine when in the systemic circulation (ie, hypotension) and fast uptake of adenosine by the erythrocytes. In another successful trial, its precursor, 5-aminoimidazole-4-carboxamide riboside (AICA riboside), was administered to increase the adenosine level [8, 9]. However, the mechanism of action of AICA riboside is still unclear.

Nucleoside transport inhibition to keep adenosine only at a target organ that is suffering from ischemia is an interesting concept. In this study, we evaluate the cardioprotective effects of a newly developed, specific, and potent nucleoside transport inhibitor, R75231 (2-(aminocarbonyl)-*N*-(4-amino-2,6-dichlorophenyl)-4-[5,5-*bis*(4-fluorophenyl)-pentyl]-1-piperazineacetamide; Janssen Research Foundation, Beerse, Belgium), in the rabbit heart preparation. We compare its effects with those of a continuous adenosine infusion to study the efficacy of nucleoside transport inhibition and endogenous adenosine.

Material and Methods

Experimental Design

All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" prepared by the National Academy of Sciences and published by National Institutes of Health (NIH publication No. 85-23, revised 1985).

New Zealand white rabbits weighing 2.5 to 3 kg were premedicated with 0.5 mL of Hypnorm (10 mg fluanisone/0.2 mg fentanyl per mL) intramuscularly, and anesthetized with sodium pentobarbital (Nembutal; Abbott Laboratories, North Chicago, IL) 20 mg/kg intravenously. The trachea was exposed after administration of local anesthesia (1% lidocaine) and cannulated with a tracheal tube connected to the mechanical ventilator with 100% oxygen. Heparin (1,000 units) and pancuronium bromide (0.4 mg) were administered intravenously. Through a left thoracotomy, the heart was removed rapidly by transection of all the major vessels. The heart was submerged in the cold perfusate and attached to the perfusion apparatus by the aorta, and the Langendorff perfusion was initiated immediately. During 10 minutes of Langendorff perfusion, the venae cavae were closed, the pulmonary artery and the left atrium were cannulated, a microtip manometer was introduced to the left

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ventricle through the apex, pacing wires were placed at the right ventricle free wall, and a thermister probe was placed at the interventricular septum. The heart was then converted to the working mode by initiation of left atrial perfusion. The heart was paced at 200 beats/min during the working mode. After 15 minutes of equilibration, control cardiac function (peak left ventricular pressure, positive and negative first time derivative of ventricular pressure [dp/dt], cardiac output, and coronary flow rate) was recorded. Coronary flow rate and aortic flow rate were measured by time collection of outflow, and cardiac output was calculated as the total sum of coronary flow and aortic flow. Myocardial temperature was maintained at 37°C throughout the experiment.

After the first measurement of cardiac function, the hearts were divided into three groups (each group, n = 6): group 1 hearts received perfusate including solvent, group 2 hearts received perfusate including 5 μmol/L adenosine, and group 3 hearts received perfusate including R75231 (0.64 mg/L). All hearts received one of these three types of perfusate throughout the remaining perfusion period. After 15 minutes of pretreatment, cardiac function was again recorded. After the second measurement of cardiac function, the aortic and left atrial cannulas were clamped to induce global ischemia. All hearts were subjected to 20 minutes of normothermic (37°C) global ischemia. After 10 minutes of reperfusion in the Langendorff mode, the heart was converted to the working mode for 30 minutes. The recovery of cardiac function was recorded every 10 minutes during this working mode perfusion. Coronary effluent was collected at 2, 5, 10, 20, 30, and 40 minutes of reperfusion for analysis of adenine nucleosides (adenosine, inosine) and purine bases (hypoxanthine, xanthine).

Perfusion Apparatus and Perfusate

Our perfusion apparatus is similar to that of Neely and associates [10]. Briefly, in the Langendorff mode, the heart was perfused through the cannulated aorta at a pressure of 70 mm Hg. In the working mode, the perfusate entered the cannulated left atrium and passed into the left ventricle, from which it was spontaneously ejected through the aortic cannula against the afterload of 70 mm Hg as mean pressure. Left atrial pressure was kept at 10 mm Hg. Diastolic pressure was kept greater than 45 mm Hg by regulating the volume of air in the compliance chamber at the beginning of the working mode, and this volume was kept constant throughout the experiment.

The aortic and left atrial pressure were monitored through the side arm of the aortic and left atrial cannulas with pressure transducers (Hewlett Packard Model 1280C; Hewlett Packard, Waltham, MA). The left ventricular pressure and its first derivatives (dp/dt) were monitored with a Honeywell microtransducer catheter (Dräger, Best, Netherlands) connected to a pressure amplifier (MB 01 C; Dimed, Antwerpen, Belgium).

The perfusate used was a modified Krebs-Henseleit bicarbonate buffer solution consisting of the following (in millimoles per liter): NaCl, 118; KCl, 4.7; $CaCl_2$, 2.5; KH_2PO_4, 1.2; $MgSO_4$, 1.2; $NaHCO_3$, 25; and D-glucose, 11. The perfusate was gassed with 95% oxygen and 5% carbon dioxide and prewarmed to maintain myocardial temperature at 37°C.

Assay of Effluent Purines

Purine concentrations in coronary effluent were determined by high-performance liquid chromatography exactly as described before [11]. The purines were separated isocratically on Select-B columns (Merck, Darmstadt, Germany) at a flow rate of 0.6 mL/min of monobasic ammonium phosphate (0.3 mol/L; pH, 4.0), methanol, acetonitrile, and tetrahydrofuran (97.6:1:1:0.4, vol/vol) and determined with an ultraviolet detector (Varian model 5500, 254 nm). Comparison with known amounts of reference compounds enabled easy quantitation of the different catabolites.

Statistics

All values are expressed as mean ± standard deviation. Cardiac function after reperfusion is expressed as percentage recovery of the corresponding control value before pretreatment. Paired *t* test was employed for evaluation of functional changes during the pretreatment period. Analysis of variance with repeated measurement was performed for comparison between groups, and modified *t* test with Bonferroni's correction was employed when the analysis of variance was significant ($p < 0.05$). Comparison between groups was done only against group 1 (solvent group). We employed Stat View 512+ (Brainpower) on a Macintosh Plus computer for the statistical analysis.

Results

Functional Changes During Pretreatment

In the solvent group, no significant changes of cardiac function were observed during the pretreatment period (Table 1). In the adenosine group, coronary flow increased significantly ($p < 0.05$). Neither positive nor negative inotropic effects were observed with 5 μmol/L adenosine infusion. The hearts treated with R75231 showed no significant functional changes during this preischemic period.

Cardiac Functional Recovery After Reperfusion

Functional recovery after reperfusion is shown in Table 2. In the solvent group, all parameters of cardiac function were depressed after 20 minutes of reperfusion (peak left ventricular pressure, 81% of control value; positive dp/dt, 64%; negative dp/dt, 74%; cardiac output, 38%; and coronary flow, 72%) and remained at the same level during 40 minutes of reperfusion.

In the adenosine group, positive dp/dt was significantly higher ($p < 0.05$) as compared with that of the solvent group at 20 minutes and at 40 minutes. The recovery of cardiac output was also better in this group as compared with the solvent group during reperfusion. The coronary flow remained greater than 100% of the preischemic control value and was significantly higher ($p < 0.01$) than that in the solvent group during the whole reperfusion period.

In the R75231 group, the hearts recovered as well as those of adenosine group, or even slightly better. The recovery of peak left ventricular pressure was significantly better as compared with the solvent group at 20 minutes and at 40 minutes. The recovery of positive dp/dt was also significantly better during the whole reperfusion period. The recovery of negative dp/dt was also better, but the difference was only significant at 20 minutes. The recovery of cardiac output was almost double that in the solvent group. The coronary flow recovered to the preischemic level at 20 minutes but decreased gradually during the reperfusion period.

Purine Release

During the pretreatment period, the hearts in the adenosine group extracted adenosine (223 ± 146 nmol/min/heart), and a part of the adenosine was converted and released as inosine (26 ± 22 nmol · min^{-1} · heart^{-1}) and hypoxanthine (6 ± 10 nmol · min^{-1} · heart^{-1}). At 2 minutes of reperfusion, 2 of 6 hearts extracted adenosine, whereas the others released adenosine (adenosine release was between −52 and +191 nmol · min^{-1} · heart^{-1}). Data from the adenosine group are not depicted in the figure because some hearts extracted adenosine instead of releasing it at some sampling points.

Table 1. Changes in Cardiac Function During Pretreatment[a]

Variable	Pretreatment Before	Pretreatment After	% of Control
Peak LV pressure (mm Hg)			
Solvent	100 ± 11	98 ± 8	98 ± 5
Adenosine	97 ± 9	98 ± 8	101 ± 2
R75231	93 ± 8	95 ± 10	102 ± 3
Positive dp/dt (mm Hg/s)			
Solvent	2142 ± 322	2133 ± 248	100 ± 7
Adenosine	2283 ± 121	2342 ± 153	103 ± 4
R75231	1967 ± 294	2100 ± 394	106 ± 7
Negative dp/dt (mm Hg/s)			
Solvent	1358 ± 191	1367 ± 199	101 ± 10
Adenosine	1667 ± 323	1650 ± 282	100 ± 8
R75231	1242 ± 278	1358 ± 363	109 ± 17
Cardiac output (mL/min)			
Solvent	158 ± 30	155 ± 8	100 ± 14
Adenosine	182 ± 42	190 ± 37	105 ± 7
R75231	134 ± 50	148 ± 55	111 ± 27
Coronary flow (mL/min)			
Solvent	71 ± 7	68 ± 8	96 ± 7
Adenosine	78 ± 15	98 ± 19[b]	126 ± 8[b]
R75231	74 ± 10	76 ± 12	101 ± 4

[a] All values are expressed as mean ± standard deviation. [b] $p < 0.05$ versus before pretreatment by paired t test.

dp/dt = first derivative of left ventricular pressure; LV = left ventricular.

Table 2. Percentage Recovery of Cardiac Function After Reperfusion[a]

Variable	Reperfusion Time 20 min	30 min	40 min
Peak LV pressure			
Solvent	81 ± 10	84 ± 11	81 ± 9
Adenosine	91 ± 6	92 ± 6	93 ± 7
R75231	95 ± 5[b]	94 ± 5	93 ± 3[b]
Positive dp/dt			
Solvent	65 ± 17	69 ± 19	64 ± 17
Adenosine	86 ± 8[b]	86 ± 8	87 ± 8[b]
R75231	92 ± 11[c]	92 ± 11[b]	89 ± 9[b]
Negative dp/dt			
Solvent	73 ± 18	78 ± 14	74 ± 14
Adenosine	81 ± 11	84 ± 16	85 ± 18
R75231	99 ± 15[b]	93 ± 15	91 ± 13
Cardiac output			
Solvent	39 ± 17	40 ± 18	38 ± 16
Adenosine	66 ± 19[b]	67 ± 21[b]	67 ± 16[b]
R75231	83 ± 26[c]	79 ± 27[b]	74 ± 23[b]
Coronary flow			
Solvent	72 ± 17	73 ± 18	72 ± 18
Adenosine	115 ± 17[c]	114 ± 19[c]	113 ± 14[c]
R75231	101 ± 12[c]	93 ± 9[b]	89 ± 7

[a] All values are percentage recovery of control values and expressed as mean ± standard deviation (%). [b] $p < 0.05$ and [c] $p < 0.01$ versus solvent group by analysis of variance and modified t test.

dp/dt = first derivative of left ventricular pressure; LV = left ventricular.

At 2 minutes of reperfusion, a similar amount of adenosine was released in the solvent group and the R75231 group (207 ± 143 versus 204 ± 58 nmol · min^{-1} · heart^{-1}). Adenosine release rapidly diminished in the solvent group (43 ± 39, 10 ± 8, 3 ± 3, and 2 ± 2 nmol · min^{-1} · heart^{-1} at 5, 10, 20, and 30 minutes of reperfusion, respectively), in contrast to the release in the R75231 group (164 ± 49, 99 ± 32, 19 ± 11, and 10 ± 3 nmol · min^{-1} · heart^{-1}, respectively; $p < 0.01$) (Fig 1).

A significantly larger amount of inosine ($p < 0.01$) was released at 2 minutes in the solvent group as compared with the R75231 group (422 ± 138 versus 36 ± 11 nmol · min^{-1} · heart^{-1}), and the major part of the inosine content was washed out during first 5 minutes in this group (Fig 2). In the R75231 group, however, inosine release was prolonged and there were small but significant ($p < 0.05$) differences at 10, 20, and 30 minutes of reperfusion (30 ± 9 versus 11 ± 3, 21 ± 15 versus 2 ± 2, and 8 ± 7 versus 1 ± 1 nmol · min^{-1} · heart^{-1}, respectively).

The ratio between adenosine and inosine was less than 1.0 in the solvent group (0.5 ± 0.3, 0.6 ± 0.2, and 0.8 ± 0.6 at 2, 5, and 10 minutes of reperfusion, respectively). Nucleoside transport inhibition reversed this ratio during the first 10 minutes of reperfusion (6.1 ± 2.2, 5.5 ± 2.0, and 3.7 ± 1.4, respectively; $p < 0.01$) (Fig 3). The ratio was not calculated after 10 minutes of reperfusion because

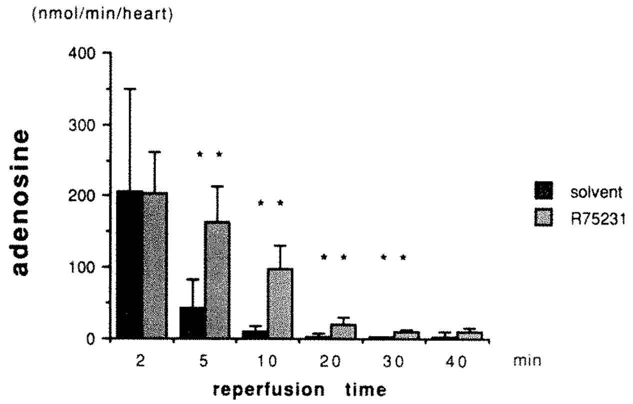

Fig 1. *Adenosine released in the coronary effluent during the reperfusion period in the solvent group and the R75231 group. (**p < 0.01 between groups by analysis of variance and modified* t *test.)*

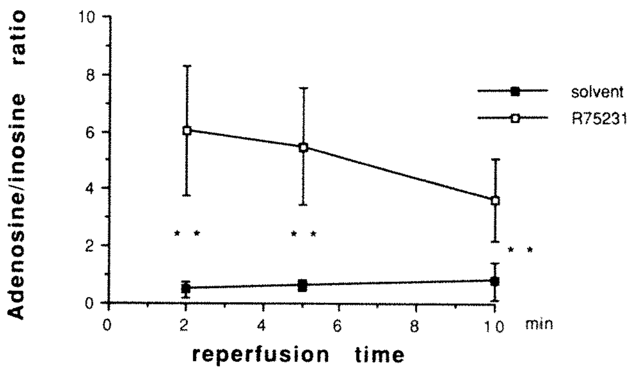

Fig 3. *Adenosine/inosine ratio in the coronary effluent during 10 minutes of reperfusion in the solvent group and the R75231 group. (**p < 0.01 between groups by analysis of variance and modified* t *test.)*

some hearts from the solvent group did not release measurable amounts of inosine after this period.

Slight release of hypoxanthine was found in the R75231 group during reperfusion, and this release of hypoxanthine in the R75231 group was significantly less ($p < 0.05$) than that of the solvent group at 2 minutes (13 ± 5 versus 55 ± 21 nmol $\cdot$ min^{-1} $\cdot$ heart^{-1}) (Fig 4).

Xanthine was not detectable in this preparation.

Comment

Many cardioprotective effects of adenosine have been reported, including (1) coronary vasodilation to improve unbalanced oxygen demand-supply relationship [1–3], (2) β-antagonism to attenuate catecholamine-induced responses [12] and thus protection of the tissue against excessive energy loss, (3) Ca^{2+}-antagonism to lower cytoplasmic ionized Ca^{2+} levels [3] and prevention of Ca^{2+}-dependent reperfusion injury, (4) suppression of neutrophil-mediated release of toxic oxygen free radicals [2, 3], (5) inhibition of platelet aggregation, probably to prevent no-reflow phenomenon [2, 3], (6) negative chronotropic effect to prevent excessive oxygen demand [1, 2], and (7) stimulation of glycolysis [1].

In this study, we infused adenosine at a concentration of 5 μmol/L. We chose this concentration from our dose-response study (unpublished data), in which 5 μmol/L was the maximum concentration without negative inotropic effects during pacing conditions. Thus, no significant functional changes were observed during pretreatment with adenosine except for a significant increase in coronary flow due to coronary vasodilation. After ischemia and reperfusion, 5 μmol/L of exogenous adenosine induced higher coronary flow as compared with the control series, and improved functional recovery. These results are in agreement with those of Ely and co-workers [6] using rat hearts, although the concentration of adenosine is much lower in our experimental set-up (5 versus 100 μmol/L).

It is well known that during ischemia adenosine triphosphate is catabolized to adenosine diphosphate, adenosine monophosphate, and adenosine. Adenosine is formed both extracellularly and intracellularly by 5′-nucleotidase. The adenosine is transported into the endo-

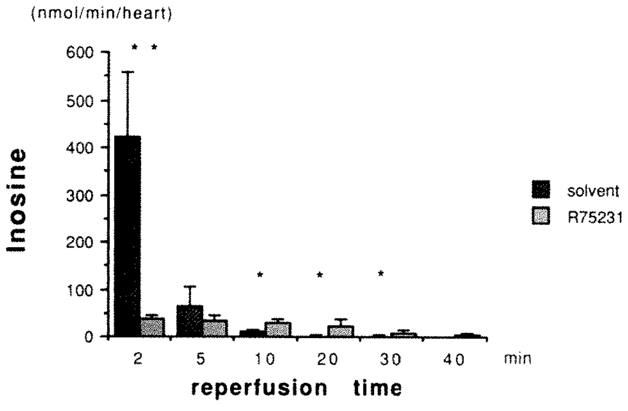

Fig 2. *Inosine released in the coronary effluent during the reperfusion period in the solvent group and the R75231 group. (*p < 0.05 and **p < 0.01 between groups by analysis of variance and modified* t *test.)*

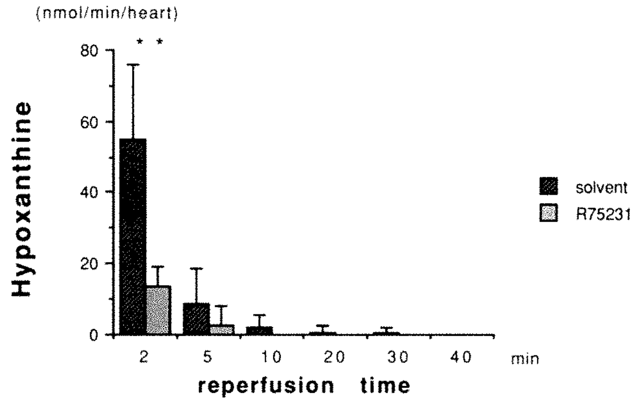

Fig 4. *Hypoxanthine released in the coronary effluent during the reperfusion period in the solvent group and the R75231 group. (**p < 0.01 between groups by analysis of variance and modified* t *test.)*

thelial cells by membrane-located nucleoside transporter and is deaminated to inosine by adenosine deaminase in the endothelial cells. Because the equilibrium constant of adenosine deaminase strongly favors the formation of inosine, the major part of adenine nucleosides is inosine. Upon reperfusion, adenosine can be released unchanged to the capillaries by diffusion, or deaminated to inosine by adenosine deaminase, or phosphorylated to adenosine monophosphate by adenosine kinase [13]. In these ways, endogenously formed adenosine is lost from the myocardium soon after reperfusion. Because approximately three times as much adenosine is taken up by the endothelial cells as by paracellular diffusion [13], most adenosine release probably occurs through the endothelial cells.

There is an increasing interest in maintaining endogenous adenosine in the target organ because of critical side effects of systemic infusion of adenosine. There are at least three other pharmacological methods to increase endogenous adenosine in the tissue. One alternative is adenosine deaminase inhibition, which inhibits further degradation of adenosine to inosine. However, this drug does not inhibit rapid release of adenosine from the tissue after restoration of flow. Another alternative is AICA riboside, which increases the endogenous adenosine level after ischemia and reperfusion [8, 9]. However, further investigations are required to clarify its mechanism for increasing endogenous adenosine. The last alternative is nucleoside transport inhibition, which inhibits transportation of adenosine into endothelial cells where it is degradated to inosine and hypoxanthine [4]. There has been increasing evidence proving the cardioprotective effects of nucleoside transport inhibition (eg, mioflazine, soluflazine) [3, 4, 14–17].

R75231 is a newly developed nucleoside transport inhibitor that has many interesting properties compared with other drugs in this class: specific and strong affinity to the nucleoside transporter, long duration of action, and good absorption when given orally [18]. When given under normoxic condition, this drug showed no significant functional changes. After ischemia and reperfusion, the hearts treated with this drug recovered as well as the hearts receiving 5 μmol/L exogenous adenosine continuously. From the results of the coronary effluent, strong nucleoside transport inhibition of this drug is apparent: reversal of the adenosine/inosine ratio was combined with inhibition of rapid release of nucleosides into the circulation. Because the release of adenosine into the coronary effluent is reflected by the presence of adenosine in the interstitial fluid [17], this long-lasting release of adenosine may be the result of maintenance of endogenous adenosine in the interstitial fluid. In the control group, accumulated adenosine is washed out by capillary flow either through the endothelial cells (nucleoside transporter) or through paracellular diffusion, or adenosine is deaminated to inosine. In the drug-treated group, on the other hand, adenosine may be slowly released by paracellular diffusion. This "storage" of endogenous adenosine in the myocardium by nucleoside transport inhibition resulted in a protective effect similar to that of a continuous infusion of 5 μmol/L exogeneous adenosine (maximum dose in this preparation).

Although the exact protective mechanism of endogenous adenosine in this study is still unclear, it is unlikely that it is due to inhibition of platelet aggregation and attenuation of neutrophil-mediated injury because we used a crystalloid-perfused system. However, we cannot neglect these mechanisms in the protection against ischemia-reperfusion myocardial damage in vivo, and further investigations are required for evaluation of this mechanism.

Prevention of the no-reflow phenomenon may be one of the mechanisms of protection. Previously, we reported the prevention of no-reflow phenomenon in the canine heart using another nucleoside transport inhibitor, mioflazine [15]. In that report, we discussed that prevention of adenosine catabolism and prevention of inosine and hypoxanthine accumulation may influence the development of reduced reflow rather than pure vasodilation. However, from this study, we cannot conclude whether this prevention of no-reflow is a direct effect of endogenous adenosine or an indirect effect such as protection of endothelial cells.

Other mechanisms such as β-antagonism, Ca^{2+}-antagonism, and negative chronotropic effects may play a minor or no role at all in this protection because of the lack of effect at the dose used and because of the pacing condition. Stimulation of glycolysis is also theoretically possible.

Prevention of hypoxanthine accumulation is another possible mechanism for protection. Hypoxanthine and its further catabolite, xanthine, are known as substrates for xanthine oxidase, from which toxic free radicals are released to cause myocardial damage [19, 20]. This mechanism is unlikely in the rabbit heart preparation, in which, as in humans, no xanthine oxidase activity was found [21].

In conclusion, the nucleoside transport inhibitor R75231 maintains endogenous adenosine in the ischemia-reperfused myocardium and enhances the ischemia tolerance of the myocardium.

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Transesophageal Echocardiography in Patients With Mechanical Circulatory Assistance

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Transesophageal echocardiography was used to assess myocardial function and to detect complications after mechanical circulatory support for 8 patients with cardiogenic shock. In 3 of 8 patients, serial transesophageal echocardiography documented improvement of systolic ventricular function, and it was possible to wean these 3 patients from the ventricular assist device. In all patients, transesophageal echocardiography added clinically important information including the extent of left and right ventricular dysfunction (6 patients), presence of atrial or ventricular thrombus (5 patients), presence of pericardial effusion or clot (2 patients), and verification of the position of the intravascular device (1 patient). Thus, transesophageal echocardiography may provide clinically useful information regarding both the underlying cardiac disease and potential complications from the mechanical circulatory assistance.

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Mechanical circulatory assist devices are used with increasing frequency for the treatment of cardiogenic shock after either open heart operations or acute myocardial infarction [1–3]. A variety of technical and medical problems occur with these devices in this group of severely hemodynamically compromised patients. Conventional transthoracic bedside echocardiography has proved to be of value in detecting cardiovascular complications and in monitoring both regional and global left ventricular function in these patients [4–6]. Transthoracic ultrasound, however, is frequently technically difficult and thus of limited value in multiply instrumented patients. In such situations, transesophageal echocardiography (TEE) may provide important additional information for the diagnosis of complications as well as for the assessment of regional and global ventricular function [7]. Our experience with the use of TEE in 8 patients supported by mechanical devices for cardiogenic shock after either a cardiac operation or acute myocardial infarction is reported.

Material and Methods

Echocardiography

Standard conventional transthoracic echocardiograms were attempted in each patient, and one to five TEE studies were also performed. Echocardiographic studies were done on request of the cardiologist or cardiovascular surgeon treating the patient. No formal prospective protocol was followed. Transesophageal echocardiography was performed with a 5-MHz transducer attached to the tip of a commercially available gastroscope (Model 21362A; Hewlett-Packard). All views were recorded on videotape for further analysis by two independent observers. Two patients with only one TEE each were followed up by transthoracic technique. Transesophageal examinations were performed with the circulatory support device either turned off or, if this was not tolerated, reduced to the lowest possible level of assistance to maintain the mean blood pressure between 60 and 70 mm Hg. The ejection fraction of each ventricle was estimated visually at the time the TEE study was performed [8, 9]. No procedure-related complications were encountered from the insertion of the TEE transducer.

Patients

Between April 1989 and May 1990 8 patients with mechanical circulatory support provided by a variety of devices including centrifugal pumps and the axial flow Hemopump (Nimbus Medical, Rancho Cordova, CA) were studied (Table 1). There were 7 men and 1 woman, 40 to 71 years of age (mean age, 57 ± 13 years). Two patients were supported in a biventricular configuration, whereas isolated right (1) or left (1) ventricular assist devices were used in 2 others. Four patients were treated with the axial flow Hemopump system positioned in the left ventricle. In 2 patients intraaortic balloon counterpulsation was operating simultaneously with the use of ventricular support devices. Five of 8 patients needed circulatory assistance immediately after an open heart operation (coronary artery bypass grafting, 3; replacement of the aortic valve and ascending aorta, 1; cardiac transplantation, 1). The other 3 patients were supported for cardiogenic shock associated with an acute myocardial infarction. Three patients received coronary angioplasty of the infarct-related vessel, in 1 patient immediately followed by a coronary artery bypass operation.

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Table 1. Patient Characteristics

Patient No.	Age (y)	Sex	Indication for Mechanically Assisted Circulation	Device(s)
1	53	F	Heart transplant/RV failure	RVAD, IABP
2	71	M	AMI/LV failure	HP
3	46	M	AMI, PTCA/LV failure	HP, IABP
4	69	M	CABG/LV + RV failure	BVAD, IABP
5	40	M	AMI, PTCA/LV failure	HP, IABP
6	56	M	CABG/LV + RV failure	BVAD, IABP
7	66	M	CompAVG + CABG/LV failure	LVAD, IABP
8	58	M	AMI, PTCA, CABG/LV failure	HP, IABP

AMI = acute myocardial infarction; BVAD = biventricular assist device; CABG = coronary artery bypass graft; CompAVG = composite aortic valve graft; HP = Hemopump; IABP = intraaortic balloon pump; LVAD = left ventricular assist device; PTCA = percutaneous transluminal coronary angioplasty; RVAD = right ventricular assist device.

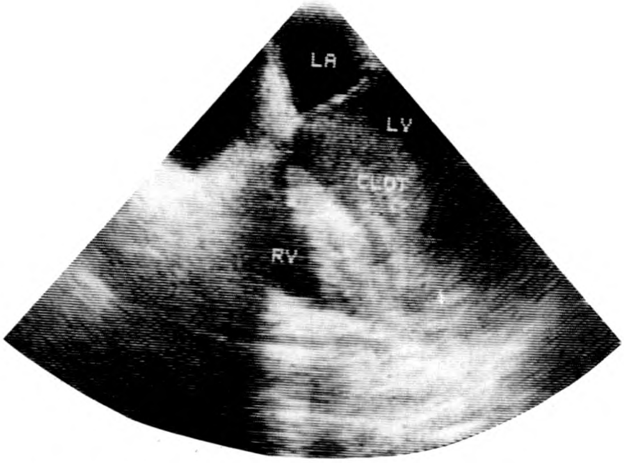

Fig 1. (Patient 7.) Transesophageal echocardiogram, modified four-chamber view, shows large thrombus (CLOT) *in the left ventricle* (LV) *extending from the apex toward the outflow tract.* (LA = *left atrium;* RV = *right ventricle.)*

Results

Conventional transthoracic echocardiographic imaging was technically satisfactory in only 3 of the 8 patients. Using the transesophageal approach, global and regional systolic ventricular function could be evaluated in all patients (Table 2). Three patients in whom left or right ventricular ejection fraction and regional wall motion was found to be improved by echocardiography were subsequently weaned from their circulatory devices. All patients without improvement in ventricular function died. The degree of improvement of ventricular ejection fraction was small (less than 10 percentage points) in all cases. One of the patients who was successfully weaned from the assist device later had development of adult respiratory distress syndrome and died. In patients with right ventricular dysfunction (acute cardiac transplant graft dysfunction or inferior wall myocardial infarction), TEE provided important information regarding the extent of right ventricular dysfunction. In 1 patient, severe concentric left ventricular hypertrophy was discovered. This information allowed for more appropriate control of the circulatory assist device and altered the weaning strategy to optimize left ventricular filling.

Complications relating to either the primary disease process or the therapeutic intervention could also be assessed by TEE. A pericardial effusion without tamponade was noted in 2 patients. Left ventricular thrombi were noted in 5 patients, and a left atrial thrombus was seen in 1 patient. Thrombi were verified in the 3 patients in whom autopsy was performed. A large thrombus extending from the left ventricular apex toward the outflow tract obstructed the prosthetic aortic valve in 1 of these patients (Fig 1). In another patient with a Hemopump, the cineangiographic studies revealed an eccentric positioning of the device across the aortic valve, suggesting an early

Table 2. Transesophageal Echocardiographic Findings, Hemodynamics, and Outcome

Patient No.	Max EF[a]		EF and WMA Improved	Thrombus	PE	CI[a] (L/min)	PCW (mm Hg)	CVP (mm Hg)	Weaned	Survived
	LV	RV								
1	NL	~0.30	Yes	No	Yes	2.4	18	26	Yes	Yes
2	~0.35	NL	Yes	No	Yes	1.8	29	16	Yes	Yes
3	~0.25	NL	Yes	No	No	2.1	16	11	Yes	No
4	~0.35	~0.20	No	Yes	No	2.6	17	9	No	No
5	~0.20	NL	No	Yes	No	2.2	18	5	No	No
6	~0.35	~0.25	No	Yes	No	2.0	13	19	No	No
7	~0.20	NL	No	Yes	No	2.0	21	16	No	No
8	~0.20	NL	No	Yes	No	1.7	23	17	No	No

[a] Measured without mechanical ventilator support or under lowest tolerable level of assistance.

CI = cardiac index measured by thermodilution; CVP = central venous pressure; Max EF = maximal ejection fraction estimated by transesophageal echocardiography; LA = left atrium; LV = left ventricle; NL = normal; PCW = pulmonary capillary wedge pressure; PE = pericardial effusion; RV = right ventricle; WMA = wall motion abnormality.

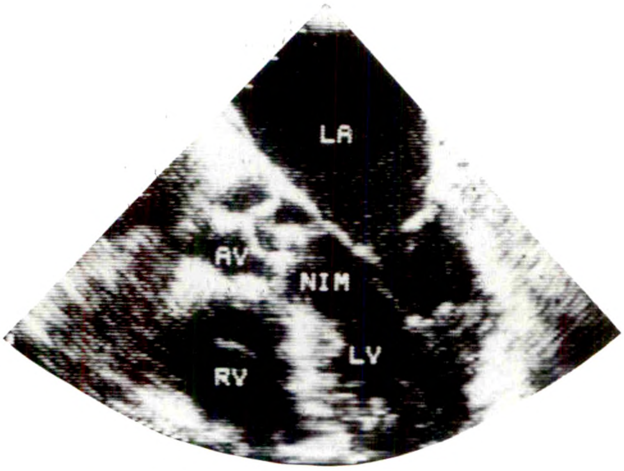

Fig 2. (Patient 3.) Transesophageal echocardiogram, modified four-chamber view, shows Nimbus Medical Hemopump catheter tip (NIM) *visible in the left ventricular outflow tract. The aortic valve* (AV) *is intact.* (LA = *left atrium;* LV = *left ventricle;* RV = *right ventricle.)*

device-related aortic dissection. Transesophageal echocardiography confirmed the appropriate placement of the device without aortic dissection (Fig 2). Aortic regurgitation as demonstrated by color Doppler interrogation was at most mild in patients with the Hemopump in place and did not increase with the addition of intraaortic balloon counterpulsation. Transesophageal echocardiography was valuable in excluding valvular vegetations in 1 patient with recurrent unexplained fevers.

Comment

Transesophageal echocardiography is playing an increasingly important role in the perioperative and intensive care management of critically ill patients [10–13]. The high quality of transesophageal images is especially helpful after cardiac operations when technically adequate transthoracic echocardiograms may not be obtainable.

Our series suggests that the primary indication for the use of transesophageal imaging is the detection of intracardiac thrombus, which was found in 5 of the 8 patients studied. In 1 patient, the intraventricular thrombus was massive and extended into the left ventricular outflow tract across the aortic valve prosthesis. The detection of left ventricular thrombus is particularly important in the setting of left ventricular support using the Hemopump rendering the patient at risk for arterial embolism.

A second indication for TEE in the setting of ventricular assist devices is the exclusion of pericardial tamponade. Two patients had at least moderately large pericardial effusions in this series. Because hemodynamic monitoring is generally not helpful in detecting pericardial tamponade in patients with ventricular assist devices, high-quality echocardiography is useful in these patients to rule out cardiac compression before weaning them from the device.

This study provides preliminary data on the third indication for TEE, ie, serial assessment of ventricular function. Reliable assessment of both regional and global left ventricular function has been demonstrated using TEE [14–16]. Weaning from the circulatory assist device was possible in the 3 patients in whom the ejection fraction and wall motion abnormality of the left or right ventricle were noted to improve. Among those in whom serial echocardiographic studies failed to document return of ventricular function during mechanical assisted circulation, there were no survivors [6, 7]. We hypothesize that weaning from a ventricular support device may be contraindicated in the face of continued severe left ventricular dysfunction on TEE. Although we believe that the weaning process should be governed by clinical and hemodynamic variables rather than echocardiographic findings, the results of the TEE studies provide additional information that might allow for better stratification of prognosis in this group of critically ill patients [6, 7, 17].

In this setting, TEE may also be useful in defining catheter position or catheter-related complications [4, 18, 19].

This study has some limitations. The assessment of changes in ventricular function was done visually, and no formal protocol with regard to timing of the echocardiogram, level of circulatory support, or loading conditions was used. These limitations render the echocardiographic assessment of subtle changes in ventricular function prone to error. Furthermore, this study did not entail a prospective comparison between the transthoracic and transesophageal imaging modalities.

In conclusion, transesophageal imaging appears to be the procedure of choice in the management of patients on circulatory assistance. When TEE is coupled with complete hemodynamic assessment, management and prognostic decisions can be facilitated.

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Vascular Reactivity of Human Internal Mammary and Gastroepiploic Arteries

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Patency rates of bypass graft conduits are thought to be influenced by the determinants of vascular tone. This study has comparatively examined the response of the human internal mammary and gastroepiploic arteries to potassium, noradrenaline, dopamine, 5-hydroxytryptamine, thromboxane, and histamine. The response to potassium was significantly greater in the gastroepiploic artery (E_{max} = 79.5 ± 9.6 mN) than in the internal mammary artery (E_{max} = 27.0 ± 6.4 mN). Dose-related constrictions were observed in both vessels to all agonists except histamine, which was ineffective in the gastroepiploic artery. Noradrenaline and dopamine produced comparable dose-related constrictions in each vessel, with similar EC_{50} and E_{max} (expressed as a percentage of potassium response) values, but 5-hydroxytryptamine (E_{max}, gastroepiploic = 10.8% ± 1.9%; internal mammary = 71.8% ± 21.2%) and thromboxane (E_{max}, gastroepiploic = 116.7% ± 4.0%; internal mammary = 169.6% ± 19.4%) were more efficacious in their constriction of the mammary artery; the potencies were similar. We conclude that there is a heterogeneity of response to some vasoconstrictors between the human internal mammary and gastroepiploic arteries. The internal mammary artery may be more predisposed to events that initiate vasospastic disorders.

(Ann Thorac Surg 1991;52:1310–4)

The determinants of vascular tone could have an important influence on graft performance after cardiac revascularization procedures [1]. The increased patency rates of internal mammary artery, (IMA) conduits compared with saphenous vein have been partly attributed to a superior capacity of arterial endothelial cells to secrete endogenous dilators [2, 3]. This dilatory protection guards against constrictors released locally from aggregating platelets and other circulating blood cells [4]. Bypass graft vascular tone can therefore be influenced by endothelial function and the response to constrictor substances encountered locally as in platelet derivatives, or globally as in perioperative and postoperative drug administration. The vascular reactivity of a specific vessel will depend on the presence, density, and distribution of different receptor types and subtypes.

For editorial comment, see page 1211.

The most commonly used alternative arterial vessel is the gastroepiploic artery (GEA), and results from short- [5, 6] and medium-term [7] experience indicate patencies comparable with those of the IMA. Functional studies have revealed it to possess a very reactive endothelium in terms of ability to release endothelial-derived nitric oxide [8]. It has also been reported to be pharmacologically equivalent to the IMA in its constrictor response to ergonovine, 5-hydroxytryptamine (5-HT) and phenylephrine [9]. To more fully characterize its contractile profile, we have compared the effect of several naturally occurring mediators of vascular tone and drugs commonly used during the perioperative period on human IMA and GEA.

Material and Methods

Tissue

Internal mammary and gastroepiploic arteries were harvested from multiorgan donors or surplus to requirement at coronary artery bypass grafting. Material from 29 patients (10 to 65 years old; mean age, 31.8 years) was used. Vessels were cleaned of excess tissue and stored in modified Tyrode's solution if not used immediately (Tyrode's solution composition in millimoles per liter: NaCl, 136.9; $NaHCO_3$, 11.9; KCl, 2.7; NaH_2PO_4, 0.4; $MgCl_2$, 2.5; $CaCl_2$, 2.5; glucose, 11.1; disodium ethylenediaminetetraacetic acid, 0.04).

Protocol

Vessel segments (3 to 5 mm in length, 153 in total) were set up as isolated organ bath preparations. Segments were suspended in 5-mL organ baths containing modified Tyrode's solution gassed with 95% oxygen and 5% carbon dioxide. The temperature was maintained at 37°C. Changes in vessel wall tension were measured by means of intraluminal L-shaped metal hooks, one of which was fixed and the other attached to a Grass FTO-3C strain gauge transducer connected to a Gould polygraph (Gould Electronics Ltd, Cleveland, OH).

A preload of 50 mN was applied to each segment, followed by a stabilization period of 40 to 60 minutes during which "relaxing-out" occurred and a steady baseline attained. To assess tissue viability and maximum

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smooth muscle contractile response, 90 mmol/L potassium chloride was applied; a very small number of segments were unresponsive to potassium and were therefore discarded. After washout of potassium the segments were again allowed to equilibrate to their baseline.

To fully investigate the smooth muscle response of IMA and GEA, cumulative doses of potassium (10 to 90 mmol/L) were applied, in duplicate, to segments from at least 4 patients. In the remaining segments, the response to other constrictors was tested. Additive doses (½ log units) of each drug were applied: noradrenaline, dopamine, 5-HT, histamine (10^{-8} to 10^{-4}mol/L), and the thromboxane analogue U46619 (10^{-10} to 10^{-4}mol/L). Each dose-effect was allowed to reach a stable plateau (usually 5 to 7 minutes) before addition of a higher concentration, and vessel segments were subjected to one drug treatment only. Dose-response curves were then constructed for each agonist.

Data Analysis

Absolute responses were expressed in millinewtons. Comparative responses between the blood vessels were analyzed by expressing constrictions as a percentage of the maximum force generated by 90 mmol/L potassium. Computed estimates of the effective concentration of constrictor that caused 50% of maximal contraction force (EC_{50}) were obtained by fitting individual sets of concentration-effect curve data to the following expression: $E = \alpha[A]^m/([A_{50}]^m + [A]^m)$, where E is the response, α the upper asymptote, A_{50} the EC_{50} concentration, A the concentration, and m the slope parameter. From these fitted equations, the mean EC_{50} value ± the standard error of the mean was calculated. The mean maximum response (E_{max}) value for each constrictor was obtained from the mean dose-response curves.

Statistical analysis was performed using an unpaired t test with significance at values of p less than 0.05, and n represents the number of patients.

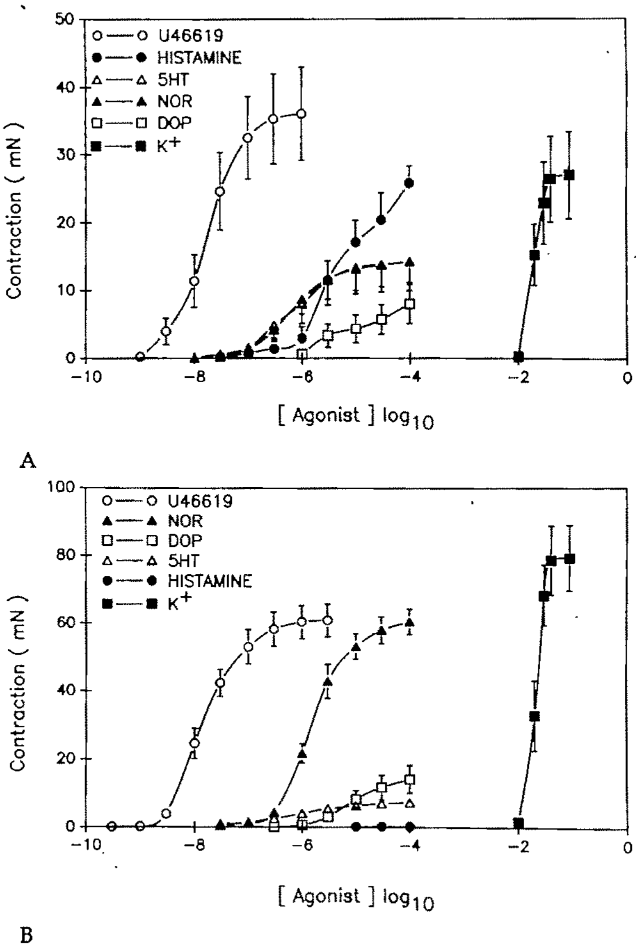

Fig 1. (A) Contractile effects of U46619 (n = 8), noradrenaline (NOR; n = 6), dopamine (DOP; n = 6), 5-hydroxytryptamine (5HT; n = 6), histamine (HIST; n = 6), and potassium chloride (K⁺; n = 4) in segments of human internal mammary artery. (B) Contractile effects of U46619 (n = 6), histamine (n = 6), 5-hydroxytryptamine (n = 6), noradrenaline (n = 6), dopamine (n = 6), and potassium chloride (n = 4) in segments of human gastroepiploic artery. Note difference in Y-axis scale between (A) and (B).

Results

There was no difference in response between vessels procured from multiorgan donors and those from bypass surgical procedures. Constrictor effects were not age-related, neither did they correlate with storage of vessels in modified Tyrode's solution for up to 8 hours. Vessels obtained in this manner have been demonstrated to be functionally intact as assessed by preserved endothelial responses [8].

Internal mammary artery (n = 4) and GEA (n = 4) responded to potassium applied cumulatively over 1 log unit (10 to 90 mmol/L) with similar mean EC_{50} values ($p > 0.05$). However, the mean maximum response in the IMA was 27.0 ± 6.4 mN compared with 79.5 ± 9.6 mN in the GEA ($p < 0.05$) (Fig 1). Segments of IMA and GEA constricted in a dose-dependent manner to all substances used except for histamine, which had no effect in the GEA (Fig 1). In the IMA, U46619 was the most potent (lowest EC_{50} value) constrictor ($p < 0.05$), followed by noradrenaline, 5-HT, dopamine, and histamine; U46619 was also the most efficacious (highest E_{max} value; $p < 0.05$). In the GEA, U46619 was also the most potent ($p < 0.05$), followed by 5-HT, noradrenaline, and dopamine; U46619 was also the most efficacious ($p < 0.05$). The EC_{50} and E_{max} values for all constrictors in both vessels are shown in Table 1.

The dose-response curves to noradrenaline and dopamine were similar in the IMA (n = 6) and GEA (n = 6) (Fig 2), with comparable mean EC_{50} and E_{max} values (see Table 1). However, 5-HT and U46619 produced more powerful constriction in the IMA (n = 6) than in the GEA (n = 6), as compared with the maximum potassium response (Fig 3), with significantly different mean E_{max} values ($p < 0.05$; see Table 1); the mean EC_{50} values were similar. Histamine produced an effect only in the IMA (Fig 4).

The absolute responses in millinewtons were greater in the GEA for all constrictors tested except histamine.

Table 1. EC_{50} and E_{max} Values of Constrictor Agonist Effects and Levels of Significance Between Human Internal Mammary and Gastroepiploic Arteries[a]

	EC_{50}			E_{max}		
Agonist	IMA	GEA	*p* Value	IMA	GEA	*p* Value
U46619	$2.7 \pm 0.6 \times 10^{-8}$	$2.9 \pm 0.5 \times 10^{-8}$	NS	169.6 ± 19.4	116.7 ± 4.0	0.01
5-HT	$1.2 \pm 0.3 \times 10^{-6}$	$1.3 \pm 0.9 \times 10^{-6}$	NS	71.8 ± 21.2	10.8 ± 1.9	<0.01
NA	$4.3 \pm 1.4 \times 10^{-7}$	$2.1 \pm 0.9 \times 10^{-6}$	NS	61.3 ± 11.2	91.0 ± 5.2	NS
DA	$0.1 \pm 4.2 \times 10^{-6}$	$6.1 \pm 1.7 \times 10^{-6}$	NS	33.9 ± 13.5	25.1 ± 13.5	NS
Hist	$1.2 \pm 0.8 \times 10^{-5}$	. . .	. . .	124.8 ± 11.7	. . .	<0.001

[a] EC_{50} values are expressed as molar concentrations, E_{max} values as percent of maximum potassium response. All values are means ± standard error of means, and significance is assigned at p less than 0.05.

DA = dopamine; GEA = gastroepiploic artery; Hist = histamine; 5-HT = 5-hydroxytryptamine; IMA = internal mammary artery; NA = noradrenaline; NS = not significant.

Comment

This study has demonstrated that the human IMA and GEA differ in their response to a range of constrictor agents that may be encountered in the circulation either as endogenous mediators of vessel tone or as a result of drug therapy associated with cardiac surgical procedures.

The magnitude of potassium response was greater in the GEA than in the IMA, and in absolute terms (millinewton response) the GEA responded more vigorously to all agonists used except histamine. Although the potencies of each agonist were similar between vessels, the maximum responses were significantly greater in the IMA when compared against its potassium response. The maximum response to potassium is an index of smooth muscle contractility against which receptor-mediated constriction can be compared. Our study therefore demonstrates that the degree of receptor-mediated constriction is greater in the IMA than in the GEA.

The constrictions to dopamine and noradrenaline were equivalent in both arteries. This supports previous work that reported a homogeneous response to catecholamines in human IMA and GEA [9] and implies that these arteries are susceptible to α-receptor stimulation by circulating amines. It also substantiates the documented constrictor effect of dopamine in human GEA reported by Toda and associates [10]; however, we did not test for the dilatory action of dopamine as previously reported [10]. The present study clearly identifies dopamine and noradrenaline as potent vasoconstrictors and therefore has important implications for their perioperative administration.

The efficacies of the thromboxane analogue U46619 and 5-HT were significantly greater in the IMA than the GEA and were similar to those previously documented in the IMA [11]. In the GEA, U46619 also caused marked constriction, but the response to 5-HT was negligible. It has been reported that these arteries are equivalent in their response to 5-HT [9]. We could not obtain comparable constriction in the GEA unless the endothelium was removed (unpublished observation). Thromboxane and 5-HT are released by aggregating platelets, and the IMA and GEA will therefore be susceptible to local effects of these endogenous constrictors.

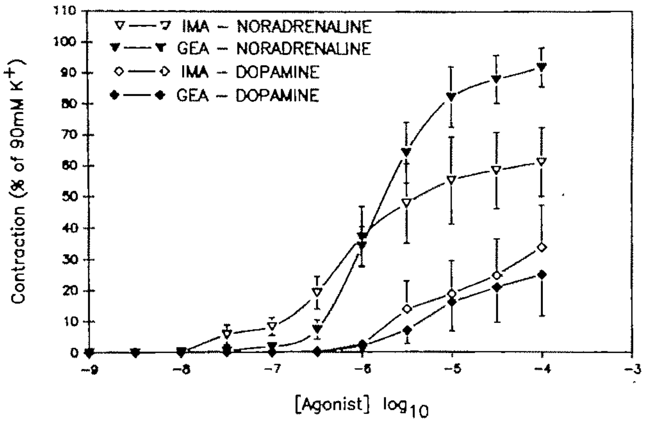

Fig 2. Comparison of the contractile responses (expressed as percent of maximum potassium constriction) of human internal mammary artery (IMA; n = 6) and gastroepiploic artery (GEA; n = 6) to noradrenaline and dopamine.

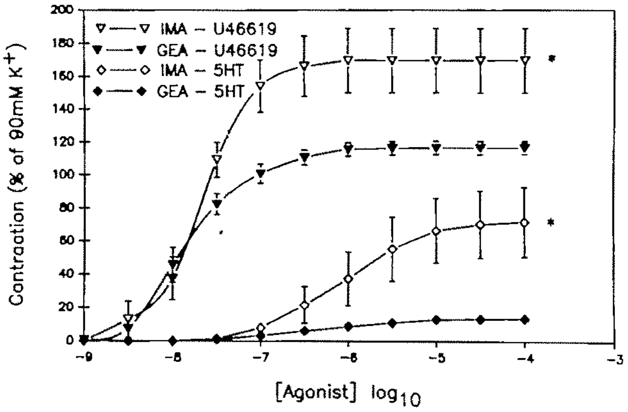

*Fig 3. Comparison of the contractile responses (expressed as percent of maximum potassium constriction) of human internal mammary artery (IMA; n = 6) and gastroepiploic artery (GEA; n = 6) to U46619 and 5-hydroxytryptamine (5HT). (*denotes significance difference [p < 0.05] between vessel type.)*

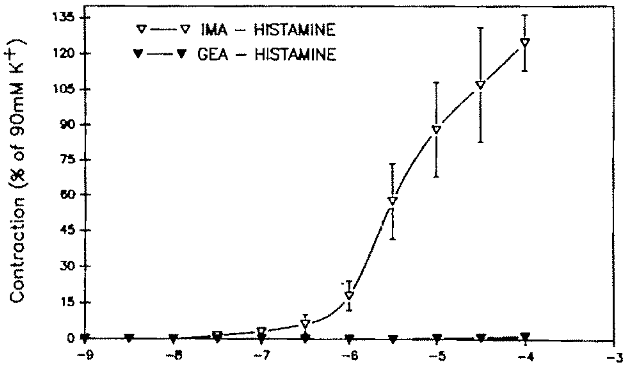

Fig 4. Comparison of the contractile responses (expressed as percent of maximum potassium constriction) of human internal mammary artery (IMA; *n* = 6) *and gastroepiploic artery* (GEA; *n* = 6) *to histamine.*

Histamine has been shown to produce coronary spasm in some patients with variant angina [12] and in miniature swine fed a high-cholesterol diet [13]. Also, autopsy has shown that the number of mast cells releasing histamine is increased in coronary artery segments involved in spasm [14]. Endogenous histamine has therefore been proposed as one candidate for the genesis of vasospasm. The heterogeneity of response of different blood vessels to histamine is well documented [15, 16]. In human coronary arteries, the response to histamine can be dilatory or constrictive, depending on the pathophysiological state of the vessels, presence or absence of variant angina, and the concentration of histamine [17–19]. In the IMA, histamine has been reported to be relaxatory at low concentrations, a trend that is converted to constriction at higher concentrations [4]. We observed constriction in the IMA at similar histamine concentrations, but did not test for its dilatory effect. However, in the GEA histamine had no constrictor properties even at high concentrations.

The disparity in response of the IMA and GEA to 5-HT and histamine may lie in the reactivity of the endothelium, and it may be that the GEA endothelium offers greater protection against these constrictors than does the endothelium of the IMA. Preferential endothelial modulation of the response to 5-HT and histamine has already been described between the IMA and saphenous vein [4] attributable to different activation of the L-arginine and cyclooxygenase pathways [20]. It appears from our experiments that this feature of the endothelium also applies to comparison of different arteries; it is also relevant that the endothelium of the GEA has an increased capacity to produce vasodilators and cyclic guanosine monophosphate, the second messenger eliciting smooth muscle relaxation after production of nitric oxide [8].

There are certain limitations in the extrapolation of the results of this study to the clinical situation. The organ bath dose-regimens cannot be used as a direct comparison with drug/mediators that circulate in vivo, but they provide important information regarding vessel differences in profile of the full dose-response curve. Such data are essential for the future characterization of the receptor subtypes involved in the genesis of vasoconstriction, which may have clinical implications for selective antagonist intervention when faced with insufficient IMA/GEA flow in the early postoperative phase. Changes in tensile strength could have important implications for reduction in IMA and GEA blood flow; however, the exact relationship remains to be investigated in future studies.

In conclusion, this study has shown that arteries from different vascular beds respond differently to certain vasoconstrictors. Drugs used during and after bypass operations, such as dopamine and noradrenaline, may decrease vessel caliber via their constrictor properties, which has important implications for postoperative care as luminal narrowing of bypass grafts would reduce blood flow to the myocardium. In addition, we have shown that endogenous mediators such as 5-HT, histamine, and thromboxane have a greater constrictor effect on the IMA than on the GEA; the IMA may therefore be more prone to events that play a role in the initiation of vasospastic disorders, particularly in areas of endothelial abnormality.

We express our gratitude to the staff of the cardiac theaters at both Harefield and The Royal Brompton and National Heart and Lung Hospitals for their assistance in collection of vessels used in this study.

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Tricuspid Valve Myxoma: A Rare Indication for Tricuspid Valve Repair

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Excision of a myxoma involving the tricuspid valve often necessitates tricuspid valve replacement or a less than optimal margin of resection. We report a successful tricuspid valve repair after en bloc resection of a myxoma involving the septal leaflet of the tricuspid valve.

(Ann Thorac Surg 1991;52:1315–6)

Myxomas of the tricuspid valve are extremely rare. Excision of the myxoma may result in varying degrees of tricuspid valve insufficiency, which is either tolerated or treated by tricuspid valve replacement. Alternatively, a suboptimal resection of the involved leaflet is performed to preserve tricuspid valve function. We report a case of tricuspid valve repair after excision of a large myxoma arising from the tricuspid valve.

The patient was a 47-year-old woman with a history of mitral valve prolapse for several years. The patient complained of minimal symptoms of shortness of breath and palpitations with effort. These symptoms were not relieved by β-blockers. A preoperative echocardiogram done before laparoscopy for unrelated pelvic pain revealed a probable myxoma arising from the tricuspid valve.

Physical examination was unremarkable except for an auscultatory examination consistent with mitral valve prolapse. The chest roentgenographic and electrocardiographic results were normal. Echocardiography showed a heterogeneous discrete multilobulated mass fixed to the atrial surface of the septal leaflet of the tricuspid valve. During systole the mass prolapsed into the right ventricle. Minimal tricuspid insufficiency was present, without mitral insufficiency or other mass lesions.

The patient was taken to the operating room for resection of the myxoma. The cavae were separately cannulated and snared. Full cardiopulmonary bypass was instituted, and the patient was cooled to 30°C. The operation was performed without clamping the aorta. A right atriotomy was made, and the myxoma was found firmly attached to the midportion of the septal leaflet (Fig 1A). It measured 4 cm × 1 cm. The mass was excised in continuity with a quadrangular resection of the midportion of the septal leaflet. The tricuspid valve was repaired by annular plication and subsequent reapproximation of the free edges of the leaflet (Fig 1B). No annular ring was placed. Pathological examination showed fibromyxoid degeneration without evidence of malignancy.

The patient's postoperative course was unremarkable, and she was discharged on the tenth postoperative day. She was in normal sinus rhythm. Postoperative echocardiography revealed no tricuspid valve insufficiency and no residual tumor. The patient is presently doing well 1 year after operation without any evidence of tricuspid insufficiency on follow-up echocardiography.

Comment

The vast majority of cardiac myxomas arise from the atrial septum. Myxomas arising from valvular tissue are extremely rare. A review of the literature reveals only 13 patients with tricuspid valve myxomas. Involvement of the mitral valve appears to be even rarer. The reports of tricuspid valve myxomas reveal no distinguishing clinical features that would allow specific diagnosis of tricuspid valve involvement. Echocardiography was able to make the diagnosis in all patients for whom it was performed. The findings of a right atrial mass that completely prolapses into the ventricle during systole suggests a valvular origin of the tumor [1].

Of the 13 patients in the literature, 11 underwent operative procedures, 1 dying intraoperatively. Five patients had tricuspid valve replacement after the excision of the tumor and the tissue from which it arose [2–5]. There are 3 cases in which the tumor was removed without excising any margin of tissue from which the mass arose [6–8]. Cole and associates [1] report 1 case in which the mass was excised with a small ellipse of septal leaflet, which was subsequently re-approximated. Dang and Hurley [9] report a similar case, though details of the subsequent valvuloplasty are not given.

In our patient, the myxoma arose from a broad-based pedicle on the septal leaflet requiring a formal quadrangular resection of leaflet tissue to ensure an adequate margin of resection. The tricuspid valve repair was performed according to the techniques developed by Carpentier for the repair of mitral valve insufficiency secondary to posterior leaflet prolapse [10].

Cardiac myxomas are usually benign tumors, but instances of invasive growth, local recurrence, and metastases have been reported. For this reason total excision of the entire primary tumor including a margin of uninvolved cardiac tissue is the optimal surgical method for

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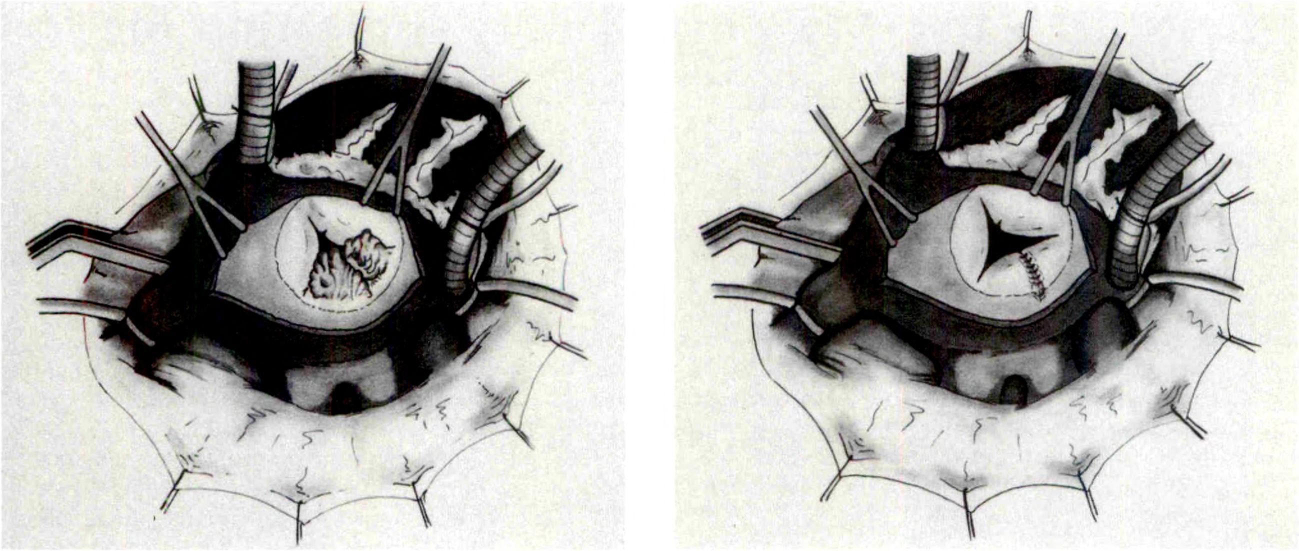

Fig 1. (A) Operative diagram of the myxoma arising from the septal leaflet of the tricuspid valve. (B) Diagram of tricuspid valve with excision of myxoma and repair of septal leaflet.

preventing tumor recurrence [2, 9]. This type of approach does not necessarily require tricuspid valve replacement. Tricuspid valve repair should be attempted whenever possible.

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Contralateral Pneumonectomy After Single-Lung Transplantation for Emphysema

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An intractable contralateral air leak developed in a 46-year-old woman after right single-lung transplantation for emphysema. A left pneumonectomy was performed on postoperative day 17, leaving the patient with only one transplanted lung. Fifteen months postoperatively the patient is well and has satisfactory pulmonary function. Survival with a good quality of life is possible after single-lung transplantation and bilateral sequential pneumonectomies.

(Ann Thorac Surg 1991;52:1317–9)

Successful transplantation therapy for end-stage emphysema has become a clinical reality in the past decade. Traditionally, heart-lung [1] or double-lung [2] transplantation has been offered to emphysematous patients with respiratory failure. Double-lung transplantation, however, has been plagued in its early experience by airway healing problems [3, 4], and heart-lung transplantation has become limited by the scarcity of satisfactory donor heart-lung grafts. Over the past 3 years single-lung transplantation has been performed successfully in an increasing number of emphysematous patients [5, 6]; nonetheless, the indications for single- or double-lung transplantation in end-stage emphysema remain controversial.

A 46-year-old woman had experienced progressive shortness of breath since 1976 and was diagnosed as having emphysema secondary to α_1-antitrypsin deficiency in 1986. Her respiratory function deteriorated despite cessation of smoking, and she was referred for a lung transplant assessment in March 1989. Physical examination revealed a debilitated patient who was wheelchair-bound and dependent on 3 to 4 L/min of nasal oxygen. She could walk only 50 m in 6 minutes. Pulmonary function tests showed a severe obstructive defect, with a forced expiratory volume in one second (FEV_1) of 0.31 L (12% of predicted), a forced vital capacity (FVC) of 0.75 L (24% of predicted), and an FEV_1/FVC ratio of 0.41. Echocardiography revealed normal left ventricular contractility, moderate global right ventricular hypokinesis, and mild to moderate tricuspid regurgitation. A quantitative perfusion lung scan demonstrated preferential blood flow to the apices of both lungs, with each lung receiving approximately 50% of the pulmonary blood flow. Right heart catheterization revealed a pulmonary artery pressure of 35/15 mm Hg, a pulmonary capillary wedge pressure of 11 mm Hg, a cardiac output of 5.2 L/min, and a pulmonary vascular resistance of 3.3 Wood units. The patient was initially accepted for a double-lung transplantation, but the operation was aborted on three occasions due to the fact that one or both donor lungs were unsuitable for transplantation. After 1 year on the waiting list, she was relisted as a single-lung transplant candidate in view of the recently reported experience with single-lung transplantation for end-stage emphysema [5, 6].

A right single-lung transplantation was performed on August 5, 1990. The donor lung was in excellent condition, and the donor had a total lung capacity more than 1 L greater than that of the recipient. Upon the induction of anesthesia in the recipient, marked hemodynamic instability developed due to bilateral tension pneumothoraces. Bilateral pleural tubes were inserted, but continued hypotension necessitated institution of cardiopulmonary bypass. The total ischemic time for the lung allograft, which was preserved using a pulmonary artery flush with modified Euro-Collins solution as well as 4°C topical saline hypothermia, was 4 hours 46 minutes. The initial post-bypass arterial blood gases were excellent, but the patient required moderate-dose dopamine and low-dose epinephrine to maintain hemodynamic stability. During the first 2 postoperative weeks, the left pleural tubes bubbled vigorously, and repeated roentgenograms documented poor expansion of the left lung, moderate subcutaneous emphysema, and mediastinal shift to the transplanted side. The patient's pulmonary mechanics precluded any attempt at extubation. A double-lumen endotracheal tube was placed, and two separate respirator circuits were employed to ventilate the transplanted right lung with a normal tidal volume, while maintaining only continuous positive airway pressure on the left lung. Despite these measures, the serious left lung air leak continued, the mediastinum remained massively shifted to the right side with hemodynamic compromise, and the transplanted lung was compressed (Fig 1).

On the 17th postoperative day a left thoracotomy was performed in an attempt to repair the source of air leak.

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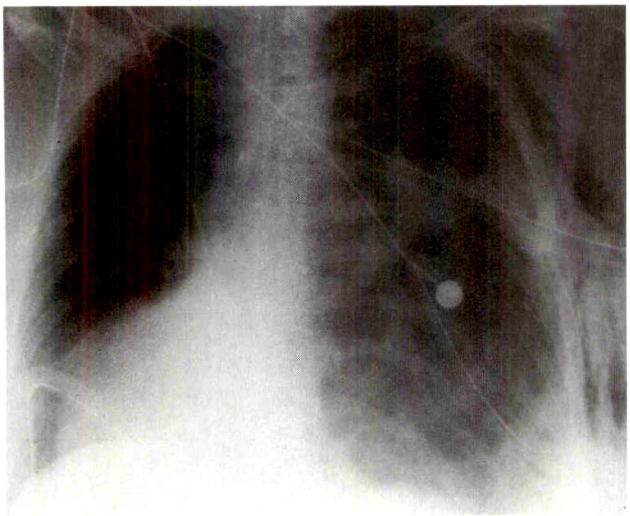

Fig 1. Chest roentgenogram 17 days after right single-lung transplantation for emphysema. The native left lung remains incompletely expanded despite the presence of two pleural tubes. There is substantial mediastinal shift to the transplanted side, and a moderate amount of subcutaneous emphysema in the left chest wall.

Unfortunately, there were multiple small, moderate-sized, and large bullae throughout the left lung, most of which were leaking air and could not be sutured or stapled. The only option was a left pneumonectomy, which was performed with pleural flap coverage of the left main bronchial stump. A tracheostomy and feeding jejunostomy were also carried out. Immediately after the operation the patient's hemodynamic condition improved, and she was rapidly weaned from inotropic support. Her ventilator weaning was prolonged because of a polyneuropathy involving the intercostal and diaphragm muscles as well as the limb muscles, which was documented by electrophysiological studies. Nevertheless, by 11 weeks postoperatively she was successfully weaned from mechanical ventilation, and by 15 weeks postoperatively from supplemental oxygen. The right lung remained clear on chest roentgenograms, and no episodes of rejection or opportunistic infection were diagnosed clinically; the risk of transbronchial biopsy was deemed to be prohibitive, and none were performed. Pulmonary function tests performed 4 months postoperatively when the patient was ambulatory revealed an FEV_1 of 1.28 L (48% of predicted), an FVC of 1.35 L (43% of predicted), and an FEV_1/FVC ratio of 0.96. Postoperative echocardiography confirmed persistent moderate right ventricular hypokinesis, although the tricuspid valve was only minimally regurgitant. The estimated pulmonary artery systolic pressure on Doppler echocardiography was 45 to 50 mm Hg. The patient was discharged home 18 weeks postoperatively and did well until May 1991, when she underwent an emergency Hartman's procedure for perforated sigmoid diverticulitis. She recovered uneventfully from this operation and is awaiting colonic reanastomosis. At present (15 months postoperatively) she can climb a flight of stairs without exertional dyspnea, and her arterial oxygen saturation on room air is 0.95 to 0.97 during moderate exercise. Her FEV_1 is 1.5 L (56% of predicted). Her current chest roentgenogram is shown in Figure 2. She has resumed an active lifestyle for the first time in more than 3 years.

Comment

The choice of lung transplant operation for end-stage emphysema remains controversial, and experience continues to accrue with single- and double-lung transplantation, as well as with heart-lung transplantation, for this condition. Khaghani and associates [1] have recently reported a series of 17 patients with emphysema who underwent heart-lung transplantation. The operative mortality was exceedingly low, and 91% of patients survived beyond 1 year. There were no complications in airway healing, and all survivors achieved an excellent level of rehabilitation. At present, heart-lung transplantation for emphysema is feasible only if the recipient's heart can be used for transplantation in a domino operation. Because of the proliferation of heart transplant programs in North America, satisfactory heart-lung grafts are increasingly difficult to obtain, and most centers have therefore opted for isolated lung transplantation for end-stage emphysema.

Successful double-lung transplantation for emphysema due to α_1-antitrypsin deficiency was first performed in November 1986 by the University of Toronto group [2]. The high incidence of airway healing complications has been the Achilles heel of this operation, and airway ischemia, anastomotic dehiscence, or late fibrous strictures have developed in at least half of the recipients of double-lung grafts in whom a tracheal anastomosis was employed [3]. The incidence of serious airway complications appears to be significantly reduced, but not eliminated, when a bilateral bronchial anastomotic technique is employed [3, 4]. Even this method of airway anastomosis

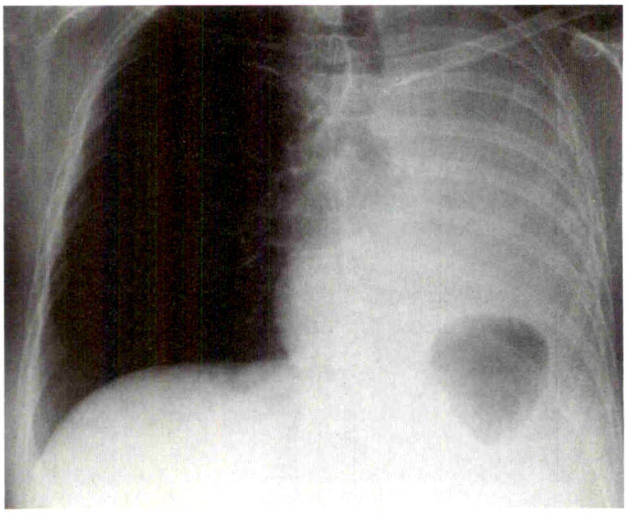

Fig 2. Current chest roentgenogram 15 months after right single-lung transplantation followed by left pneumonectomy.

does not engender airway healing that is as secure as in combined heart-lung transplantation, where uneventful tracheal healing is the rule even when corticosteroids are administered from the first postoperative day [7].

Experience with single-lung transplantation for emphysema is increasing, and a recent series comparing 7 such patients with 11 double-lung recipients has been published [8]. Despite the initial belief in the literature that single-lung transplantation for emphysema would result in preferential ventilation of the remaining native lung as opposed to the allograft, experience has indicated that in the absence of rejection, infection, or poor preservation most of the ventilation and perfusion is in fact distributed to the allograft [5, 6, 8].

Our single-lung recipient was severely debilitated, cachectic and not ambulatory before operation. The development of bilateral pneumothoraces upon the institution of positive-pressure ventilation was not an unexpected complication, given her marginal pulmonary reserve and multiple bilateral lung bullae. The persistence of a massive air leak from her native left lung, however, markedly exacerbated the mediastinal shift to the transplanted side and caused serious hemodynamic instability. Her postoperative convalescence would likely have been more rapid if a double-lung transplant operation was performed; nonetheless, the donor's contralateral lung was used in another recipient in a different city and was not available to us at the time of operation.

We would not advocate single-lung transplantation and bilateral sequential pneumonectomies in patients with cystic fibrosis or end-stage bronchiectasis, in whom this approach would be unlikely to succeed due to progressive infection of the allograft by the contralateral lung, even if it were removed early postoperatively. We were fortunate that the donor lung was well preserved and that our recipient did not incur a clinically detectable episode of lung rejection or infection, any of which would have posed a serious threat. Nevertheless, the fact that good quality intermediate-term survival is possible with only one transplanted lung should permit pneumonectomy to be considered in cases of unilateral airway necrosis after double-lung transplantation before the availability of a new allograft. Similarly, the application of single-lung transplantation to many candidates with end-stage emphysema should ameliorate the current severe demand-supply imbalance plaguing clinical lung transplantation.

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Aprotinin Used in Emergency Coronary Operation After Streptokinase Treatment

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We describe a patient with evolving myocardial infarction who underwent emergency intracoronary thrombolysis followed by immediate coronary artery grafting. Aprotinin was administered intraoperatively to control the potential bleeding problems associated with thrombolysis. Total postoperative blood loss was 260 mL. The case illustrates a further use for aprotinin in cardiac operations when excessive bleeding is anticipated.

(Ann Thorac Surg 1991;52:1320–1)

The use of aprotinin (a naturally occurring protease inhibitor) in patients with an increased risk of blood loss during open heart operations is now well established and is routinely practiced in many institutions in the United Kingdom and in Europe. In patients undergoing redo operations or operations for septic endocarditis, high doses of aprotinin have been shown to be effective in reducing intraoperative and postoperative blood loss and in lessening the requirement for blood and blood product transfusion [1]. The significance of the hemostatic effect, together with the absence of severe adverse reactions, have extended the indications for aprotinin in both cardiac and noncardiac surgical practice [2–4].

We describe a patient who underwent emergency coronary artery bypass grafting 1 hour after intracoronary thrombolysis for evolving myocardial infarction, and to whom aprotinin (Trasylol; Bayer AG, Leverkusen, Germany) was administered in an attempt to minimize the potential bleeding problems often associated with thrombolytic therapy.

A 60-year-old man with ischemic heart disease and previously well controlled exertional angina was admitted to the Hammersmith Hospital with unstable angina. Coronary angiography showed two-vessel disease with a moderate stenosis of the left anterior descending artery and a particularly severe stenosis of the proximal right coronary artery. His symptoms were initially controlled with β-blockers, nitrates, calcium antagonists, and full heparinization. He was referred for early surgical revascularization to be performed on the next available operating list.

He remained free of pain until 4 AM of the day the operation was scheduled to take place, severe chest pain developed, associated with marked inferior ST segment elevation on the electrocardiogram, suggestive of evolving myocardial infarction. Emergency coronary angiography revealed complete occlusion of the previously stenosed right coronary artery (Fig 1A). Intracoronary streptokinase (total dose of 40,000 IU) was administered, with angiographic resolution of the occlusion (Fig 1B), relief of the pain, and resolution of the electrocardiographic changes. One hour later the patient was transferred to the operating room. In view of the recent thrombolysis, we elected to administer aprotinin, using a modified dosage regimen of 2×10^6 KIU intravenously before cardiopulmonary bypass, with a further 2×10^6 KIU added to the pump prime. The operation consisted of a left internal mammary artery graft to the left anterior descending artery, and saphenous vein grafts to the distal right coronary artery and the first diagonal branch of the left anterior descending artery. During early chest opening and mobilization of the left internal mammary artery, there was considerable bleeding from all cut surfaces. After completion of revascularization and cessation of cardiopulmonary bypass, the operating field was dry. Bypass was discontinued easily, without any inotropic support.

Postoperative blood loss was 170 mL in the first 12 hours and 90 mL in the subsequent 12 hours; a total of 260 mL chest tube drainage. The patient did not require any blood or blood product transfusions. There was no electrocardiographic evidence of myocardial infarction, and there was no elevation of the serum creatine kinase level in the perioperative period. After an uneventful recovery he was discharged home on the seventh postoperative day.

Comment

In recent years there has been an increasingly active approach toward the management of acute myocardial ischemia, particularly with the widespread application of coronary thrombolysis. However, large numbers of patients are now being referred for emergency coronary operations with refractory myocardial ischemia, having undergone recent thrombolytic treatment. In view of the high probability of excessive perioperative bleeding, cardiac surgeons have been cautious of operating under such circumstances. In previous reports [5, 6] the average postoperative blood loss in patients undergoing emergency coronary operation after intracoronary thromboly-

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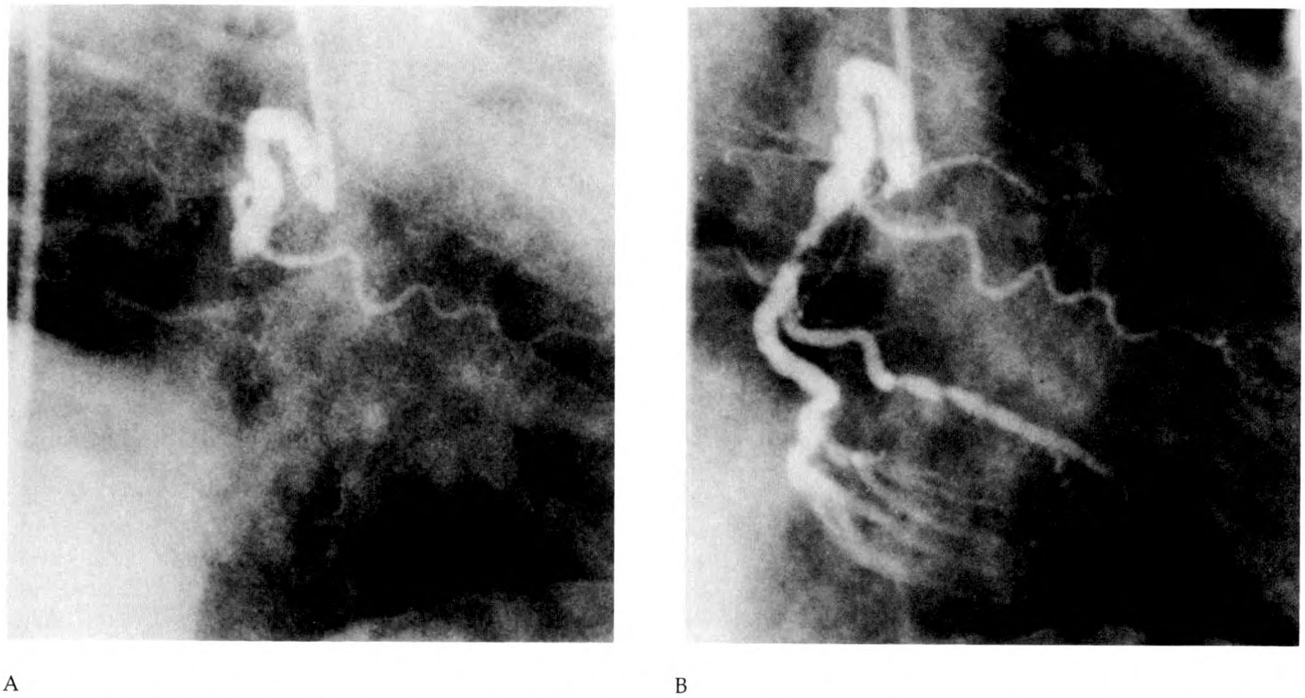

Fig 1. End-diastolic frame of the right coronary artery in the right anterior oblique projection. (A) Complete occlusion of the vessel. (B) Patent but severely strictured vessel after intracoronary administration of streptokinase.

sis was significantly greater than for elective cases, with an increased need for surgical reexploration. Skinner and colleagues [6] reported a mean postoperative blood loss of 1,450 mL, with 5 of 24 patients bleeding in excess of 2,000 mL. In their series the average transfusion requirement per case was 8.2 units of blood, 8.0 units of fresh frozen plasma, and 11.0 units of platelets.

In the case we have described, sudden occlusion of the right coronary artery resulted in rapidly evolving myocardial infarction and a deteriorating clinical condition. To prevent myocardial necrosis, we chose emergency thrombolysis as a first line of management, and we administered the lowest dose of intracoronary streptokinase to open the occluded vessel. Having temporarily stabilized the clinical situation, we sent the patient for urgent surgical revascularization. We were gratified to observe that the use of aprotinin completely prevented the potentially hazardous bleeding problems associated with streptokinase administration.

The precise mechanism of action of aprotinin is still unclear. Although most of the recent studies of aprotinin have been in cardiac surgical patients, the fact that efficacy has been demonstrated in other forms of surgery might suggest that aprotinin acts in a nonspecific manner, rather than on any one particular hemostatic disorder induced by cardiopulmonary bypass. Two possible, though not necessarily conflicting, theories concerning the mechanism of action have been proposed: the first favors maintenance of platelet function by preservation of surface receptors [7]; the second favors an antifibrinolytic effect through its antiplasmin activity [3, 8]. The prevention of thrombolytic therapy-induced bleeding in the present case would appear to lend support to the latter.

It is clear that a better understanding of the exact mechanism of action of aprotinin will allow a more rational approach to its therapeutic applications. We believe that, although many cardiac surgeons are already familiar with aprotinin, its full potential in cardiac and noncardiac surgery has not yet been fully exploited. The case described illustrates a further possible indication for the use of aprotinin.

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Mixed Tumor of Salivary Gland Type Arising in the Bronchus

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During bronchoscopy for atelectasis of the middle lobe, a 61-year-old man was found to have a polypoid mass obstructing the bronchus. Right middle and lower lobectomy disclosed a tumor growing into the lumen of the middle lobe bronchus. The histological features of the tumor were indistinguishable from those of the mixed tumor of the salivary gland. A review of the literature reveals only 7 cases of this extremely rare tumor.

(Ann Thorac Surg 1991;52:1322–4)

Mixed tumor, which is also called pleomorphic adenoma, is the most common neoplasm of the salivary glands. The tumor may be found in the palate, tongue, nasopharynx, or larynx [1]; however, there are rare reports of this neoplasm arising in the bronchial tree. This article reports a case of mixed tumor of salivary gland type arising in the bronchus and presents a review of the literature.

A 61-year-old man was found to have a total atelectasis of the middle lobe of the right lung on a chest roentgenogram. The patient had a history of bronchial asthma in his childhood and 3 months before admission, but otherwise no episode of cough, sputum, hemoptysis, shortness of breath, or fever. He had been smoking one package of cigarettes a day for 40 years. Evaluation of the family history disclosed that his two elder brothers had lung cancer.

On admission, physical examination revealed no particular abnormality. Tumor markers were found to be in normal range (α-fetoprotein, 5.9 ng/mL; carcinoembryonic antigen, 1.7 ng/mL; carbohydrate antigen 19-9, 4.0 U/mL). A computed tomogram of the chest revealed atelectasis of the right middle lobe and complete obstruction of the middle lobe bronchus (Fig 1).

Fiberoptic bronchoscopy revealed a white polypoid mass obstructing the orifice of the middle lobe bronchus. Histopathological findings of the intrabronchial mass obtained by multiple biopsies suggested malignant tumor consisting of epithelial cells and myxochondroid tissue.

Right middle and lower lobectomy was performed in January 1990. Grossly, the tumor was a grayish-white polypoid mass, measuring 5.3 by 3.5 cm, growing into the lumen of the middle lobe bronchus. No invasion into the peribronchial lung tissue was seen. Microscopically, the tumor consisted of an intermingling of epithelial elements and myxomatous stroma developing into cartilage (Fig 2). Epithelial tumor cells were arranged in closely packed sheets, anastomosing cords, or interweaving bands. Multiple foci of squamous metaplasia with keratinization were apparent (Fig 3). The cartilaginous areas showed a strong metachromatic reaction with toluidine blue. Foci of bone formation were found. Immunohistochemical staining demonstrated positive reactions with polyclonal anti-S-100 protein and anti-actin antibodies in tumor cells in the myxoid region. The squamous epithelial cells showed strong immunoreactivity for cytokeratin. Electron microscopic studies revealed the tumor cells had moderately developed cytoplasmic organelles and cytoplasmic processes occasionally joined by desmosomelike structures. Secretory granules were found in some tumor cells, and intracytoplasmic filaments were not evident. The microscopic features of the tumor were indistinguishable from those of mixed tumors arising in the salivary gland. Dissected mediastinal and pulmonary hilar lymph nodes showed no involvement.

Comment

Mixed tumor of salivary gland type is an extremely rare neoplasm among the tumors of the bronchus, although the morphological and histogenetic resemblance between the salivary and the bronchial mucous glands implies the occurrence of this tumor arising in the bronchus to be logical [2]. A review of the literature reveals that only 7 cases [2–6] of this rare tumor have been reported since Payne and colleagues [2] described 2 cases that were believed to be genuine examples of mixed tumor of salivary gland type in 1965.

The clinical features of the reported cases are shown in Table 1. Six of the 8 patients had cough and sputum, and 3 patients had an attack or episodes of pneumonia. The diagnosis of the respiratory episode 3 months before admission in the present patient as bronchial asthma was retrospectively considered to be wrong. If the respiratory symptoms in patients 2, 3, 4, and 7, which persisted for 2 years or more, were referable to existence of the tumor, the clinical history of the patients with mixed tumor of salivary gland type in the bronchus suggests a relatively benign behavior of the neoplasm [3].

No one method is universally accepted as an optimal

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Table 1. Reported Cases of Mixed Tumor of the Salivary Gland Type Arising in the Bronchus

Patient No.	Author	Year	Age (y)	Sex	Smoking (packs, years)	Location (affected bronchus)	Symptoms	Duration	Bronchoscopy	Operation	Follow-up
1	Payne et al [2]	1965	50	F	?	Right LLB	Cough, sputum, attack of pneumonia	4 mo	Polypoid tumor	Bilobectomy	Alive, 11 y
2	Payne et al [2]	1965	47	M	?	Right ULB	Episodes of pneumonia	2 y	Polypoid tumor	Lobectomy	Alive, 11 y
3	Davis et al [3]	1972	52	F	?	Right ULB	Attack of pneumonia	4 y	Jellylike growth	Pneumonectomy	Postoperative death
4	Ushijima et al [4]	1975	60	F	1, 40	Left BSB	Fatigue, cough	20 y	NE	Lobectomy	Alive, 2 mo
5	Nakamura et al [5]	1977	70	M	2, 40	Left BSSB	Cough, sputum, dyspnea	1 mo	Polypoid tumor	Pneumonectomy	Alive, 4 y
6	Nakamura et al [5]	1977	71	F	0	Left BSB	Cough, sputum	?	Deviated bronchi	Lobectomy	Alive, 2 y
7	Ebihara et al [6]	1980	72	M	?	Right SLLB	Dyspnea, sputum	2 y	NE	No (autopsy)	No
8	Present case	1991	61	M	1, 40	Right MLB	Episodes of asthma	3 mo	Polypoid mass	Bilobectomy	Alive, 16 mo

BSB = basal segment bronchus; BSSB = basal subsegment bronchus; LLB = lower lobe bronchus; MLB = middle lobe bronchus; NE = not examined; SLLB = superior lower lobe bronchus; ULB = upper lobe bronchus.

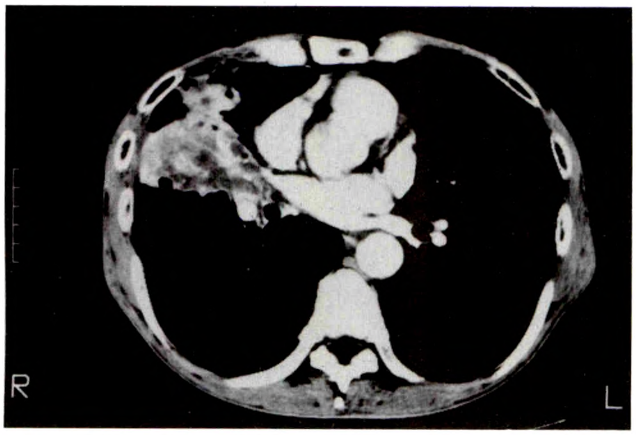

Fig 1. Computed tomogram of the chest demonstrating complete atelectasis of the middle lobe and low-density mass filling the dilated lumen of the bronchus. Small calcification in the mass is noted.

treatment for mixed tumor of the bronchus. Wide surgical excision probably can be curative [2, 5]. Regarding prognostic implications, it is difficult to draw any definitive conclusions because of the small number of reported cases of this neoplasm.

Histological features of the mixed tumor of salivary gland type arising in the bronchus are similar to those of the mixed tumor originating in the salivary gland, which consists of diverse components: epithelial elements and myxochondroid stroma [1, 7].

The histogenesis of the mixed tumor of the salivary gland remains controversial [1, 7], although the roles of the myoepithelial cell are being clarified [1, 7, 8].

Slow development of the clinical manifestations, good survival after operation in 4 of the 8 patients, and absence of lymph node involvement or distant metastasis suggest that this neoplasm is relatively benign; however, it is not preferred to call the lesion benign conclusively. Biological behavior of the mixed tumor of salivary gland type arising in the bronchus seems to resemble that of the pleomor-

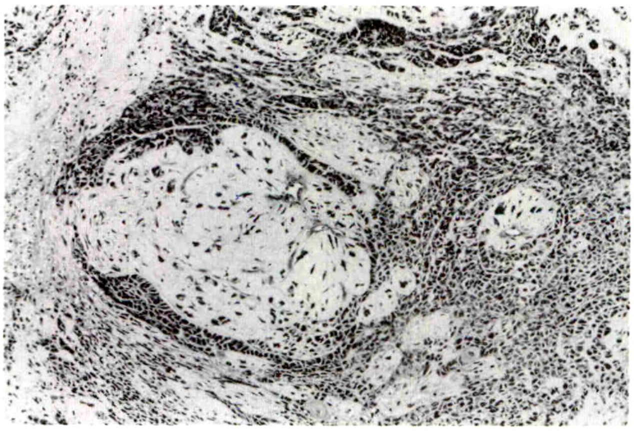

Fig 2. Photomicrograph of the tumor in the resected lung demonstrating epithelial cells arranged in anastomosing cords and myxochondroid stroma. (Hematoxylin and eosin, ×40 before 33% reduction).

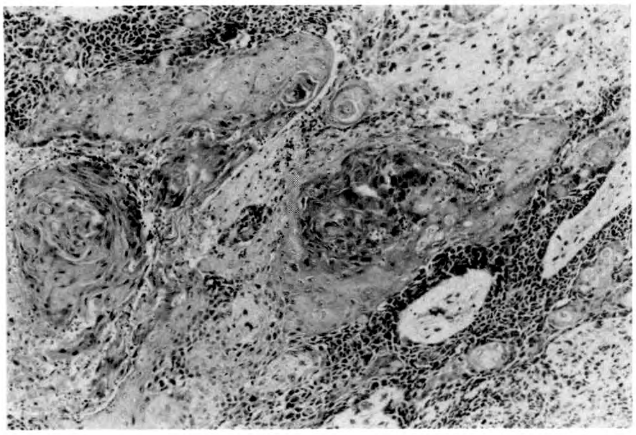

Fig 3. Photomicrograph of the same specimen as in Figure 2 showing foci of squamous metaplasia with keratinization. (Hematoxylin and eosin, ×150 before 33% reduction).

phic adenoma arising in the salivary gland and, therefore, it should be regarded as potentially malignant [1, 5]. Histological criteria of the possible malignant behavior of the mixed salivary tumor, however, are difficult to define. Extension of the growth into the perineural or other lymphatics or densely cellular areas with many mitotic figures, prominent nuclei, and foci of necrosis may relate to malignancy [1].

Further accumulation of cases of mixed tumor of salivary gland type arising in the bronchus might make it possible to define the biological behavior and to establish the adequate measures for treatment of this rare neoplasm.

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Perioperative Spinal Canal Narrowing in Patients With Down's Syndrome

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Two patients with Down's syndrome undergoing intracardiac operations had segmental and generalized myoclonic movements postoperatively and eventual brain death. Electroencephalography in 1 patient showed no seizure despite the presence of the myoclonic movements. Computed tomographic scan showed possible cerebellar hemorrhage. Ultrasound showed cerebral edema when the pupils became fixed and dilated. Because known postoperative neurologic complications could not fully explain the clinical course, and the myoclonic movements suggested spinal origin, we considered the possibility of atlantoaxial instability causing spinal cord damage related to perioperative head and neck positioning. Postmortem study on the second patient revealed 50% reduction of the spinal canal with hyperextension and 90-degree external rotation of the head and neck. In contrast, similar maneuvers in 3 infants without Down's syndrome resulted in only mild spinal canal narrowing. Although the myoclonic movements could be explained by spinal cord compression at the atlantoaxial level, the explanation for the eventual brain death is unclear. However, kinking of the vertebral arteries related to the positioning could have caused cerebellar ischemia, hemorrhage, and increased intracranial pressure. We believe that attention to the problem might bring further answers in the future.

(Ann Thorac Surg 1991;52:1325–8)

The occurrence of atlantoaxial dislocation in a substantial number of patients with Down's syndrome is well known [1–3]. Because life-threatening spinal cord compression can develop, identified patients are followed up closely and their activities restricted [4, 5]. Neurologic symptoms can occur spontaneously or follow a nasopharyngeal infection or minor trauma [6–8]. We report 2 patients with Down's syndrome who had neurologic injury, possibly due to spinal cord compression, after a cardiac operation.

Material and Methods

Unexplained postoperative neurologic problems developed in 2 female patients with Down's syndrome who underwent intracardiac operation within 2 months of each other at 15 and 8 months of age. Weights were 7.6 and 4.7 kg. Both patients had induction of anesthesia with intramuscular ketamine followed by inhalation of halothane and oxygen. Anesthesia was maintained with halothane, nitrous oxide, oxygen, intermittent intravenous fentanyl, and paralyzing doses of pancuronium. Cephalosporin and methylprednisolone (20 mg/kg) were also given intravenously. Anesthetic management was uneventful in both patients.

Midsternotomy was performed in both patients, and cardiopulmonary bypass was instituted through transatrial bicaval and ascending aorta cannulation. A hollow fiber oxygenator (Terumo Corp, Somerset, NJ), a dual heater-cooler, and a roller pump were used. A left ventricular sump was placed and hypothermia was induced to a rectal temperature of 25°C. Blood cardioplegia was administered at 15- to 20-minute intervals.

Patient 1 underwent total repair of complete atrioventricular canal and pulmonary artery debanding. Total bypass time was 160 minutes, lowest esophageal temperature was 23°C, duration of hypothermia with rectal temperature at 25°C or less was 75 minutes, and pump flow was 2,000 $mL \cdot min^{-1} \cdot m^{-2}$. Initial myocardial contractions were poor but gradually improved, and the patient was weaned from bypass on infusions of isoproterenol (Isuprel; Winthrop Pharmaceuticals, New York, NY) dopamine, and dobutamine.

Postoperatively, the patient was kept on the respirator and was hemodynamically stable. Initially, there was no spontaneous movement or response to tactile stimuli although the patient would become agitated with endotracheal suctioning. Nine hours postoperatively, the patient started having twitching of the hands, occasionally progressing to the shoulders. Phenobarbital (15 mg/kg) was given. Twitching of the right hand and occasionally of the left hand became rhythmic and clonic in nature. The patient did respond to painful stimuli. Twelve hours postoperatively, generalized seizures developed with stiffening of upper extremities and decorticate posturing.

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Carbamazepine (Tegretol; Geigy Pharmaceuticals, Ardsley, NY) and paraldehyde were administered. In spite of the continued rhythmic clonic movements of the hands, electroencephalogram showed no seizure activity. Slow intermittent and jerky movements of the neck, shoulders, and left abdominal muscles were also noted. The patient would withdraw from painful stimuli even in the presence of the clonic, jerky movements. A neurologic diagnosis of generalized and segmental myoclonus was made, probably due to damage to the spinal cord, and clonazepam was administered. Early on the second postoperative day, the patient aroused easily and had spontaneous eye opening. However, there was no purposeful spontaneous movement and bilateral clonic movements of the extremities continued. On the afternoon of the second postoperative day, the pupils suddenly became fixed and dilated. Head ultrasound showed cerebral edema, and computed tomographic scan of the brain showed increased densities of the cerebellar areas consistent with intracranial hemorrhage. Jerky movements of the shoulders and legs continued until brain death was established by clinical criteria and absence of cerebral flow on the ninth postoperative day.

Patient 2 underwent closure of a ventricular septal defect and an atrial septal defect and ligation of the patent ductus arteriosus. Total bypass time was 75 minutes, lowest esophageal temperature was 25°C, duration of hypothermia with rectal temperature of 25°C or less was 15 minutes, and pump flow was 2,000 mL $\cdot$ min^{-1} $\cdot$ m^{-2}. Although the patient was easily weaned from bypass, isoproterenol and dopamine infusions were started because of cardiomegaly from serious shunts.

Postoperatively, the patient was also kept on the respirator and was hemodynamically stable. Initially, movement of all extremities was present. Three hours postoperatively, the patient started having twitching of the extremities and eyelids. Phenobarbital, 15 mg/kg, was given. Intermittent mild twitching of the extremities continued, and carbamazepine and paraldehyde were added. The patient had sudden hypotension and metabolic acidosis. Dobutamine infusion was started. Supplemental intravenous fluids were administered, and the metabolic acidosis was corrected. Shortly thereafter, the pupils became fixed and dilated. Ultrasound of the brain revealed cerebral edema. The patient stabilized hemodynamically; however, on the third postoperative day, brain death was established by clinical criteria and absence of cerebral blood flow. Roentgenography of the cervical spine suggested atlantoaxial subluxation. Postmortem examination of this patient revealed substantial reduction of the spinal canal (50%) with hyperextension and 90-degree external rotation of the head and neck (Fig 1). There was no narrowing with flexion and only mild narrowing with hyperextension. In contrast, similar postmortem maneuvers in 3 infants (aged 1 to 5 mo) who died of complex congenital heart defects, but did not have Down's syndrome, only produced mild narrowing of the spinal canal.

In patient 2, macroscopic sections through the cervical cord showed central hemorrhagic necrosis of the cord extending to the lower cervical segments. Microscopic

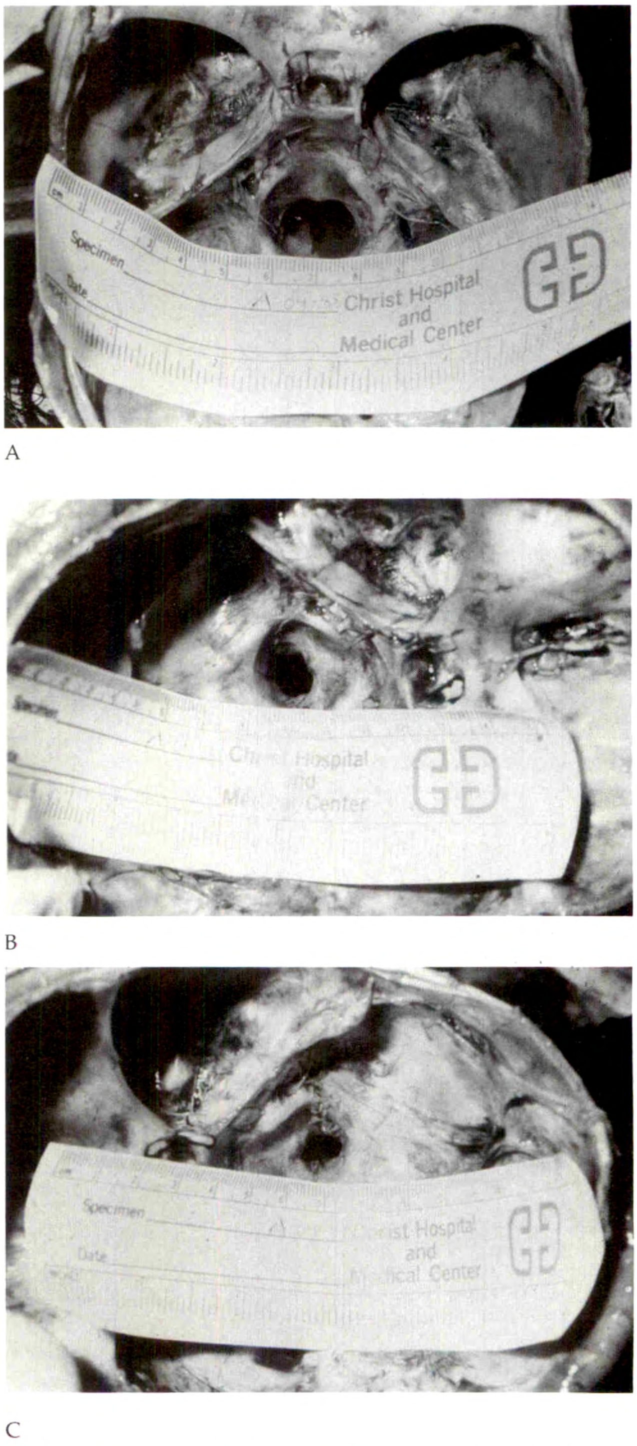

Fig 1. Postmortem study of the size of the spinal canal at the atlantoaxial junction of patient 2. The spinal canal was viewed and measured through the base of the skull. The top of the skull was removed. (A) The head is in neutral position and the spinal canal measures approximately 2 cm in diameter. Although not shown, no substantial change was noted with flexion. (B) The head is externally rotated (90 degrees) to the right with some hyperextension of the neck. There was substantial reduction of the size of the spinal canal. Although not shown, no substantial change was noted with hyperextension alone. (C) The head is externally rotated (90 degrees) to the left with some hyperextension of the neck. There was substantial reduction (50%) in the size of the spinal canal.

sections showed acute necrosis and loss of neuronal cell bodies in the hippocampus and Purkinje cell layer of the cerebellum. Scattered acutely necrotic neuronal cell bodies were seen in the neocortex, corpus stratum, thalamus, and brainstem, segmental and basal nuclear groups, and the dentate nucleus of the cerebellum. Similar changes with recent hemorrhage were seen in the posterior gray and white columns of the upper cervical cord.

Comment

Known neurologic complications such as cerebral infarction, emboli, and serious intracranial hemorrhage did not fully explain the postoperative neurologic problems that occurred in these 2 patients. Although the myoclonic movements suggested spinal cord damage, the sudden fixation and dilation of the pupils was puzzling. Encephalography did not reveal any seizure activity, despite the presence of myoclonic movements. Except for the initially poor myocardial contractility in 1 patient, the conduct of anesthesia, operation, and cardiopulmonary bypass were uneventful. Because both patients had Down's syndrome and the second patient had radiographic evidence of atlantoaxial subluxation, we suspected spinal canal narrowing related to perioperative hyperextension and external rotation of the head and neck.

Although the myoclonic movements could be explained by spinal cord compression at the atlantoaxial level, the explanation for the eventual brain death is unclear. As spontaneous cerebellar infarctions have been reported in patients with Down's syndrome, presumably from kinking of the vertebral arteries at the level of an atlantoaxial dislocation, it is possible that compression of the vertebral arteries related to the perioperative positioning might have contributed to the neurologic problems [9].

Various cervical abnormalities have been noted in patients with Down's syndrome [10, 11]. The most common is atlantoaxial instability, which is found in 9% to 31% of patients and with higher prevalence in female than male patients (2:1) [5]. Subluxation is presumed to be due to laxity of the ligaments at the atlantoaxial junction [12]. Although the incidence of atlantoaxial instability is high, clinical evidence for spinal cord compression occurs in only a small number of patients (1.5% to 2%) [13].

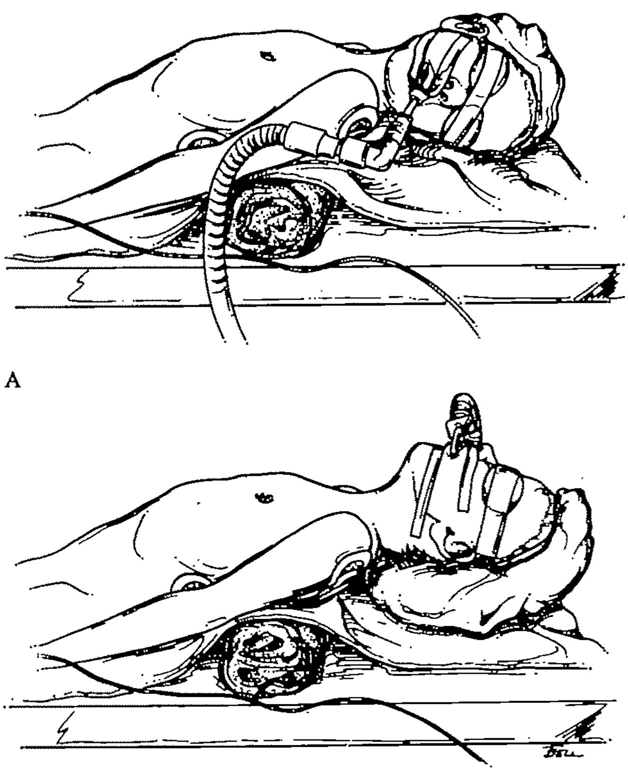

Fig 2. (A) A position commonly used in cardiac surgery that can cause spinal canal narrowing in patients with Down's syndrome: a shoulder pad is used and the head is externally rotated. (B) Recommended position: a shoulder pad is still used, but the head is kept in neutral position.

Two previous case reports documented symptomatic atlantoaxial dislocation associated with Down's syndrome in the perioperative period [14, 15]. Neck pain with limited mobility developed in 1 patient 5 days after closure of an atrial septal defect, whereas the other patient was unable to walk after closure of a ventricular septal defect. The head and neck positions of these patients at the time of operation were not mentioned. At the present time, there are no clear guidelines in the preoperative, intraoperative, and postoperative management of patients with Down's syndrome with regard to atlantoaxial instability. One report recommended that all children older than 4 years who are to undergo operation be screened for atlantoaxial instability and that cervical fusion be carried out in symptomatic patients before any other surgical intervention. In asymptomatic patients, neurosurgical guidance was advised regarding cervical stabilization before and after anesthesia. One report suggested avoiding hyperextension [15], whereas another suggested avoiding forceful flexion [16] of the neck during tracheal intubation. Neither report addressed the issue of external rotation during hyperextension.

Varying degrees of hyperextension and external rotation of the head and neck occur during positioning for open heart operations, using a shoulder pad to bring the great vessels anteriorly and remove the patient's chin from the field (Fig 2A). A similar position can also occur postoperatively in the intensive care unit. We have shown that perioperative hyperextension and external rotation of the head and neck can cause serious narrowing of the spinal canal in patients with Down's syndrome, whereas flexion or hyperextension alone appears to be safe. In the last 3 years, we have been particular in the positioning of patients with or without Down's syndrome in the perioperative period. We still use a shoulder pad, but we avoid external rotation of the head and neck (Fig 2B).

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Aplasia of the Right Aortic Cusp in a Neonate: A Life-Threatening But Curable Anomaly

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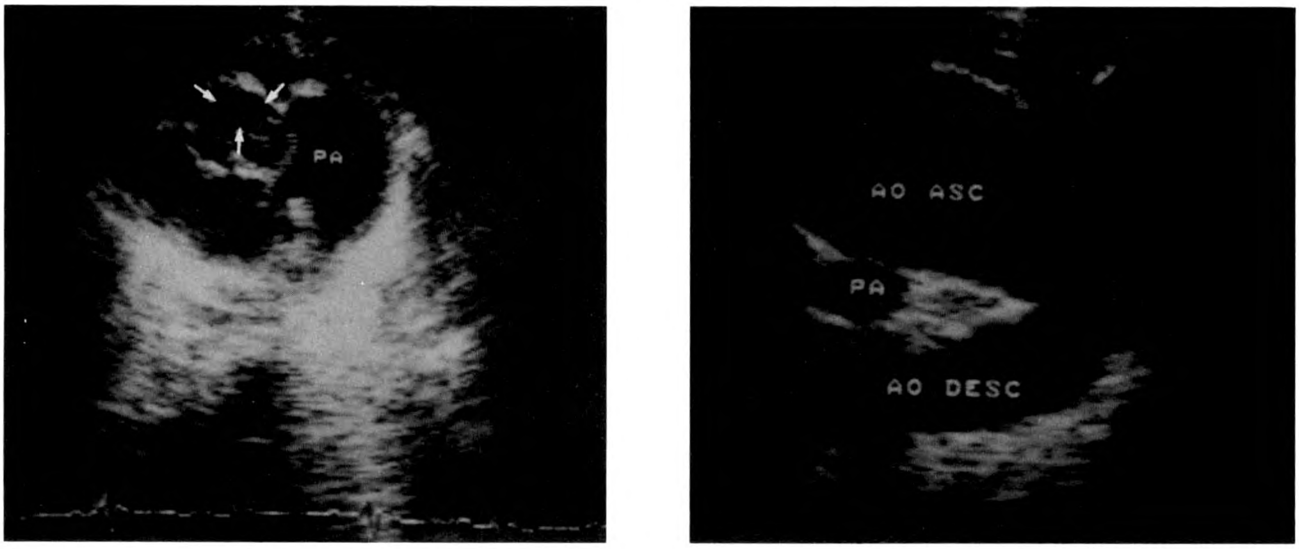
A case of absent right aortic cusp causing serious aortic incompetence in a neonate is reported. A "bicuspidalization" repair broke down within 24 hours, but reoperation with annular enlargement and insertion of a 19-mm prosthetic valve resulted in complete recovery. This rare but life-threatening anomaly of the aortic valve can be recognized with Doppler echocardiography. Valve replacement in conjunction with aortic annular enlargement should be performed as primary repair.

(Ann Thorac Surg 1991;52:1329–30)

Bicuspid aortic valve is known to occur in about 5% of the population. Such valves usually become incompetent or stenotic in later life. We report a case of aplasia of the right aortic cusp in an aortic root with three coronary sinuses.

The patient was a boy, born in the 36th week of gestation, with a birth weight of 2,540 g, a length of 47 cm, and clinical signs of marked valvular incompetence (Fig 1, Table 1). At the age of 4 months, when his weight was 4,370 g, progressive biventricular failure made surgical intervention mandatory. At operation on May 3, 1988, the aortic valve was found to be "bicuspid" and the right aortic cusp was absent (Fig 2). The coronary orifices were normally located. Half of the "larger" left cusp was detached, mobilized leftward, and reapproximated to the annular defect by means of mattress sutures (Fig 2). This "bicuspidalization" repair broke down after 24 hours, with serious hemodynamic consequenses and renal failure necessitating peritoneal dialysis.

Reoperation was performed 2 days later. The aortic annular circumference was enlarged with a 3 × 5-cm



A

B

Fig 1. (A) Parasternal short-axis Doppler echocardiographic view of the aortic valve. Arrows *indicate absence of the right cusp. (B) Two-dimensional echocardiogram showing dilation of the ascending aorta* (AO ASC) *in relation to the descending aorta* (AO DESC). (PA = *pulmonary artery.)*

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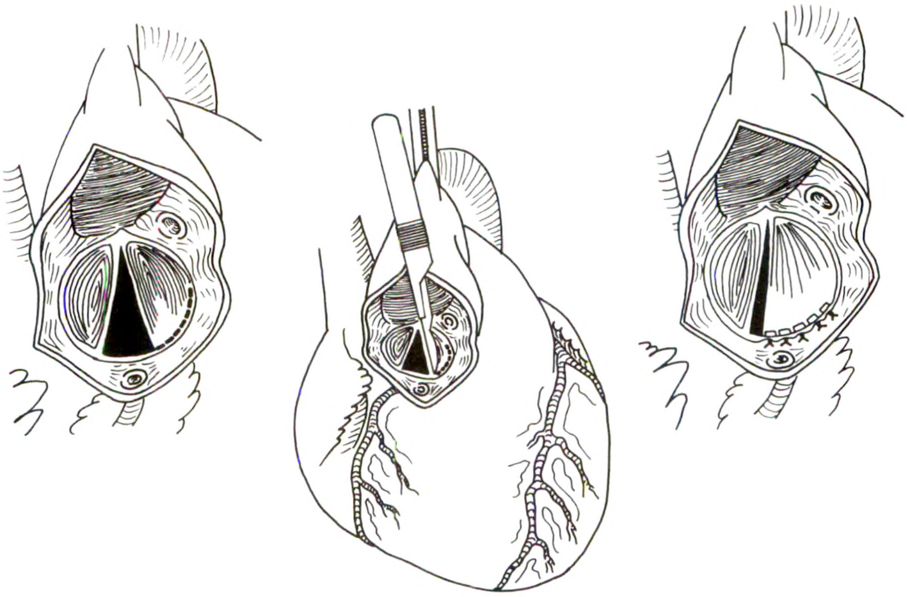

Fig 2. Operative view of aplasia of the right aortic cusp before and after bicuspidalization.

"noncoronary sinus" patch of glutaraldehyde-preserved bovine pericardium. This permitted insertion of a 19-mm St. Jude mechanical bileaflet prosthesis. After 10 critical days the boy recovered, and at 6 months follow-up he was in excellent condition, with well-conducted warfarin anticoagulation and no thromboembolic events. He was in sinus rhythm and the prosthetic valve functioned normally, with no sign of leak (see Table 1). Ventricular function had recovered and renal function was normal.

The histologic appearance of the excised aortic cusps was consistent with valve dysplasia due to myxomatous, fibrotic, noninflammatory degeneration.

Table 1. Doppler Echocardiographic Investigation

Variable	Preoperative	6 Months Postoperative
Body weight (kg)	4.2	6.5
Diameters (mm)		
Aortic annular	16	19[a]
Ascending aorta	20	26
Descending aorta	10	13
Flow velocity across (m/s)		
Aortic valve	4.3	1.5[a]
Pulmonary valve	1.7	0.9
Gradient across aortic valve (mm Hg)	50	15[a]

[a] Prosthetic valve.

Comment

Being unaware of the possibility of aplasia of the right aortic cusp, we thought that aortico-left ventricular tunnel might be present [1]. Attempted angiographic exploration of the aortic root had to be abandoned because of the infant's unstable hemodynamics. Retrospective review of the preoperative Doppler echocardiographic images showed how the aplasia appeared in parasternal short-axis view of the aortic valve (see Fig 1A). Doppler echocardiography thus can permit distinction of this anomaly from aortico-left ventricular tunnel [2].

Bicuspidalization cannot be recommended in aplasia of the right aortic cusp, partly because of the anomaly's myxomatous nature. Valve replacement in conjunction with aortic annular enlargement should instead be the primary procedure.

We had planned to perform an aorticoventriculoplasty, but refrained because of the pronounced biventricular hypertrophy, with the right ventricular outflow tract elevated 1.5 cm above the aortic root. Widening with a patch in the noncoronary sinus proved to be a simpler and more appropriate option.

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Extended Aortic and Left Main Coronary Angioplasty With a Single Pericardial Patch in a Patient With Williams Syndrome

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In a 9-year-old girl with supravalvular aortic stenosis combined with severe left main coronary artery trunk obstruction (Williams syndrome), the repair for both lesions was performed successfully by simultaneous extended aortoplasty and direct coronary angioplasty using a single double-flanged pericardial patch.

(Ann Thorac Surg 1991;52:1331–3)

In congenital supravalvular aortic stenosis (SAS), known as Williams syndrome [1], coronary artery abnormality is one of the major associated cardiovascular lesions [2, 3]. Obstructing lesion, particularly of the left main coronary trunk (LMT), is a critical condition for the natural history as well as for the surgical management of this disease. As the surgical procedure for the LMT lesion, excision of the fibrous ridge, excision of the hypertrophic intima at the ostium, or saphenous vein aortocoronary bypass grafting has been proposed [4, 5]. Recently, Martin and associates [6] reported an approach with coronary angioplasty and simultaneous aortoplasty using two pericardial patches. For SAS, extended aortoplasty with a double-flanged patch [7, 8] appears to have advantages over simple aortoplasty [9]. In this article, we report a new method of simultaneous LMT coronary angioplasty and extended aortoplasty using a single double-flanged pericardial patch in a patient with SAS and LMT obstruction.

The patient was a 9-year-old girl known to have Williams syndrome since the age of 3 years. Cardiac catheterization and angiography at 5 years of age showed typical SAS with a pressure gradient of 34 mm Hg. Multiple pulmonary peripheral stenosis was also demonstrated, and balloon angioplasty was attempted. The pressure gradient at SAS increased to 58 mm Hg at repeated cardiac catheterization at 9 years of age, when she started to have exertional chest pain with ischemic electrocardiographic changes. Aortography demonstrated a localized 90% narrowing of the LMT (Fig 1). Surgical treatment was indicated for SAS and LMT obstruction.

Operation was performed using hypothermic cardiopulmonary bypass and median sternotomy. For the preparation of the LMT, the dissection was limited only to mobilize the pulmonary artery trunk from the ascending aorta because of the dense fibrous tissue over the LMT area. The aorta was cross-clamped, and aortotomy was first made above the stenotic lesion, extending down to the right coronary sinus and crossing the supravalvular stenosis and fibrous ring (Fig 2). The right coronary ostium was intact. The left main coronary orifice was very small, with an overhanging fibrous shelf. The ostium did not admit a coronary probe. Aortotomy was extended in a reversed Y shape starting at 1 cm above the stenotic fibrous ring toward the left coronary orifice. The incision was further extended to the LMT beyond the obstruction, reaching to the bifurcation.

A long piece of autologous pericardium, treated with glutaraldehyde solution on the table (6 minutes in 0.6% solution), was prepared for extended aortoplasty with two flanges, making the left one as long as for coronary angioplasty. This patch was first sutured to the edge of LMT incision using 7-0 Prolene (Ethicon, Sommerville, NJ) continuous suture, and covered the LMT up to the left coronary sinus. The width of the patch over the LMT was 7 mm. The patch was then sutured to the right coronary sinus using the right side flange. The rest of the suture for the aortoplasty above the aortic valve area was performed using 6-0 Prolene continuous suture (see Fig 2). The width of the patch for aortoplasty was 15 mm.

Postoperative recovery was uneventful. Cardiac catheterization 1 month later showed a pressure gradient of 8 mm Hg across the left ventricular outflow tract, and aortography demonstrated a widely patent LMT and smoothly enlarged aortic root (see Fig 1B).

Comment

The preferable surgical method for the relief of combined LMT obstruction in SAS appears to be direct coronary angioplasty rather than aortocoronary bypass. This is because the obstruction usually is located at the orifice or in a short segment of LMT, and atheromatous changes may be less serious in this syndrome. The method described here is a combination of extended aortoplasty (Doty) and coronary patch angioplasty as a single procedure, in contrast to the method reported by Martin and associates [6], in which both procedures were done sep-

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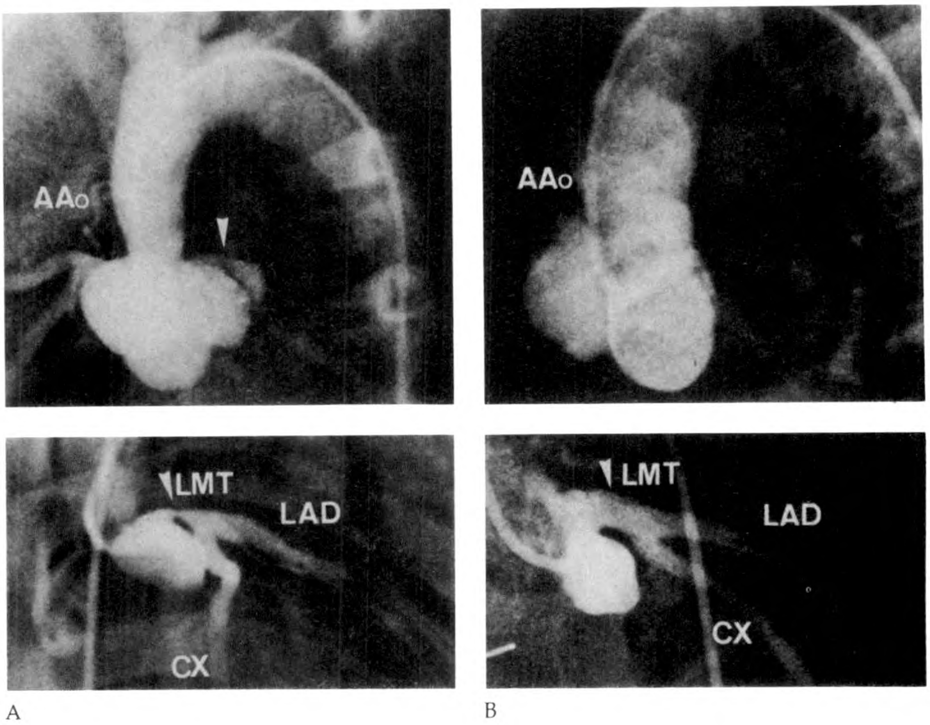

Fig 1. (A) Preoperative aortogram demonstrates the supravalvular aortic stenosis and localized narrowing of the left main coronary artery (LMT). *(B) Postoperative aortogram shows widened aortic root; left main trunk was also enlarged without obstruction.* (AAo = *ascending aorta;* CX = *circumflex coronary artery;* LAD = *left anterior descending coronary artery.)*

arately. The characteristic feature of this method is to offer the beneficial effects of both procedures to SAS and LMT obstruction in a single method, which should provide adequate relief of SAS by extended aortoplasty, excising the overhanging stenotic ring, and placing a wide double-flanged patch [7, 8].

For LMT lesions in SAS, intervention should be made at the time of operation for SAS, because the LMT lesion may cause serious early and late complications such as myocardial infarction or sudden death. Angioplasty has been advocated for LMT lesions even for acquired coronary artery disease [10], and it seems advisable when such lesions are seen in a patient with SAS because the atheromatous change may not be serious.

Regarding the material for aortoplasty and coronary angioplasty, autologous pericardium may be a preferable material because of its thin and pliable nature. Although the pericardium in this case was treated with glutaraldehyde solution to prevent shrinkage and aneurysmal change by pressure, the width for aortoplasty was rather

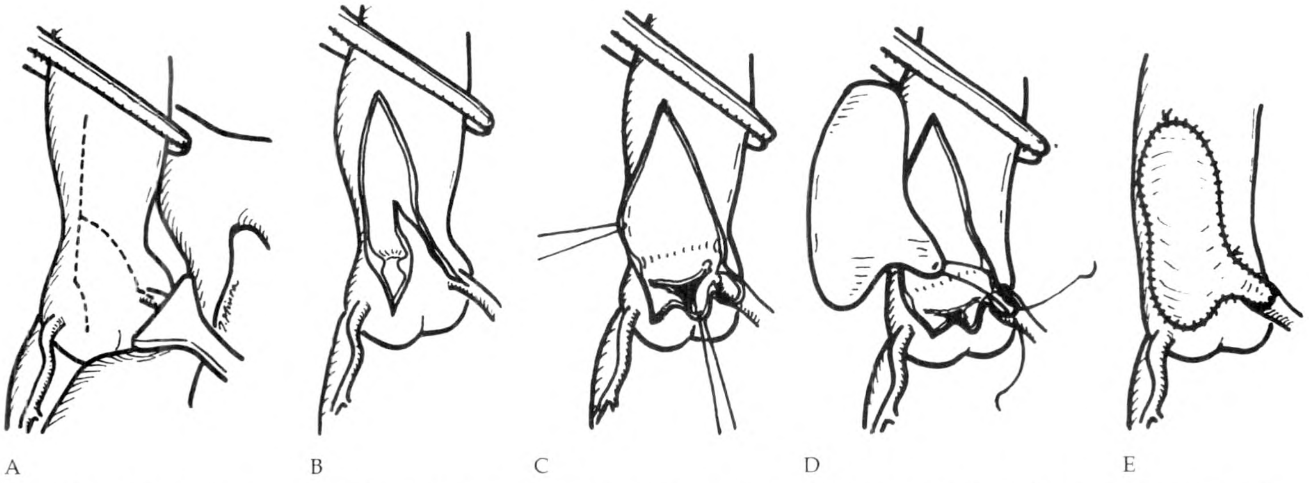

Fig 2. Operative technique employed with extended aortoplasty and simultaneous angioplasty of left main orifice with a single pericardial patch: (A, B) aortotomy, (C) aortotomy for left coronary sinus extending to the left main coronary trunk, (D) placement of double-flanged pericardial patch, (E) completion of the procedure.

large compared with the rest of the native aortic wall. Therefore, careful follow-up is required for restenosis as well for aneurysmal dilatation.

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INVITED COMMENTARY

Although obstruction to coronary blood flow has long been recognized as a possible complicating feature of supravalvular aortic stenosis, the mechanism of obstruction caused by narrowing of the coronary ostium itself has received little attention in the past. This article by Matsuda and associates reports yet a new variation on the technique of extended aortoplasty for supravalvular aortic stenosis when there is associated stenosis of the ostium of the left main coronary artery. Matsuda and associates used the inverted Y type of patch augmentation as popularized by Doty and co-workers [1]. One limb of the patch is used both to enlarge the left main coronary artery and to divide the supravalvular ring in a manner similar to the technique previously reported by my colleagues and me in *The Annals* [2]. However, in Matsuda's technique the incisions are placed in the left and right sinuses of Valsalva rather than in the right and noncoronary sinuses as done by Doty or in the left and noncoronary sinuses as done by ourselves. The advantage of placing the first incision across the stenotic area and into the noncoronary sinus, however, may be important as visualization of the coronary ostia is often difficult in the presence of severe narrowing, which limits the surgeon's view from above. After the area of narrowing is divided into the noncoronary sinus, a second, more precise incision can be made in the left coronary sinus and then into the ostium of the left main coronary artery itself. Although this latter approach may be somewhat safer, both techniques appear effective in relieving the supravalvular and coronary ostial stenoses.

The use of autologous pericardium treated with glutaraldehyde, as was done by Matsuda and also by us, provides a pliable, hemostatic patch material to augment the stenotic origin of the left coronary artery and is a helpful technical advantage. Late follow-up of our 2 previously reported patients reveals no evidence of aneurysmal dilatation of the pericardial patches and continued effective relief of both the coronary and aortic obstructions.

This report by Matsuda and associates again serves to emphasize this important mechanism of obstruction to coronary artery blood flow in patients with supravalvular aortic stenosis. Its recognition and relief, by whatever technique chosen, is essential for a satisfactory result.

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Vagal Schwannoma Involving Esophagus

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A rare case of vagal schwannoma (neurilemoma) with involvement of the distal esophagus is presented. Chronic progressive dysphagia was the only complaint related to the schwannoma. Barium swallow showed narrowing of the distal esophagus. The tumor was embedded in the wall of the esophagus. After biopsy and diagnosis of benign schwannoma, it was dissected from the esophageal muscle and mucosa. Diagnosis of benign schwannoma should be made before excision of the lesion to avoid an unnecessary esophagectomy.

(Ann Thorac Surg 1991;52:1334–6)

Schwannomas (neurilemomas) may arise in any nerve where Schwann cells occur. Schwann cell tumors of the peripheral nervous system were given the name neurilemoma by Stout and Carson in 1935 [1]. Such tumors characteristically produce few symptoms, exhibit slow growth, and rarely cause serious neurological disturbances. Schwannomas arising from the vagus nerve are rare; here, we report a vagal schwannoma involving the esophagus.

A 67-year-old white man was admitted with a mass in the right parotid gland (for biopsy and possible operation). An admission chest roentgenogram revealed a large mass in the right posterior and lower mediastinum. The patient stated that he had had some difficulty in swallowing which gradually became worse. Before hospital admission the patient had been receiving captopril, 25 mg twice per day, and glipizide, 5 mg twice per day. Barium swallow showed narrowing of the distal esophagus with a large right posterior mediastinal mass, displacing the distal esophagus to the left (Fig 1). A chest computed tomographic scan showed involvement of the distal esophageal wall by tumor. The mucosa of the esophagus was essentially intact (Fig 2). On physical examination there was no cervical lymphadenopathy. The right parotid gland was enlarged (3 × 3-cm), firm, and fixed. No lesion was seen in the oral cavity, pharynx, or larynx. The chest was clear, and the cardiovascular examination results were within normal limits. Laboratory test results, including electrolyte levels, white blood cell count, hematocrit, and urinalysis, were within normal limits.

Because of dysphagia and severe narrowing of the distal esophagus, it was decided to treat the mediastinal mass before biopsy and excision of the right parotid tumor. With the presumed diagnosis of a posterior mediastinal mass, most likely neurogenic in nature, the patient was explored through a right sixth intercostal space thoracotomy. At exploration there was a large movable mass located over the distal esophagus. There was no connection between the mass and intercostal nerves or the paravertebral area. The right vagus nerve along the esophagus entered the tumor mass and could not be traced until 2 cm below the mass. Part of the tumor was embedded in the wall of the esophagus. The medial third of the circumference of the esophagus was free. There was no obvious demarcation between the tumor and the other two thirds of the esophagus for approximately 4 cm. A biopsy and frozen section showed the mass to be benign schwannoma. With meticulous dissection the tumor was dissected from the esophageal muscle and mucosa, then it was excised in conjunction with a segment of involved vagus nerve. In the involved portion of the esophagus in which the esophageal muscle was cut, the mucosa partially bulged out between the edges of the cut muscles.

Pathologic analysis showed a 12.5 × 12 × 6.5-cm

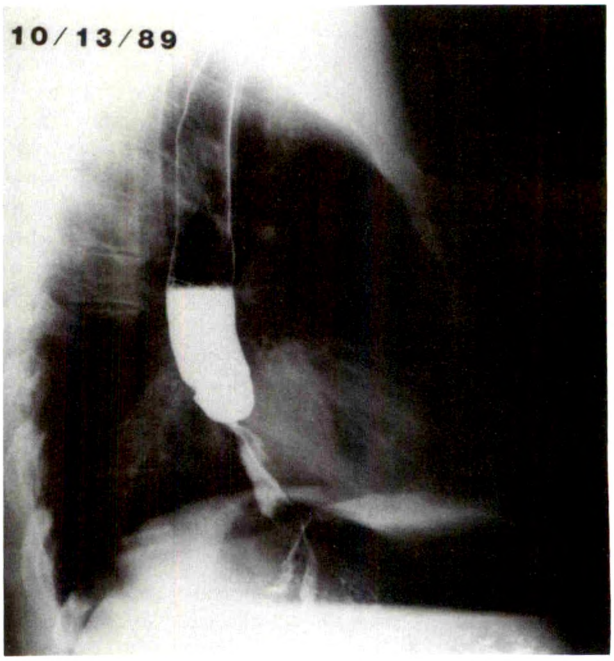

Fig 1. Lateral chest roentgenogram with barium swallow.

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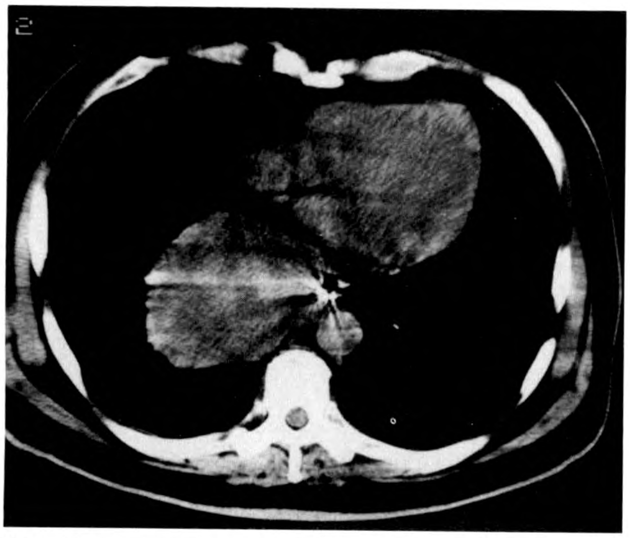

Fig 2. Chest computed tomographic scan of distal esophagus showing tumor involving the esophageal wall.

encapsulated mass weighing 580 g (Fig 3). Histologically, the tumor showed densely cellular spindle shaped cells in palisading arrangements (Antoni A pattern) with Verocay body formation, characteristic of benign schwannoma. Postoperative recovery was uneventful. A barium swallow 7 days after operation showed minimum postoperative changes and an otherwise normal esophagus (Fig 4).

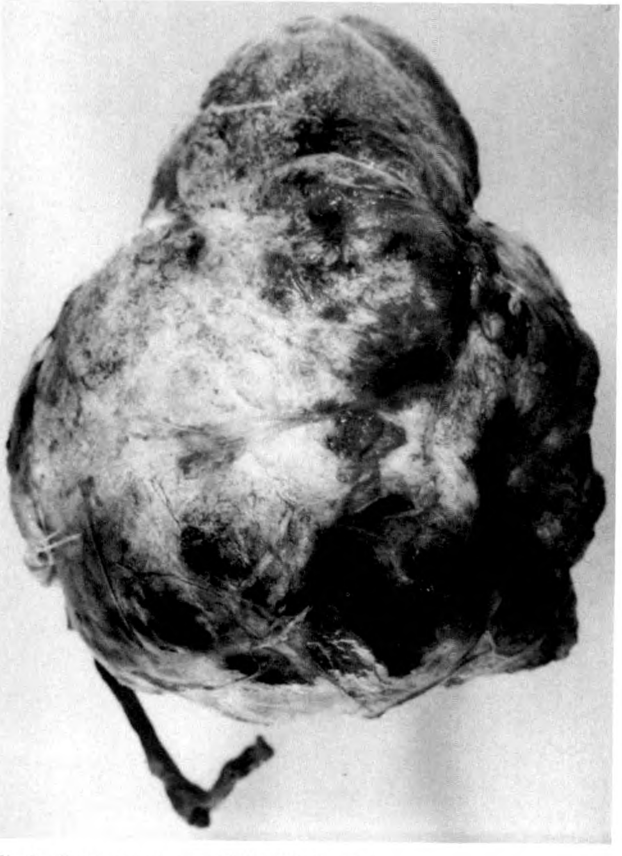

Fig 3. Gross appearance of the tumor mass.

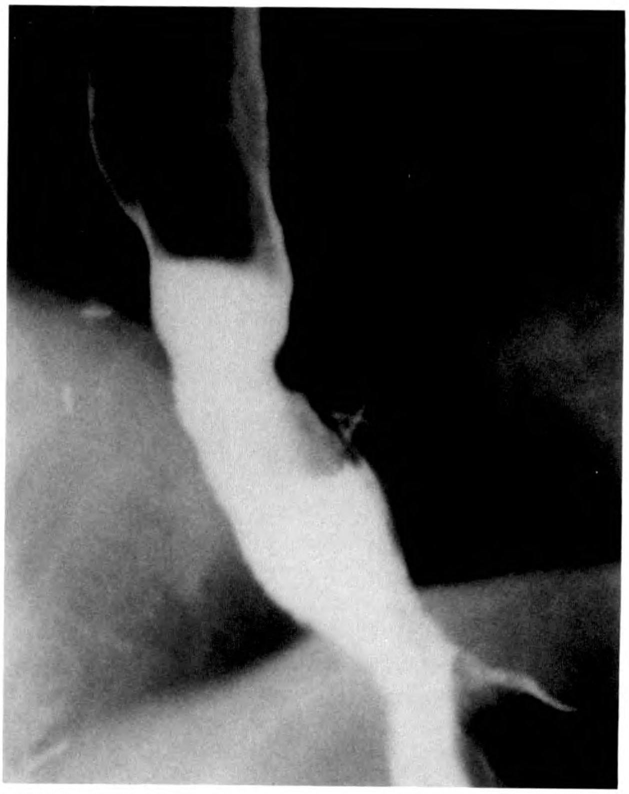

Fig 4. Barium swallow showing minimum postoperative changes and an otherwise normal esophagus.

Subsequently, after open biopsy of the parotid gland and diagnosis of undifferentiated carcinoma, the patient underwent parotidectomy and radiation therapy. At present he is asymptomatic. Chest roentgenograms remain normal.

Comment

Intrathoracic neural tumors are uncommon, but account for 75% of tumors of the posterior mediastinum [2]. Vagal schwannoma is very rare. In 1990 Dabir and associates [3] reported 2 cases of vagal schwannoma involving the thoracic portion of the vagus nerve adjacent to the aortic arch. In reviewing the literature they found 27 cases of vagus nerve tumors of which 9 were schwannomas. In the same year Heitmiller and co-workers [4] reported another case of intrathoracic vagal schwannoma, for a total of 12 cases. In 1956 Davis and Brown [5] reported an intrathoracic neurofibroma of the left vagus nerve at the level of the gastroesophageal junction associated with diaphragmatic hernia. The patient had no digestive tract symptoms. In our patient, schwannoma arising form the vagus nerve involved the esophagus, causing dysphagia. Peripheral tumors are first identified as a painless mass, whereas intrathoracic tumors are usually identified incidentally on chest roentgenograms. When symptomatic, or if associated with Von Recklinghausen's disease, malignant schwannoma should be suspected. In our patient dysphagia was due to extraluminal pressure by a large mass in an otherwise normal esophagus. These tumors are benign, but malignant degeneration has been reported [6]. Furrer and Fox [7] reported a case of fibrosarcoma of the left vagus sheath.

In our case, biopsy and frozen section before the excision of the lesion was very helpful. The tumor was large and embedded in the wall of the esophagus. Without a diagnosis of benign schwannoma, one may mistakenly attempt to resect the esophagus. Nasogastric tube within the lumen of the esophagus was used as a guide, and the tumor was dissected from the esophageal muscle and mucosa without injuring the mucosa.

Das Gupta and associates [8] reported that 49 or 16.1% of 303 patients with benign solitary schwannomas had a malignant tumor unrelated to peripheral nerves. Twenty-two patients gave a history of being treated for cancer before the development of a schwannoma. Sixteen patients had cancer concomitant with the benign solitary schwannoma, and cancer developed in 11 patients at a later date. Das Gupta and associates found this was a relatively high incidence of malignant tumors in otherwise normal patients. Although no conclusions regarding association of schwannomas with other types of cancers was drawn, they suggested that a careful study be made to ascertain if any such association does in fact exist. In our patient, carcinoma of the right parotid gland was concomitant with the esophageal schwannoma.

Enucleation of the vagal schwannoma from the esophageal wall is the treatment of choice. In the differential diagnosis of esophageal tumors, vagal schwannoma should be included.

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Use of a Pericardial Patch to Bridge the Conduction Tissue During Tricuspid Valve Replacement

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Tricuspid valve replacement carries a risk of causing complete heart block. To reduce this risk, a technique was developed to bridge the conduction system during prosthetic valve insertion in the tricuspid position. This technique, in which a pericardial patch is used to bridge the triangle of Koch, was performed successfully in 4 patients who underwent tricuspid valve replacement for Ebstein's anomaly of the tricuspid valve.

(Ann Thorac Surg 1991;52:1337–9)

Tricuspid valve replacement (TVR) is performed only when valve repair is impossible or has failed. Compared with tricuspid valve repair, TVR carries an increased risk for early and late mortality, especially in children [1]. Each of the currently available valve prostheses has limitations in the tricuspid position. Tissue valves become obstructive and undergo accelerated degeneration in young patients [2]. Mechanical valves are prone to thrombosis [3]. Kirklin and Barratt-Boyes [4] proposed using unstented aortic or pulmonary valve homografts in the tricuspid position to achieve valve durability and avoid the need for anticoagulation. McKay and associates [5] described a technique for TVR using an unstented pulmonary homograft. We have used the porcine valve as the valve of choice in the tricuspid position. Owing to the proximity of the conduction tissue to the tricuspid valve annulus, TVR is associated with a 5% early risk of the development of complete heart block and an additional 6% late risk of this complication [4]. The risk of the development of complete heart block is particularly high in patients undergoing simultaneous mitral and tricuspid valve replacement (25% will need a permanent pacemaker within 10 years postoperatively [4]) and in patients operated on for Ebstein's anomaly of the tricuspid valve [6] or for tricuspid valve endocarditis [4].

To avoid the conduction tissue during TVR, we have developed a technique that incorporates a pericardial patch to displace the suture line posterior to the triangle of Koch, thus bridging the conduction tissue.

Technique

Operation is performed using cardiopulmonary bypass, bicaval venous cannulation, moderate hypothermia, and cold blood cardioplegia for myocardial protection. After the appropriate prosthesis is chosen, suturing is begun anterior to the coronary sinus and continued with a continuous technique using Prolene suture material (Ethicon, Somerville, NJ) counterclockwise to a point anterior to the conduction tissue at the junction of anterior and septal leaflets (Fig 1). A glutaraldehyde-pretreated autologous pericardial patch is then sutured to the sewing ring of the prosthesis between the ends of the previous suture line. The pericardium is then fashioned into a triangular patch so that its inferior margin is sutured to the right atrium, aiming for a point just inferior to the coronary sinus. The suture line is then continued just behind the anterior lip of the coronary sinus orifice and posterior to the tendon of Todaro, coming back to the tricuspid annulus anterior to the anteroseptal commissure to complete the suture line (see Fig 1). In cases where there is

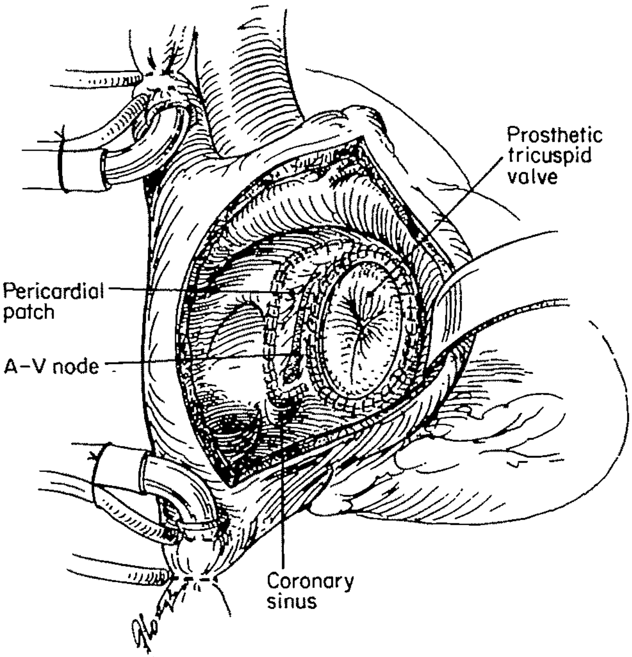

Fig 1. Valve insertion is begun anterior to the coronary sinus using a Prolene suture. The suture line continues counterclockwise and ends anterior to the commissure between anterior and septal leaflets. Thereafter, a glutaraldehyde-treated pericardial patch is sutured to the rest of the prosthesis sewing ring along the annulus of the septal leaflet. Then, the free margin of the tailored pericardial patch is sutured posterior to the vulnerable area of conduction tissue. (A-V = *atrioventricular.)*

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Table 1. Summary of Clinical Details

						Follow-up		
Patient No.	Age (y)	Sex	DO	Preop Diagnosis	Operation	Duration (mo)	FC	Results
1	58	F	4/83	Ebstein's TV ASD	TVR 35-mm Hancock, patch closure ASD, removal of endocardial pacemaker, permanent epicardial pacemaker	91	I	Preop intermittent CHB
2	3	F	12/84	Ebstein's TV ASD Severe MR	TVR 29-mm Hancock, MV repair, patch closure ASD	71	I	No conduction disturbances
3	5	M	5/85	Ebstein's TV ASD	TVR 31-mm Hancock, imbrication right ventricle, patch closure ASD	66	I	No conduction disturbances
4	6	F	8/88	Ebstein's TV RVOTO VSD ASD s/p right BTS RPA stenosis	TVR 29-mm Carpentier-Edwards Closure ASD, VSD Rt. modified Glenn Reconstruction RPA Takedown right BTS RVOT reconstruction Unroofing CS to LA	27	I	No conduction disturbances

ASD = atrial septal defect; BTS = Blalock-Taussig shunt; CHB = complete heart block; CS = coronary sinus; DO = date of operation (month/year); FC = functional class; LA = left atrium; MR = mitral regurgitation; MV = mitral valve; RPA = right pulmonary artery; RVOTO = right ventricular outflow tract obstruction; s/p = status post; TV = tricuspid valve; TVR = tricuspid valve replacement; VSD = ventricular septal defect.

concern about the remaining size of the coronary sinus orifice or when right atrial pressure is expected to remain high postoperatively, it may be advisable to unroof the coronary sinus into the left atrium and close the atrial septum and orifice of the coronary sinus into the right atrium. Using these techniques, the prosthesis is inserted bridging the conduction system and the coronary sinus is drained unobstructed into the lower-pressured atrium.

This technique for TVR has been performed in 4 patients with Ebstein's anomaly of the tricuspid valve in whom satisfactory repair could not be achieved. Their pertinent clinical details are summarized in Table 1. Patient 1 had intermittent complete heart block preoperatively, for which a permanent transvenous endocardial pacemaker was inserted. She underwent TVR and atrial septal defect closure, and the pacemaker was changed to epicardial. During follow-up, the length of periods of complete heart block was unchanged after operation.

The follow-up period for all patients was 27 to 91 months (mean, 64 months). Complete heart block has not developed in any of the other patients. All 4 patients are in functional class I, no valve re-replacements have been necessary, and there have been no perivalvar leaks or pericardial patch aneurysm formation found on echocardiography.

Comment

In patients undergoing TVR for organic rheumatic disease, usually the valve leaflets and annulus are thickened and suturing the prosthesis along the septal leaflet is safe [4]. However, when combined mitral and tricuspid replacement are performed, or when TVR is undertaken in patients with distorted or delicate annulus and leaflets (Ebstein's anomaly, endocarditis), the risk of injury to the conduction system is high and a modification of the current techniques seems necessary. Barnard and Schrire [7] suggested displacing the suture line along the septal leaflet posteriorly, behind the coronary sinus. The concern with this technique is the coronary sinus drainage into the right ventricle and distortion of the position of the prosthetic valve by the irregular suture line, which can bring the annulus or struts up against the conduction tissue. An alternative suggestion has been to place interrupted sutures along the septal leaflet annulus on the normothermic, perfused, beating heart; should heart block occur, the offending stitch is removed and repositioned [4].

We suggest that the technique described in this article is particularly suitable for patients undergoing TVR for Ebstein's anomaly when repair is not possible, for tricuspid valve endocarditis, or for patients undergoing combined mitral and tricuspid valve replacement. By insertion of the pericardial patch, the conduction system is bridged, the coronary sinus drainage into the atria is undisturbed, and the bioprosthetic valve lies in good position without impingement on the conduction tissue.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1994 may begin the recertification process in 1992. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is May 1, 1992. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Cosmetic Approach for Placement of the Automatic Implantable Cardioverter-Defibrillator in Young Women

Steven Curiale, MD, Lynda E. Rosenfeld, MD, and John A. Elefteriades, MD

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A surgical approach is described for a more cosmetically acceptable placement of the automatic implantable cardioverter-defibrillator in young women. The transvenous sensing lead and the vena caval spring electrode are placed through a small subclavicular incision. The left ventricular patch electrode is placed through an anterior minithoracotomy in the crease under the left breast. A small transverse incision in the left lower quadrant is used to place the generator under the external oblique fascia in the low abdominal wall. Minimal cosmetic impairment from incisions and hardware results.

(Ann Thorac Surg 1991;52:1340–1)

The automatic implantable cardioverter-defibrillator (AICD) (Ventak; CPI, Inc, Minneapolis, Minnesota) has dramatically improved the outlook for patients with life-threatening ventricular arrhythmias. Consequently, use of the AICD has burgeoned [1]. Whereas most patients are elderly or have advanced ischemic or idiopathic cardiomyopathy, a small number of AICD patients are young and have normal ventricular function. Although for any patient having placement of an AICD, protection from life-threatening arrhythmia must be the highest priority, cosmetic results may also be a factor, especially for young women who otherwise have led normal lives. A number of approaches have previously been described for placement of the AICD, but none is cosmetically favorable [2–4].

Material and Methods

We have encountered 3 young women with similar clinical circumstances who required placement of the AICD. Ages ranged from 30 to 35 years. One had suffered cardiac arrest and required full cardiopulmonary resuscitation, the second had required electrical conversion from hypotensive ventricular tachycardia, and the third had multiple episodes of syncope with pleomorphic ventricular tachycardia documented by ambulatory monitoring. Interestingly, all 3 were graduate students pursuing masters or doctorate degrees. All had normal ventricular function. Two had prolonged QT interval as the only identifiable etiologic factor for arrhythmia. Despite a clear understanding of the life-threatening nature of the cardiac condition, each young woman was concerned about the cosmetic impact of the placement of the AICD.

An approach was selected to minimize the cosmetic impact of AICD placement (Fig 1). A small subclavicular incision is used for transvenous placement of the sensing electrode and the superior vena caval "spring" electrode. This incision need only be several centimeters long, as no device other than these wires will be placed at this site. The sensing electrode is placed through the cephalic vein, and the spring electrode through subclavian vein puncture. An anterior minithoracotomy is used to provide access for placement of the left ventricular patch electrode. The incision is placed in the crease under the left breast. The patch is placed either extrapericardially or intrapericardially, depending on threshold determinations. A small transverse incision is placed low in the left lower quadrant along the lines of the skin creases for implantation of the AICD generator. The device is placed under the external oblique fascia, where a pocket is created by sharp and blunt dissection. The leads from the sensing electrode and the caval spring (passed through the free pleural space, in front of the diaphragm, and into the abdominal wall) are long enough to reach the left lower quadrant without extension or modification. In one of the 3 patients, thresholds with the spring-patch configuration were marginal, possibly because of treatment with the antiarrhythmic medication encainide, and a mirror-image thoracotomy was made in the area under the breast on the right side to allow placement of a second patch electrode.

Results

There was no instance of sensing abnormality, threshold problem, wound hematoma, or wound infection with this

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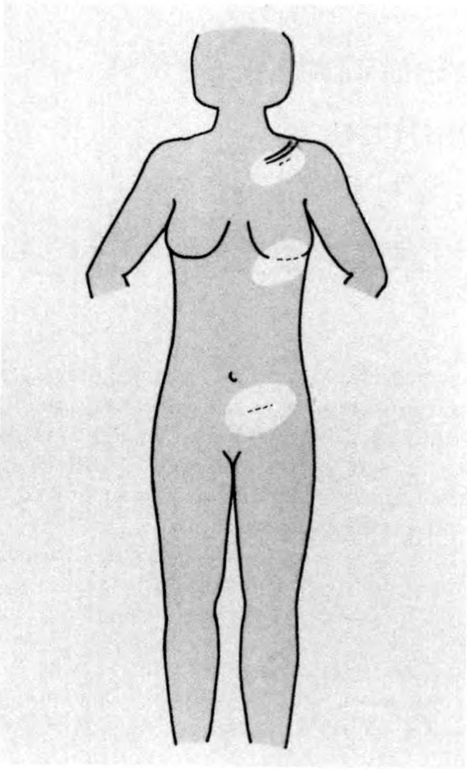

Fig 1. Cosmetic approach for implantation of automatic implantable cardioverter-defibrillator.

approach. All final defibrillation thresholds were less than or equal to 20 J. All 3 women were very pleased with the cosmetic result.

Comment

This approach produces an acceptable cosmetic result for these female patients. The subclavicular incision is hardly noticeable in the shadow under the clavicle. The anterior thoracotomy is hidden entirely by the breast. The left lower quadrant incision hides in the skin creases. The placement of the generator under the external oblique fascia minimizes the protrusion of the contour of the device seen with subcutaneous placement. Having the device reside low in the abdominopelvic region avoids the obvious cosmetic impact of the usual subcostal location of the generator. The overall cosmetic results have been acceptable even in a bathing suit. The generator lies well below the belt line, avoiding clothing problems resulting from placement of the generator higher in the abdominal wall.

Location of the skin incisions in protected sites and subfacial placement of the generator in this way minimizes the cosmetic impact of placement of the AICD in young women.

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Subclavian Vein-to-Right Atrial Bypass for Symptomatic Venous Hypertension

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A new surgical technique for bypassing subclavian vein thromboses in patients undergoing hemodialysis is presented. Subclavian vein stenosis or occlusion can occur after the use of temporary access catheters in subclavian vein dialysis. If this occurs in a patient with an arteriovenous access fistula of an ipsilateral upper extremity, venous hypertension, massive edema of the arm, and dysfunction of the access graft may result. In 2 patients with this condition, we successfully performed axillary vein-to-right atrial bypass, which resolved swelling and restored function of the access graft. This may be an appropriate surgical option for symptomatic venous hypertension in such patients.

(Ann Thorac Surg 1991;52:1342–3)

Once renal failure has been diagnosed in a patient, hemodialysis access is usually obtained by a percutaneous subclavian vein catheter, which is removed after an upper extremity arteriovenous access graft or fistula has been created and allowed to mature. This sequence of hemodialysis access may be repeated on the upper extremities many times as subsequent grafts fail and are revised. When an arteriovenous fistula is created in an arm that has a subclavian vein occlusion or stenosis from a previous venous catheterization, immediate venous hypertension and subsequent edema of the involved extremity may result. The swelling may be massive, uncomfortable, and refractory to conservative measures, but the real risk is the potential failure of the life-sustaining access graft after alternative sites have been depleted. Ligation of the hemodialysis access graft or fistula, which is probably the most frequently used treatment, can successfully aid in resolving the symptoms caused by the venous hypertension [1]. Although bypass around the occluded subclavian vein could potentially salvage the graft and resolve the edema, this has not become standard treatment because patency rates for most subclavian venous reconstructions in the absence of an arteriovenous fistula are very low.

We successfully used a new technique for bypassing subclavian vein thromboses in 2 patients who had been on long-term hemodialysis and had required multiple procedures for arteriovenous access. Both had thrombosed grafts that were deemed unsalvageable. In both patients, a graft was placed in the opposite arm; however, severe edema developed in the arm overnight despite appropriate postoperative care.

In patient 1, contrast venography was used to diagnose severe stenosis of the innominate vein. The patient underwent intraluminal balloon angioplasty; the edema resolved initially, but symptoms returned after 1 month. Repeat contrast venography showed recurrence of severe stenosis. Resection of the stenotic venous segment and spiral vein graft reconstruction were undertaken with initial success, but after 3 months, the patient was again seen with massive edema of the arm.

In patient 2, transfemoral digital subtraction angiography with loop and venous phases disclosed subclavian vein occlusion with collateral development around the site of occlusion.

Technique

To salvage an access graft and resolve upper extremity edema, a left axillary vein-to-right atrial appendage bypass is performed. With the patient in the supine position, an infraclavicular exposure of the axillary vessels is completed. The pericardium is entered by way of a median sternotomy. A tunnel from the infraclavicular incision through the second interspace into the left pleural space and into the mediastinum is created with a blunt clamp. Heparin is administered systemically (1 mg/kg). An end-to-side anastomosis is created between the inferior aspect of the vein and a 16-mm externally supported polytetrafluoroethylene graft by use of 5-0 polypropylene. The graft is passed through the chest and mediastinum to the right atrium. An end-to-end anastomosis between the right atrium and the free end of the graft is performed with use of 5-0 polypropylene (Fig 1). Clamps are then released, and protamine sulfate (4.5 mg/kg) is administered. A left chest tube and mediastinal tube are inserted. The sternum is wired and the wounds closed in layers. A 48-hour course of antibiotics is administered, arm elevation is encouraged, and a postoperative regimen of aspirin for 3 months is recommended.

Comment

Arteriovenous fistulas are used frequently in venous reconstruction of the lower extremities and the inferior

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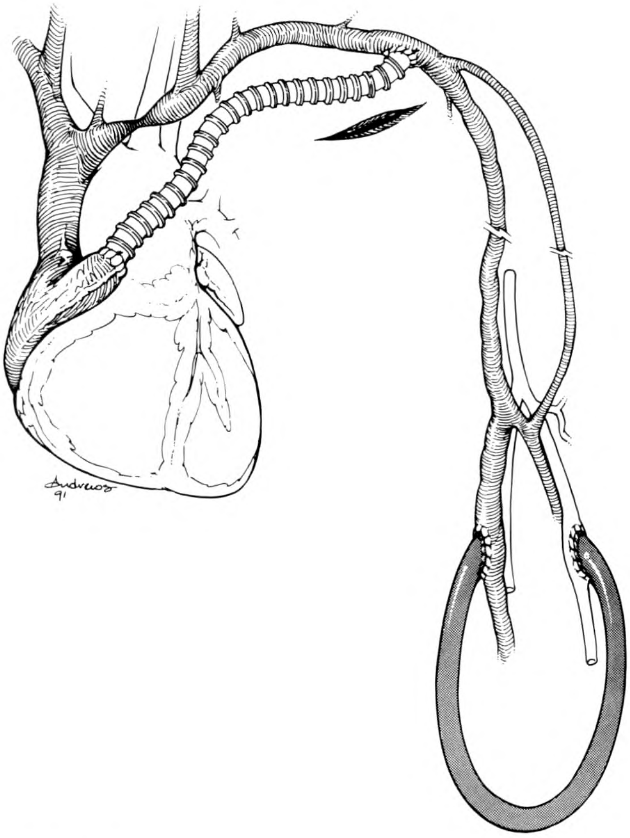

Fig 1. Axillary vein-to-right atrial appendage bypass with use of a 16-mm externally supported polytetrafluoroethylene graft. The right atrium may be approached by way of a median sternotomy or a right anterolateral thoracotomy.

vena cava as an adjunct to improve patency rates, but they are rarely applied to the upper extremity. Venous reconstruction of the subclavian or innominate veins in the absence of an ipsilateral arteriovenous fistula is known to frequently fail from low flow, small diameter of the conduit, or both.

In our patients, the new technique described was successful in eliminating upper extremity edema within 24 hours of operation. A minimal degree of morbidity was related to the median sternotomy. Both patients were discharged within 7 days of the procedure with the function and appearance of the hand totally restored to its preoperative condition. Postoperative follow-up exists for 18 months (patient 1) and 3 months (patient 2). Neither patient has had recurrence of arm swelling or loss of graft function.

The patency rates observed in these patients with preexisting fistulas, as well as those observed in other reports on patients with subclavian vein thrombosis and an ipsilateral arteriovenous fistula [2–6], suggest that the long-term patency of subclavian–innominate vein reconstruction may be enhanced by the creation of either a temporary or permanent arteriovenous fistula at the time of reconstruction. The placement of a temporary fistula has been shown experimentally to enhance patency in reconstructions of the superior vena cava as well [7], although long-term patency with spiral vein bypass grafts in the absence of an arteriovenous fistula has been documented [8].

The technique described in this report could be modified to include use of a more conservative right anterolateral thoracotomy instead of a median sternotomy for exposure of the right atrium. Although more experience is needed with this technique before drawing firm conclusions, the outcome in these initial cases supports our belief that axillary vein-to-right atrial bypass in the presence of an arteriovenous fistula is a reasonable treatment for patients with symptomatic stenosis of the subclavian vein.

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Repair of the Torn, Friable Right Atrium After Decannulation

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We describe a technique to repair the torn, friable right atriotomy cannulation site. It results in a tension-free, hemostatic repair.

(Ann Thorac Surg 1991;52:1344–5)

Right atrial cannulation is routinely used for the venous limb of the cardiopulmonary bypass circuit. A pursestring suture is placed at the right atrial cannulation site, usually the right atrial appendage. At the completion of cardiopulmonary bypass and with decannulation, the pursestring is tied. Minor oozing from this pursestring suture site can be controlled, if needed, by placing either a simple stitch or a pledgeted buttress suture. However, major hemostatic problems with atrial tears can occur if the atrium is distended or friable. This complication can be devastating, leading to excessive bleeding and even exsanguination. We present a technique by which adequate hemostasis can be achieved when a serious tear occurs after removal of a right atrial venous cannula.

Method

When the patient is placed on cardiopulmonary bypass, venous cannulation of the right atrial appendage is performed in the standard fashion using a nonabsorbable pursestring suture. Thereafter, the right atrial appendage is amputated distal to the pursestring suture and the venous cannula is placed through the right atriotomy (Fig 1). The patient is subsequently placed on cardiopulmonary bypass and the planned surgical procedure is performed.

At the completion of the operation and with successful weaning from cardiopulmonary bypass, the patient is decannulated. The venous cannula is removed and the pursestring suture is tied down. If the right atrium is torn as the pursestring is tied, the venous cannula is reinserted and cardiopulmonary bypass reinstituted. Bypass is temporarily interrupted to place a woven Dacron tube graft (6 cm in length by 24 mm diameter) over the venous cannula. The graft is sewn circumferentially to the right atriotomy site using a running stitch (see Fig 1). The patient is then weaned from bypass and decannulated.

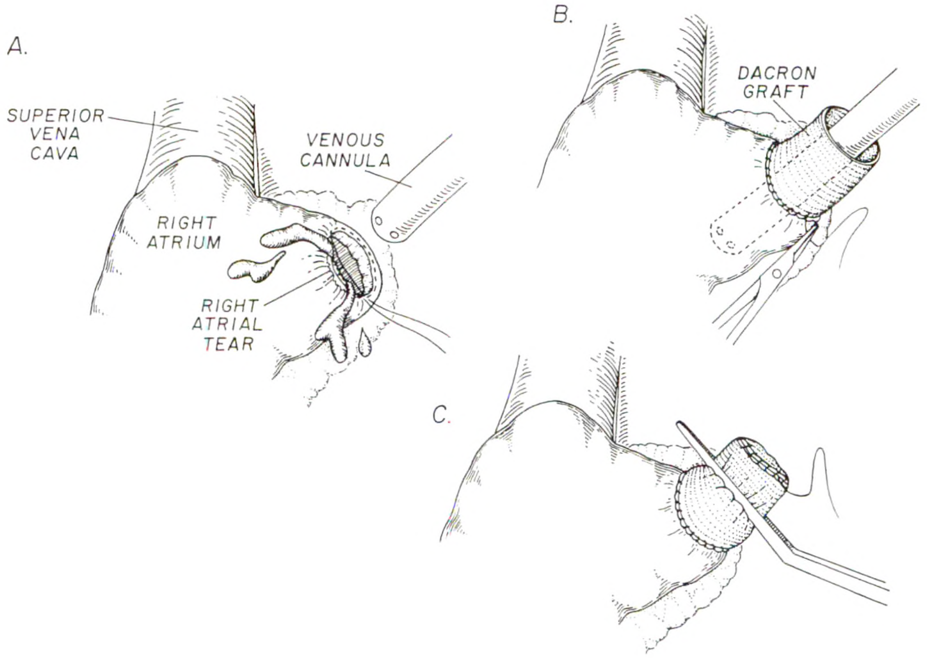

Fig 1. Technique for repair: (A) The venous cannula is reinserted through the right atriotomy and cardiopulmonary bypass is reinstituted. (B) Cardiopulmonary bypass is temporarily interrupted to introduce a Dacron tube graft over the venous cannula. The tube graft is then sewn circumferentially to the right atrium with a running suture. (C) The patient is weaned from cardiopulmonary bypass and the venous cannula is removed from the tube graft. A vascular clamp is applied to the tube graft as its free end is oversewn.

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The remaining open free end of the synthetic graft is then oversewn using an initial horizontal mattress suture and then a subsequent over-and-over simple running stitch.

Comment

Rarely, a tear is seen at the right atrial appendage cannulation site when decannulation from cardiopulmonary bypass is performed. It may result from either extreme atrial distention or a friable atrium, often seen in elderly patients. The resultant hemorrhage from this type of tear can be excessive, even leading to exsanguination. Attempts at repairing this tear with a simple suture technique (pursestring suture, simple suture, or pledgeted suture) may obtain hemostasis. More commonly, these maneuvers extend the tear. We present this reparative technique as a safe and effective method in repairing this complication of an initial tear seen at the atrial venous cannulation site with decannulation.

Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas, TX, on February 7, 1993. The closing date for registration is August 1, 1992.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

A candidate applying for admission to the certifying examination must fulfill all the requirements of the board in force at the time the application is received.

Please address all communications to the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Replacement of the Aortic Valve With a Pulmonary Autograft: The "Switch" Operation

Donald Ross, FRCS

Harley Street Clinic, London, England

The transfer of the patient's own pulmonary valve to the aortic position developed from our earlier work with aortic homografts. The valve shows no progressive tissue failure and offers the prospect of a permanent valve replacement for young people. Like homografts, the valves can be inserted in the subcoronary position or as a root replacement. In infants and growing children root replacement should be used to benefit from the valve's growth potential.

(Ann Thorac Surg 1991;52:1346–50)

In this procedure the patient's own pulmonary valve is removed together with the main trunk of the pulmonary artery and used to replace the aortic valve. The valve can be inserted in an orthotopic subcoronary position or used as a root replacement with reimplantation of the coronary arteries (Fig 1).

The operation was devised and applied in 1967 [1], 5 years after the first homografts [2] and at the time when degenerative changes in homografts were first becoming evident [3]. It seemed clear then that the only prospect of a permanent valve replacement was to use either a living homograft plus immunosuppression or a living autograft having the same anatomical configuration as the aortic valve.

An obvious choice was the patient's own pulmonary valve, and there was experimental evidence to support this view [4, 5]. This had shown that the autogenous pulmonary valve would function as a mitral or aortic substitute in dogs. An unknown factor was whether the transposed pulmonary valve would support aortic diastolic pressure in humans. This was soon decided clinically, and there has never been an acute or early failure in any valve in more than 350 insertions. This clinical observation has been substantiated by subsequent biomechanical studies [6].

The primary indication for the operation is to provide a permanent valve replacement in young patients (approximately 30 years or less) who, with a mechanical valve, would be subject to perhaps 40 years of embolic and hemorrhagic hazard plus continuous cardiac supervision or, in the case of a bioprosthetic valve, the prospect of three to four operations with escalating hazard. More recently an increasingly important indication for the operation has been young infants or children with aortic

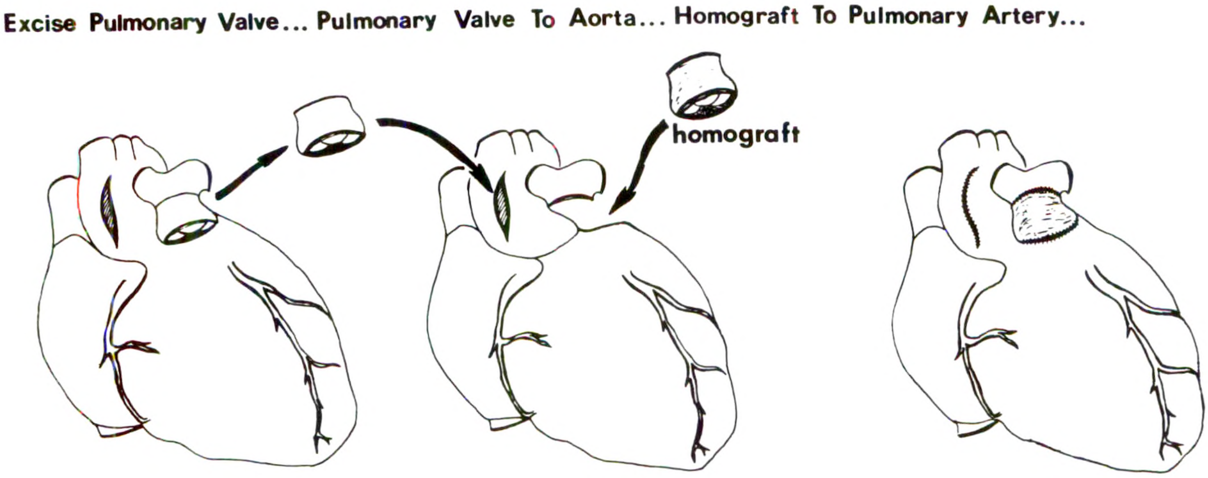
Excise Pulmonary Valve... Pulmonary Valve To Aorta... Homograft To Pulmonary Artery...
homograft

Fig 1. Steps in the operation of pulmonary autograft translocation to the aortic area.

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Address reprint requests to Mr Ross, 25 Upper Wimpole St, London W1M 7TA, England.

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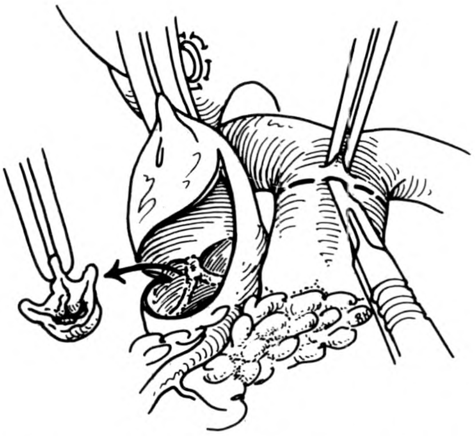

Fig 2. The pulmonary artery is divided at the point that has been marked before the start of bypass.

valve disease in whom there is not only the prospect of a permanent valve but also one with growth potential [7, 8; Elkins R, personal communication, 1991].

Principles

The first step is to decide whether the case is suitable for an autograft operation. This implies that the pulmonary valve will suit the aortic ring. Because the cardiac output of right and left ventricles is identical, it follows that the size of the right and left outflow orifices should be similar; this is in fact the case in practical terms. Also, the excised pulmonary autograft is very elastic and conforms easily to the new dimensions of the aortic root.

If the patient has a dilated or hypoplastic aortic root the autograft can be inserted as a root replacement, but if the pulmonary artery is markedly distended as a result of pulmonary hypertension it may be better to abandon the operation in favor of a homograft. In any event one is not committed to the operation until the pulmonary valve has been inspected.

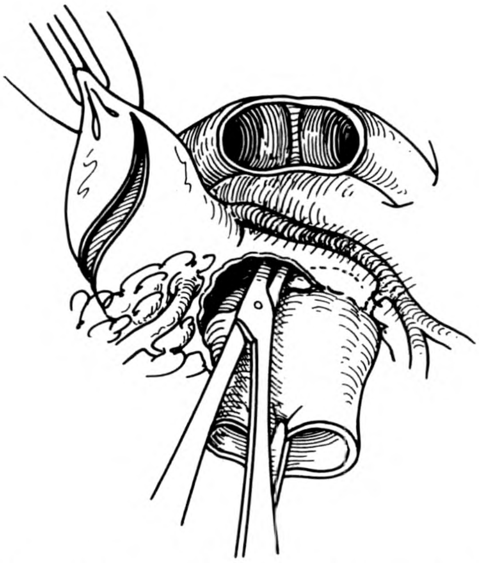

Fig 3. The divided pulmonary trunk is dissected from above down until right ventricular muscle is exposed.

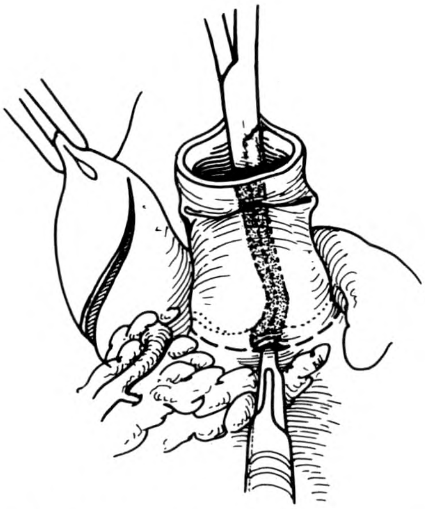

Fig 4. A curved artery forcep is passed through the pulmonary artery to mark the base of the cusps. An incision is then made in the overlying muscle.

Technique

Subcoronary or Orthotopic Implantation

When the chest is opened, the relative size of the pulmonary and aortic roots are appraised and a small scissors

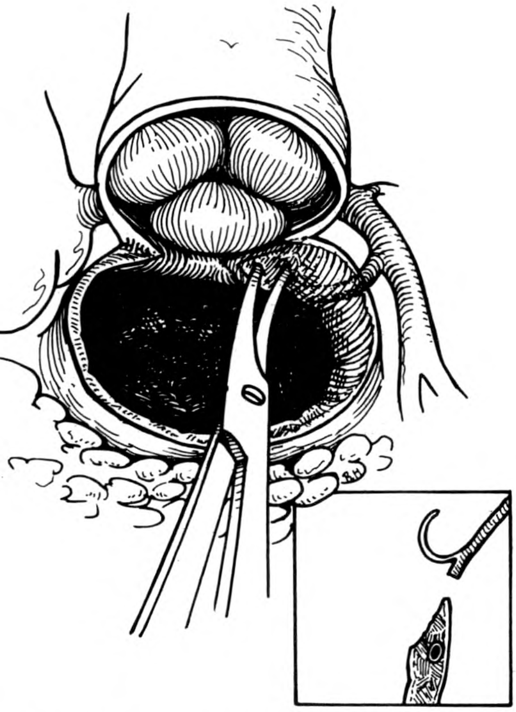

Fig 5. The autograft is excised from the right ventricular outflow. In the region of the first septal artery the dissection is obliquely angled away from the artery. Inset shows the dissection angled out in right ventricular muscle.

cut is made in the adventitia of the pulmonary artery just below its bifurcation and well above the commissures. This is a guide to the site of subsequent transection before the pulmonary artery collapses and familiar landmarks are lost.

It is perfectly satisfactory to establish bypass with a single venous cannula as the tricuspid valve will remain competent, but double cannulation is acceptable.

The aorta is opened by an oblique incision into the noncoronary sinus exactly as for homograft replacement, and the aortic valve is inspected and excised. It is usual to introduce flexible, plastic balloon-tipped coronary cannulas at this stage through which blood, and subsequently, cardioplegia solution, can be perfused.

Once the surgeon has decided on an autograft, a transverse incision is made in the pulmonary artery at the point selected and the valve is inspected from above to exclude a diseased or bicuspid valve (Fig 2). If the valve is satisfactory, the pulmonary artery is transected just below its bifurcation and dissected from above down with scissors. The plane of the dissection is continued posteriorly keeping close to the pulmonary artery wall until right ventricular muscle is fully exposed (Fig 3). There is often a small area of dense adherence between pulmonary artery and aorta, possibly where the truncus had divided, but most of the dissection is through loose areolar tissue.

It is convenient during this phase of the dissection to use conventional coronary perfusion. Because the dissection is relatively bloodless, this will indicate whether the plane of dissection is too close to the left coronary branches. Any small capillary or venous oozing can be gently coagulated during intermittent perfusion of the coronary arteries.

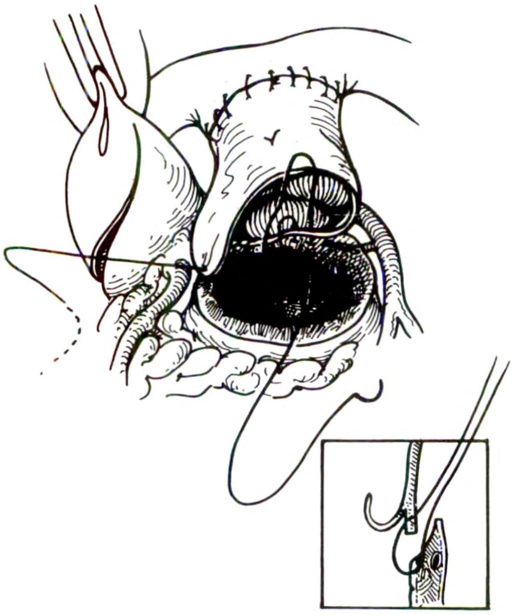

Fig 6. A proximal running suture takes tension off the subsequent sutures in the region of the first septal artery. Inset shows superficial bites picking up endocardium and some muscle.

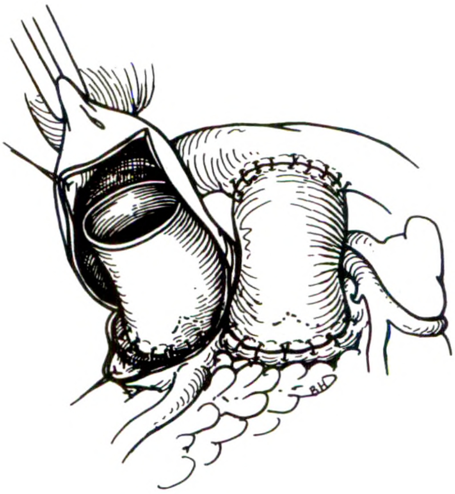

Fig 7. The untailored autograft is fixed in place in the aortic root with multiple interrupted 4-0 sutures. It can then be tailored appropriately or anastomosed side-to-side to the coronary arteries.

By looking down the inside of the transected pulmonary artery it is possible to see the sinuses and judge the position of the incision on the right ventricular outflow muscle so as to avoid the cusps. A small stab incision is made at an appropriate level (Fig 4). The muscle incision is then developed across the outflow so as to run about 2 to 3 mm below the base of the sinuses. Over the posterior wall, and particularly toward the left coronary area, the valve is enucleated obliquely with a minimum of muscle from below up to avoid damage to the septal branches of the anterior descending artery [9] (Fig 5).

Once the autograft has been removed it is customary to let it lie in the blood within the pericardial cavity or in a blood-filled container. A further perfusion of blood down the left coronary orifice will identify any bleeding points that need attention, after which cardioplegia can be induced.

A suitable pulmonary homograft [10, 11] is chosen, erring preferably in the large size; here size is not particularly important. The pulmonary trunk of the homograft is divided about 1.5 cm above the commissures, and excess muscle is trimmed across the base of the sinuses, leaving more anteriorly to be matched to the ventriculotomy later. The pulmonary homograft should not be too long and is inserted in its normal anatomical position with two sinuses anteriorly and one posteriorly.

The distal anastomosis to the pulmonary artery bifurcation is made with a running 4-0 Prolene suture (Ethicon, Somerville, NJ), starting at the midpoint of the carina between the right and left pulmonary arteries. It should be locked at least twice to avoid a pursestringing effect.

The anastomosis to the right ventricular muscle starts at the midpoint posteriorly using a running 4-0 Prolene suture. The objective here is to take all tension from the area of the septal artery by carrying this suture toward the operator and taking good, deep, full-thickness bites. It is locked proximally.

With the approximating tension taken by this first suture line, the adjacent edges of ventricle and homograft

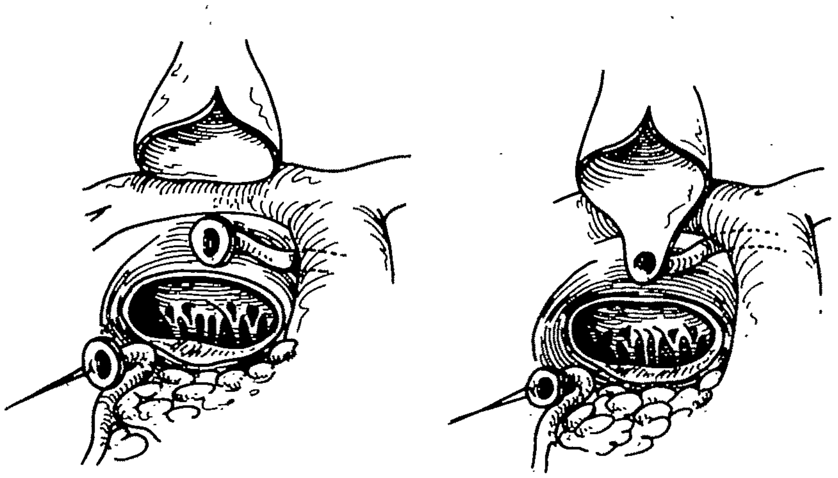

Fig 8. When the autograft is inserted as a free-standing root the aorta is divided, and the coronary arteries are preserved on buttons or on a tongue of aorta.

will lie in close proximity (Fig 6). The suture is then continued in a direction away from the operator, picking up only endocardium and approximately half of the right ventricular wall thickness until the posterior suture line is complete and locked at both ends. The anterior layer is then completed after the homograft muscle is trimmed appropriately, and full thickness bites are again used. Venous pressure is raised just before final closure to expel air from the right ventricle. Attention is then directed to the aorta.

The autograft is then trimmed of excess fat, taking care not to buttonhole the thin pulmonary artery wall, and the lower edge of the autograft muscle is trimmed away in a straight line about 1 mm below the lowest point of the cusps. Excess muscle outside the endocardium can be pared away. No attempt is made to sculpt the sinuses, and the pulmonary artery wall is kept intact and held firmly in a clamp during insertion of the autograft.

The subsequent steps (Fig 7) are identical with the technique for inserting an aortic homograft [12, 13], but the strength and elasticity of the living autograft tissue make handling and insertion of the sutures much less hazardous than with a friable homograft. Also, because the pulmonary valve is anatomically symmetrical, its placement within the aorta does not have to conform to any anatomical configuration but is oriented at the convenience of the surgeon.

Autograft Root Replacement

Apart from the theoretical advantages of root replacement [14, 15], which ensures an entirely competent valve and a consequent reduction in turbulence and probable increase in cusp durability, the insertion of the autograft valve in children should probably always be as a root. This allows for growth of the valve ring and cusps without any surrounding tissue restraints. The technique is simpler than conventional subcoronary placement and applies equally to autografts and homografts.

Once the autograft has been harvested and the valve removed, the aortic root is exposed more clearly by

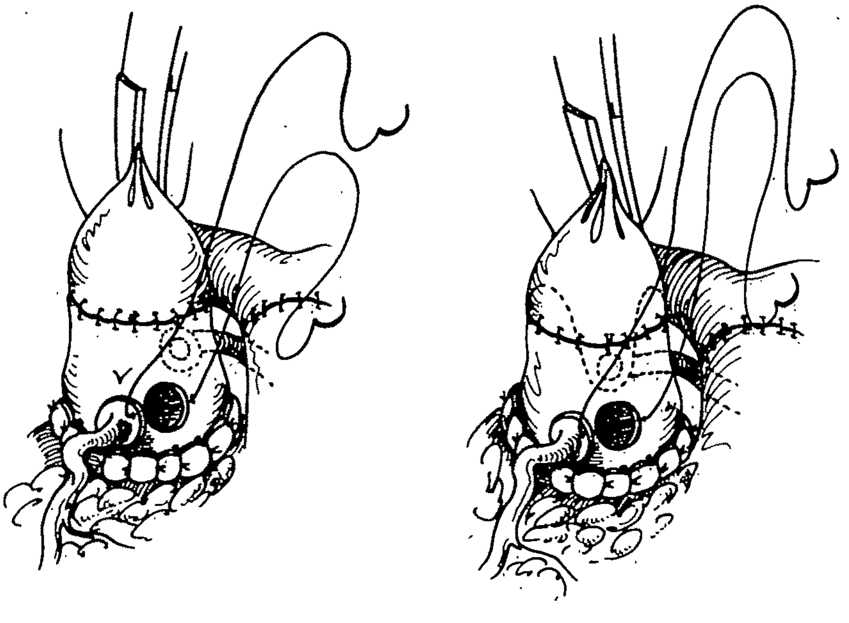

Fig 9. The lower interrupted sutures can be tied over a thin strip of Teflon or pericardium to aid hemostasis.

transection of the aorta above the left coronary artery or, as in the Elkins modification, the left coronary orifice is retained on a tongue of aortic tissue (Fig 8).

A curved blunt probe is passed down the left coronary artery to determine the lie of the left anterior descending and circumflex branches. The left coronary artery with a button of aorta is well mobilized so as to avoid subsequent tension on it. The right coronary artery is cleared and mobilized also with an adequate button of aortic tissue and retracted with a suture.

The aortic root is then cleared in a roughly horizontal manner, leaving a rim of aortic tissue for attachment of the graft or along the top of the subaortic curtain of the mitral cusp and skirting along the upper margin of the membranous septum to avoid conducting tissue.

The lower suture line is made with multiple interrupted 4-0 Prolene sutures. These can be conveniently tied over a thin strip of Teflon or autogenous pericardium to ensure hemostasis (Fig 9). However, if growth is an important factor or infection is suspected, the Teflon should be omitted.

The coronary arteries are reanastomosed to corresponding holes made on the autograft, the surgeon having noted or marked the position of the commissures to avoid damage to them and the cusps. Where the left coronary artery is retained on a tongue this is incorporated in the upper suture line with running 4-0 Prolene.

As an added precaution it is a good policy to complete the left coronary and aortic anastomosis first and temporarily open the aortic clamp. This indicates the most appropriate position for attachment of the right coronary, which may vary from case to case.

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Thoracoplasty: The How and the Why

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Starting as a procedure designed to obliterate empyema spaces by decostalization, thoracoplasty evolved along two lines: (1) the Alexander type that quite successfully collapsed the cavities of pulmonary tuberculosis until supplanted by antimicrobial chemotherapy and pulmonary resection, and (2) the Schede type that was the springboard for still-evolving techniques for reducing intrathoracic spaces or filling them with living tissue implants. It is the therapeutic objective that determines the technical details of any proposed thoracoplasty.

(Ann Thorac Surg 1991;52:1351–3)

Customary format would have this essay describe the evolution of the Alexander thoracoplasty and the thorax-resection of Schede, because these names come to mind when thoracoplasty is mentioned. Seeking the origin of these operations, I go back to the late 19th century to offer an operation that simply resected ribs over empyema spaces (to bring the walls into apposition) as the prototype from which both the Alexander and the Schede thoracoplasty derive.

Without claiming to assign strict priority for that procedure, the operation that decostalized the chest wall over empyemas can be referred to as an Estlander thoracoplasty. To quote Paget, "With all these operations, two names are chiefly associated—those of Estlander and Schede. The term, 'Estlander's operation' is very loosely used: nor was he the first to do free resection in these cases" [1]. Similar attitudes are echoed by Graham and associates [2], Meade [3], to some extent Alexander [4], and others.

To follow the evolutionary steps that led from that primitive thoracoplasty to the one ultimately offered by Alexander would be laborious, starkly historical, and monotonously repetitive because the contributions of many surgeons would require detailed description to make clear the variations in technique. Included in such technical variations, I would consider the location of the ribs to be resected, the number of ribs to be resected, as well as where to begin the operation (from above downward or below upward), what constitutes an adequate operation (when to quit), and finally, whether to do the job all at once or break it up into stages. Ultimately, the impact of these variations on morbidity and mortality would need to be assessed along with their overall effectiveness.

Fortunately, this has all been done and I can do no better than point to Alexander's two books in which we can find this displayed in meticulous detail [4, 5].

By way of background, the 1925 book was written under very depressing circumstances. Alexander was at "bedrest" on a Bradford frame and in a plaster cast at Saranac Lake, New York. A pleural effusion and later Pott's disease developed in him after he joined the Surgical Faculty of the University of Michigan in 1920 [6]. As if stimulated by these formidable handicaps, he devised ingenious mechanical contraptions to hold his working materials, including books, where he could make use of them [6].

At this time, American surgery was fully competitive, including the treatment of tuberculosis in areas other than lung; however, little enthusiasm seems to have been generated along these lines, particularly for the "rib operations." "Only about 300 cases of pulmonary tuberculosis have been reported as having been operated upon by 17 surgeons in the United States during the past seven years" [5]. The greatest activity in surgical therapy for pulmonary tuberculosis seemed to be emanating from Europe, notably Germany, Scandinavia, and Switzerland.

Handicapped though he was by the awkwardness and immobility imposed by the treatment of his illness, Alexander nevertheless put together an outstanding, yet workable, summary of surgery for pulmonary tuberculosis. In support of this view, on the title page of the 1925 book we find: ". . . this monograph has been awarded the 1925 quinquennial Samuel D. Gross Prize by the Philadelphia Academy of Surgery."

His knowledge in the field at this time was not all gleaned from reading. After military service in France he studied under Leon Berard, who was applying surgical methods to the treatment of pulmonary tuberculosis. In recognition of this period of study, he received a Certificate in Surgery and Anatomy from the Université de Lyon in 1919 [6].

What other surgical experience he may have had in the interim is not clear, but he did publish four articles in 1924 and 1925, including the description of his periosteal elevator depicted in both of his books [5]. In 1926 he returned to the University of Michigan, engaging in general and thoracic surgery until 1928. In 1928 he became involved in thoracic surgery on a full-time basis and initiated his thoracic surgical training program [6].

By this time the value of adding collapse to bedrest in management of pulmonary tuberculosis was well recognized, and the exceptional value of surgical collapse was

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looming stronger in the minds of many. The relaxation provided by collapse reduced the elastic recoil in the lung, permitting cavity walls to approach each other, magnifying the tendency to contracture inherent in the cicatricial process [5]. It follows logically that reducing the circumference of the chest cavity by rib resection would solidify this objective and in addition make it permanent.

With the Estlander thoracoplasty as a model, the search was on for the proper operation to deal with the cavitary lung lesions of tuberculosis. It appears to be a time of groping for the correct operation that would achieve what was believed could be achieved, while still experiencing substantial morbidity and mortality. (14% to 30% mortality, securing only 36% clinical cures with 24% considered improved. Some 20% died from the progression of their disease [4].)

Enter now Alexander along with others who, determined to increase the percentage of cavity closures, began independently circa 1928 to resect greater lengths of the uppermost ribs and lesser lengths of such other next lower ribs as required. This, carried out in multiple stages, became the "modern posterolateral thoracoplasty."

The following outline of the technique is taken from Alexander's own description and the practice on his service: A periscapular incision permitted elevation of the scapula. The upper digitations of the serratus anterior muscle were separated. The third and second ribs were resected subperiosteally from the level of the transverse process to approximately the mid and anterior axillary lines, respectively. The first rib and its cartilage was resected along with the tip of the transverse process to the sternum. The second and third transverse processes with the underlying rib were resected to the level of the lamina of the vertebra. The periosteal beds were dried and rubbed with 10% formalin to delay regeneration. The wound was closed without drainage.

In 3 weeks the incision was opened and all reattachments in the subscapular space were freed. The fourth and fifth ribs were subperiosteally resected, along with the corresponding transverse processes. The anterior stump of ribs two and three were further shortened. The anterior end of ribs four and five were brought into line, shelving posteriorly. The rib beds were treated with formalin. The wound was closed with drainage.

In another 3 weeks the incision was again opened and all attachments within the subscapular space were freed up. Ribs six and seven were resected from the level of the vertebral lamina, bringing them in line with the shelving anterior stumps. The rib beds were treated with formalin. The wound was closed with drainage. An anterior stage was added when necessary [4].

This thoracoplasty, therefore, had as its objective the collapse of tuberculous cavitary lung disease in the classic position, namely, apical and posterior.

Although eminently successful in its day, thoracoplasty was supplanted by other therapy in due time. It was preeminent in the decades of the 1930s and 1940s, fading out thereafter, as the use of antimicrobials clearly permitted uninhibited pulmonary resection or avoided surgical intervention altogether in many cases.

The other operation evolving from the Estlander stem developed as a result of failures following its use, even though the extent of decostalization could be carried to or well beyond the margins of the empyema space.

In contrast to a thoracoplasty for pulmonary tuberculosis, it is blood loss or trauma and not the flail chest that limits the extent of decostalization at any one time, because the underlying lung is imprisoned by the visceral coat of the empyema and the costal wall of the empyema is thickened and stiff. It is clear that obliteration of the residual empyema space, after an Estlander thoracoplasty, is by cicatrization as the parietal wall is pulled in onto the visceral wall. It requires little imagination, however, to realize that sulci can persist, particularly along the diaphragmatic gutter or along the spine, militating against ultimate closure.

For this reason, maneuvers other than the mere subperiosteal resection of ribs to free up the parietal wall were required. The next logical step in this direction is exemplified by the Schede thoracoplasty or his thorax-resection.

Paget provides a description in English of the technique as offered by Schede at the Medical Conference at Vienna in 1890 [1]. I give the salient points: Using a large U-shaped incision carried to rib level, a flap of skin and extracostal soft tissue is created and raised. All the ribs over the cavity are resected subperiosteally, from a point a little beyond their angles posteriorly to the costochondral junction anteriorly. The ribs are divided at about their midpoint with bone forceps. The cut ends are drawn apart and broken off. Anteriorly they break at the costochondral junction, and posteriorly they almost always break at or close to the tubercle. (I say—why not cut them sharply instead of breaking them?)

The thickened pleura and intercostal muscles over the whole cavity are cut away with strong scissors. To avoid bleeding from the intercostal artery twice, he divided the posterior boundary first. Each vessel was compressed between thumb and forefinger just before cutting and immediately ligated afterward. The previously developed soft tissue flap is then secured into the unroofed space.

It is obvious that the Estlander thoracoplasty is to be carried out over an externally drained pleural space. To do so over an undrained space is to omit a very fundamental step in empyema management, without which the need for thoracoplasty collapse cannot be properly determined.

Parenthetically, on the modern scene, if the septic features of a chronic empyema can be brought under control clinically through the use of antimicrobials and if excision of the pleural residual by decortication is deemed possible, preliminary drainage is not considered mandatory. Historically, it must be remembered that in the pre-antimicrobial era, it was strongly recommended that "pure" tuberculous empyema not be openly drained. To quote Alexander, "tube drainage rarely favorably affects the active tuberculous lesions in the wall of a pure tuberculous empyema and sooner or later drainage almost invariably results in a complicating, harmful pyogenic infection of the pleural cavity, the organisms passing from the skin through or around the drainage tube into the pleural cavity" [4]. Thus, the "pure" tuberculous empyema underwent thoracoplasty collapse without preliminary drainage.

Accepting the above constraint, collapsing an undrained empyema results in mere displacement of the empyema pocket to whatever degree permitted by the underlying lung, whereas the empyema remains essentially unchanged. Aspirating the pleural content may mitigate this effect, but the chest wall has been deformed and ventilatory function is reduced proportionately.

Continuing now the evolutionary perspective, maneuvers to coax the lung into contributing to the obliteration of space by expanding to whatever degree possible came next. (Schede experimented with this concept in his day.) These consisted of efforts to thin the visceral peel by shaving or cross-hatching to weaken the involucrum restraining the lung. Such efforts led directly to instances of decortication by several surgeons before the end of that century.

Decortication became an established procedure for managing organizing and infected hemothorax during World War II. Although the pathogenesis of suppurative pleuritis and that of an organizing and infected hemothorax are different, decortication providing for filling of the empyema space by an expanded lung is, of course, the proper answer if possible.

The admittedly brutal appearance of the Schede operation, particularly over large spaces, has spawned efforts to sterilize those spaces without bronchopleural fistulas through debridement, mechanical cleansing, and antimicrobial instillation. Also, further modifications, including preservation of intercostal tissues and the use of living tissue implants to close bronchopleural fistulas or to obliterate space, form a modern and still-evolving chapter on this subject. This, however, lies outside the scope of this essay.

In brief summary, the term *thoracoplasty* conjures up a veritable kaleidoscope of operations from which the skilled surgeon will choose that particular modality or combinations thereof designed to adequately cope with the anatomicopathologic requirements of the case at hand.

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Blood Cardioplegia: A Review and Comparison With Crystalloid Cardioplegia

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The Oxford International Symposium on myocardial preservation provided an appropriate milestone and impetus to survey one aspect of operative myocardial preservation, namely blood cardioplegia, and to contrast it with the more popular crystalloid cardioplegia. This review is by no means complete or exhaustive but represents my best effort to summarize important information that has accumulated in the literature as blood cardioplegia, and our understanding of it, has evolved. It is appropriate to compare blood and crystalloid cardioplegia with respect to biochemical and physiological differences. Clinical comparison has been limited, for the most part, to randomized studies, and a number of differences and details of clinical management of the two techniques have been omitted, either because they seemed unimportant or there was no good information that would allow an objective comparison of their significance. Hopefully, the reader will recognize the intent to focus on meaningful differences and similarities between the two techniques and to present them fairly.

(Ann Thorac Surg 1991;52:1354–67)

Blood cardioplegia, as practiced today, was reintroduced in 1977 [1, 2], having been first used by Melrose and associates in 1955 [3]. This initial effort involved potassium concentrations in the range of 245 mEq/L, which were subsequently found to cause myocardial damage [4–8]. This injury might have been avoided had hypothermia, rather than normothermia, been used [9].

After 1977, blood cardioplegia was not widely employed. In the last 5 years, its use has been expanded coincident with (1) a better understanding of the physiology of blood as a vehicle for cardioplegia, (2) randomized clinical studies favoring blood cardioplegia, and (3) development of adjunctive measures to enhance blood cardioplegia.

For editorial comment, see page 1209.

The advantages of blood cardioplegia, as initially promulgated, have endured and need to be only slightly modified. These were (1) rapid cardiac arrest in an oxygenated environment; (2) intermittent reoxygenation with delivery of cardioplegia solution; (3) intermittent reoxygenation, which makes it unnecessary to add anaerobic substrate (glucose and insulin); (4) whole blood, which provides oncotic constituents that otherwise must be added as plasma protein, mannitol, or dextran; and (5) avoidance of the cost of and need for preparing a complex pharmacologic mixture, although the apparatus for delivery is more complex and costly than for crystalloid cardioplegia [1].

Physiologic Basis for Blood Cardioplegia

Oxygen Delivery

At normothermia, hemoglobin carried by red cells provides an effective system for delivery of oxygen to the tissues, but as the temperature is reduced, the oxyhemoglobin dissociation curve shifts to the left [10] so that oxygen is less readily available and requires increasingly reduced tissue oxygen tension before oxygen is released. At 20°C, blood cardioplegia released 50% of its total oxygen content, and at 10°C, only 37% to 38%, which is in contrast to oxygenated crystalloid cardioplegia, which releases all of its oxygen at either temperature [12]. These facts have led to the conclusion that blood cardioplegia does not effectively deliver oxygen to the myocardium during hypothermic cardioplegia beyond that which is physically dissolved in the vehicle and can therefore be equally well delivered by crystalloid cardioplegia that has been fully equilibrated with oxygen. On the other hand, there is little doubt that some myocardial uptake of hemoglobin-bound oxygen occurs during hypothermic blood cardioplegia [12–21]. Favoring the release of hemoglobin-bound oxygen is the mild myocardial acidosis that develops during blood cardioplegia arrest [22–23]. This results in local red cell acidosis with a shift to the right of the oxyhemoglobin dissociation curve (Bohr effect, see below). Additionally, hypothermia results in a greater tissue affinity for oxygen [24]. Others have not found unfavorable oxyhemoglobin dissociation kinetics at temperatures of 30°C [25] and 20°C [26].

The potassium-paralyzed heart maintained at 22°C has a myocardial oxygen consumption (MVO_2) of 0.3 mL · 100 g^{-1} · min^{-1} [27] and when the temperature is reduced to between 10° and 12°C, the MVO_2 is 0.135 mL · 100 g^{-1} · min^{-1} [28]. Experimental studies conducted at a myocardial temperature of 27°C indicated that hemoglobin-

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mediated oxygen delivery to the myocardium was important for cardioplegic myocardial preservation [14–29]. When myocardial temperature was reduced to between 10° and 12°C, hemoglobin transport of oxygen was not critical to myocyte preservation provided that the cardioplegia solution was fully oxygenated [17, 30], and at 4°C, blood was associated with reduced preservation of left ventricular function despite more oxygen delivery with blood than with crystalloid [18]. Either vehicle (blood or crystalloid) will carry more dissolved oxygen as the temperature is lowered to 4°C and the solution fully oxygenated [17, 31].

The studies cited above have many variables, but in aggregate they indicate that hemoglobin-mediated delivery of oxygen during blood cardioplegia becomes less important as myocardial temperature is reduced to between 5° and 20°C, where oxygen requirements are small and dissolved oxygen can meet these needs. With regard to the issue of whether oxygen delivery is necessary for the success of blood cardioplegia, it has been clearly demonstrated in a recent report that this component is necessary for optimal myocardial protection by comparing three levels of oxygen content (1.1, 4.3, and 10.2 vol%) with a cardioplegic temperature of 4°C and a myocardial temperature of 10° to 12°C [21].

Buffering Capacity

Buffering capacity of blood is primarily a function of the histidine component of plasma proteins and hemoglobin. Hemoglobin has approximately six times the buffering capacity of the plasma proteins in blood owing to its high concentration (nearly four times greater than that of plasma proteins) and its 38 histidine residues (more than twice as many as albumin, even though molecular weights of albumin and hemoglobin are similar) [32]. The imidazole group of histidine provides a buffer with temperature characteristics such that the pH is shifted toward alkalinity with hypothermia, a shift that preserves the balance between H^+ and OH^- [33]. Thus, there is an alkalotic shift of myocardial pH of 0.1 to 0.15 units on cooling the heart from 37° to 20°C [22] and of 0.27 units when the heart is cooled to less than 15°C [23]. These animal data correspond closely to the recognized 0.015 to 0.022 unit rise in pH per degree centigrade of hypothermia in blood and tissues [34–36]. This increase in pH with cooling closely follows the change in neutrality of water; the pH of neutrality of water, or 0.5 pK_a, is nearly 7.45 at 0°C and 5.8 at 37°C [37]. Thus, an appropriate rise in blood and tissue pH with cooling does not represent a net change in acid-base balance, but failure of the pH to rise with cooling indicates acidosis.

A further contribution to the effectiveness of hemoglobin as a blood buffer is the fact that deoxygenated hemoglobin has imidazole groups with a somewhat higher pK_a than oxygenated hemoglobin [38]. Thus, once hemoglobin becomes deoxygenated in the capillaries, it is better able to bind the H^+ ions that diffuse into the red cell from the tissues or are formed when CO_2 enters the red cells from the tissues.

Another mechanism for tissue buffering, which also facilitates oxygen delivery, is through the activity of red-cell–carried carbonic anhydrase, which catalyzes the reaction of CO_2 with H_2O to create H_2CO_3, a weak acid that ionizes to H^+ and HCO_3^- lowering red cell pH. The increase in hydrogen ion concentration decreases the oxygen affinity of the hemoglobin (Bohr effect) and facilitates oxygen delivery to the tissues. The increased HCO_3^- ions diffuse out of the red cell and are replaced by chloride ions (chloride shift) [39]. The removal of CO_2 creates a gradient with the tissues and promotes diffusion of CO_2 from the myocytes across the freely permeable sarcolemma. Although the same reaction of CO_2 with water occurs in the plasma and tissues, the rate is much slower, thereby limiting the formation of cellular H^+ and consequent tissue acidosis. Other major sources of hydrogen ion accumulation in the ischemic myocyte are from hydrolysis of adenosine triphosphate [40] and from the accumulation of lactic acid during anaerobic glycolysis [41].

Myocardial pH and pH of the coronary venous effluent are indicative of the degree of tissue acidosis and correlate with other indicators of the adequacy of myocardial preservation during cardioplegia [42].

When washed red cells were added to crystalloid cardioplegia at 27°C, the pH of the coronary effluent stabilized after 30 minutes of ischemia and was 7.20 after 105 minutes versus 6.4 in the control (crystalloid only) group [13]. The effluent carbon dioxide tension declined progressively during cardioplegia to 17.4 ± 3.3 mm Hg, whereas the control value was 37.4 ± 3.3 mm Hg [13]. In a similar study with blood cardioplegia at 20°C, myocardial pH fell during the initial 30 minutes of ischemia to 7.23 and stabilized at 7.19 (180 minutes of ischemia); values for crystalloid cardioplegia were 7.03 and 6.54 [22]. Histidine-buffered crystalloid cardioplegia (Bretschneider's solution) was as effective as blood in preventing a continued decline in myocardial pH [22]. When myocardial temperature was maintained at less than 15°C with blood cardioplegia, myocardial pH fell to 7.32 during the first 60 minutes and to 7.22 during the second hour, with values for crystalloid cardioplegia of 7.16 and 6.76 [23].

Two of these three reports [22, 23] suggest that blood has a favorable effect on myocardial pH during cardioplegia, whereas the third suggests that this can be achieved with washed red cells alone [13] without the buffering provided by plasma proteins.

Capillary Flow Distribution

Zweifach [43] observed the absence of capillary flow in frogs when the circulating medium was crystalloid or colloid. The addition of red cells or carbon particles resulted in complete perfusion of the capillary bed. In the isolated canine heart, the addition of red cells (hematocrit, 0.02) resulted in increased myocardial oxygen consumption, reduced myocardial edema, and improved left ventricular stroke work and prevented a rise in coronary vascular resistance [44]. The authors of the latter study postulated that the improvement in myocardial function was a direct result of the mechanical effect of red blood cells to improve capillary flow distribution in the canine heart. Glomerular filtration improved when nitrite-treated red cells (no oxygen transport function) were added to an oxygenated perfusion system [45]. Again, the favorable effect was attributed to improved intrarenal flow distribution related to red cells acting as microparticles. Perfusion of the canine posterior papillary muscle was better with blood cardioplegia than with crystalloid [16]. Blood cardioplegia has been reported to result in better perfusion distal to coronary stenoses than crystalloid cardioplegia [46].

These observations suggest that red cells, or other microparticles, are critical for capillary perfusion. Although a few red cells may serve this purpose, a high hematocrit (0.30) rather than a low one (0.10) was associated with greater enhancement of, or more uniform distribution of, capillary flow manifest in lower coronary venous oxygen tension and higher AVO_2 [16], apparently owing to facilitation of uniform oxygen delivery from the vascular compartment to the myocytes [47].

Free Radicals

There is increasing evidence that oxygen free radicals contribute to myocardial damage occurring with ischemia and reperfusion [48–53]. A free radical is an atom or molecule containing an odd number (unpaired) of electrons in outer orbit giving it an open or half-bond, making it chemically reactive [54]. If two radicals react, both are eliminated. If a radical reacts with a nonradical, another free radical is created. Thus, chain reactions may be initiated and radicals may serve as oxidants or reductants.

Superoxide (O_2^-) appears to be generated by the enzyme xanthine oxidase, which is synthesized as xanthine dehydrogenase and converted to the former in ischemic tissue [54]. Superoxide dismutase, a ubiquitous mammalian enzyme, can dismutate the superoxide anion to hydrogen peroxide; catalase and glutathione peroxidase catalyze reactions that reduce hydrogen peroxide to water and oxygen. The major danger of superoxide anion and hydrogen peroxide accumulation is the potential for production of hydroxyl radicals ($OH^{\cdot}$) through the Haber-Weiss and Fenton reactions. This radical does not exist under physiologic conditions, it has no natural enemies, and it can cause extensive tissue destruction through lipid peroxidation. Allopurinol, which can block xanthine oxidase [55], and mannitol, which can react with the hydroxyl radical [56], have been of demonstrated value in reducing injury where there was potential for free radical generation.

In addition to the protective mechanisms already mentioned, human plasma has free radical-trapping antioxidant activity provided by urate (35% to 65%), plasma proteins (10% to 15%), ascorbate (0% to 24%), and vitamin E (5% to 10%) [57]. Vitamin E (α-tocopherol) is the only lipid-soluble antioxidant in the plasma and is therefore able to trap peroxyl radicals present in or at the interface of the lipid phase. Vitamin C (water soluble) appears to be the only plasma antioxidant capable of regenerating vitamin E from the α-tocopherol radical [57]. Thus, human plasma is highly resistant to peroxidation.

Experimental studies of free radicals in cardioplegia have centered on global and regional ischemic and crystalloid cardioplegia [49–53, 56–58], with a single report of free radical injury related to blood cardioplegia [59]. In addition to the antioxidants contained in plasma, red cells contain catalase, superoxide dismutase, and glutathione, all of which participate in reactions to scavenge free radicals. Glutathione is synthesized in red cells and is mostly present in the reduced form; it may be important in the detoxifying of hydrogen peroxide through glutathione peroxidase with oxidation of glutathione, which in turn is rapidly reduced by glutathione reductase [60].

Although blood appears attractive from the standpoint of inherent mechanisms for prevention of free radical generation and chain-breaking of their effects, it must be recognized that this whole phenomenon remains largely unexplored, particularly in a quantitative sense [61].

Oncotic Pressure

The plasma proteins normally provide oncotic pressure to counteract the intraluminal physical pressure, which tends to drive fluid across the wall of the capillary. It was originally proposed that blood cardioplegia would provide sufficient plasma protein to prevent or minimize myocardial edema. The bypass circuit is commonly primed with 2,000 mL of crystalloid (occasionally, 500 mL of 5% albumin is included in that 2,000 mL), which dilutes a 5,000-mL blood volume by almost 30% and therefore plasma proteins to the same degree. Blood cardioplegia is commonly given as a 4:1 dilution (4 parts blood and 1 part crystalloid), which dilutes plasma proteins by another 20% (total of 50%). This would reduce albumin to a fairly critical level of 22 g/L, so that the balance between filtration and absorption across the capillary membrane would be shifted toward filtration. On the other hand, the time of exposure to hydrostatic pressure is relatively brief and limited to those periods of cardioplegic infusion, which would aggregate 12 to 14 minutes (including terminal warm infusion) for 1 hour of cross-clamp time or 18 to 20 minutes for 2 hours. Thus, the time for edema formation, and therefore its severity, are limited. Myocardial edema has not been a significant occurrence in blood cardioplegia [1, 16, 62] or, for that matter, in oxygenated crystalloid cardioplegia [16].

Hemodilution

Systemic hemodilution with secondary systemic fluid gain has been an occasional clinical problem after infusion of large volumes (3,000 to 5,000 mL) of crystalloid cardioplegia. Isolation of the right side of the heart will permit recovery of most of the antegradely infused cardioplegia, but this is not commonly done. With a trend toward warm induction and terminal warm infusion, larger volumes of cardioplegia are being used. A 4:1 dilution of blood cardioplegia results in the addition of 600 mL of crystalline for 3,000 mL of cardioplegia or 1,000 mL for 5,000 mL, which represents substantially less crystalloid load than associated with crystalloid cardioplegia. Continuous cardioplegia is associated with even greater volumes.

Experimental Comparison of Blood and Crystalloid Cardioplegia

Hypothermic blood cardioplegia has been studied extensively using the in situ heart of the dog and pig (Table 1). To reach a consensus with regard to these data is difficult because of the many variables involved, most of which are not listed in the table, and furthermore, vary from report to report. There may be some value in commenting on a few of the strengths and weaknesses of these reports beyond a simple accounting, as well as providing a sense of the evolution of blood cardioplegia.

The second listed report (Table 1) suffered in that the infusion intervals were 30 and 15 minutes for blood and crystalloid cardioplegia, respectively, and that topical hypothermia was applied only in the blood group so that myocardial temperature was lower (7°C versus 15°C) [2]. Engleman and associates [14] were far ahead of their time in fully oxygenating crystalloid cardioplegia, which is now recognized as an important component of crystalloid cardioplegia [16, 30, 31, 75]. Takamoto and co-workers [65] observed that suboptimal blood cardioplegia (single dose versus multiple dose) resulted in inferior myocardial protection, although their interval of infusion was 30 minutes as opposed to the generally used 20 minutes (except for the recent reports by Catinella and Feindel and their associates [68–70]). Kanter and colleagues [17] compared blood, crystalloid, and Fluosol cardioplegia and observed strikingly greater MVO_2 during ischemia with each Fluosol infusion (203.8 mL O_2/100 g dry weight versus 20.4 for crystalloid and 39.2 for blood), as well as better recovery of systolic function. Similar observations were made by Novick and co-workers [19] in the hypertrophied heart, although the MVO_2 with Fluosol (5.8 vol%) was only slightly greater than for blood (5.0 vol). Despite this apparent advantage of Fluosol, there was only 70% to 80% recovery of left ventricular systolic function [17, 19].

To dissect the use of blood cardioplegia, Bing and colleagues [13] added red cells to crystalloid cardioplegia (hematocrit, 0.20) and concluded that the improved myocardial recovery was the result of buffering action of the red cells and improved oxygen delivery. In a subsequent nearly identical study, red cells in one group were treated with sodium nitrite, resulting in methemoglobin formation (red cells able to buffer pH, but unable to transport oxygen), which prevented increases in coronary sinus effluent carbon dioxide tension and the decrease in pH noted with crystalloid cardioplegia [29]. However, the methemoglobin group was inferior to the intact red cell group with regard to ischemic phosphocreatine depletion and systolic functional recovery and inferior to the red cell and crystalloid groups with regard to lactate production during ischemia and recovery MVO_2. Thus, Bing and associates concluded that myocardial protection provided by red cells was not the result of buffering but was related to oxygen delivery. Although this work represents an important effort to determine the mechanism(s) of myocardial protection by red cells, it is limited by the myocardial temperature of 27°C, which is warmer than clinical cardioplegia (10° to 20°C), so that oxygen delivery becomes relatively more important because of a higher metabolic rate [73]. Furthermore, use of the isolated heart model does not allow for noncoronary collateral flow, which may be an important source of oxygen and substrate, particularly when the heart is warmer [76]. The issue of myocardial temperature is emphasized in the work of Magovern and associates [19] who found that blood cardioplegia was most effective at 20°C, of no additional benefit (over crystalloid) at 10°C, and inferior to crystalloid cardioplegia at 4°C. Similarly, Rousou and co-workers [73] observed that at 10°C blood and oxygenated crystalloid cardioplegia were equally effective and that whether the hematocrit was 0.10 to 0.20 made no difference.

Pursuing a similar approach, Heitmiller and colleagues [16] added red cells to crystalloid cardioplegia (hematocrit of 0.10 and 0.30) and compared this with crystalloid, all of which were fully oxygenated and delivered at 4°C (myocardial temperature, 10°C) every 20 minutes for 5 hours. Ischemic MVO_2 was significantly greater in the red cell groups. Functional recovery was impaired in the low-hematocrit (0.10) group, myocardial adenosine triphosphate content was maintained in all groups, and ultrastructure was well preserved in all groups. Interestingly, cardioplegic perfusion of the posterior papillary muscle (microspheres) was best maintained in the high-hematocrit (0.30) group. Heitmiller and colleagues concluded that there was no beneficial effect of added red cells beyond that of enhanced ischemic MVO_2, enhanced posterior papillary muscle perfusion, and the favorable effect of associated calcium on left ventricular function.

Illes and associates [30] have made a further contribution in this regard by studying cardioplegia with a 50% plasma-crystalloid solution and two similar solutions containing red cells to give a hematocrit of 0.17 or 0.082 during 2 hours of arrest at 10°C. Systolic function and MVO_2 versus stroke work relationship were unchanged after arrest, but basal postarrest MVO_2 was increased in the plasma group (indicating inferior myocardial protection). Because the plasma concentration was the same in all three groups and oxygen availability the same in the low-hematocrit (0.082) and plasma groups, the efficacy of blood cardioplegia cannot be attributed to greater oxygen delivery by the red cell, but must lie in other properties of the red cell.

Comparison of blood and crystalloid cardioplegia in the setting of acute coronary occlusion (60 minutes at 37°C) followed by 1 hour of global cardioplegia (but with warm induction in the blood group) revealed better recovery of systolic function in the ischemic segment with blood cardioplegia [72].

Clinical Comparison of Blood and Crystalloid Cardioplegia

Several early reports suggested that blood cardioplegia was clinically useful, but these were nonrandomized and involved comparison with prior or concurrent experience using alternative techniques of myocardial protection [1,

Table 1. Experimental Comparison of Blood, Crystalloid, Oxygenated Crystalloid, Oxygenated Plasma, and Fluosol Cardioplegia

First Author	Year	Isch Time (h)	Interval (min)	Myo Temp (°C)	Function		Ischemic		Recovery		Lactate		MVO_2		Myo pH	Myo H_2O	Ultra-structure
					Sys	Dias	ATP	CP	ATP	CP	Isch	Rec	Isch	Rec			
Follette [63]	1978	2.0	20	20	B > C									B > C		B > C	
Barner [64]	1979	2.0	30, 15	7, 15	B = C	B = C	B > C	B = C	B > C			B = C		B = C		B > C	B = C
Engelman [14]	1980	3.0	15	8–15			B > C	B > C	B = C	B > C			B > C				
		3.0	15	8–15			B > OC	B > OC	B = OC	B = OC			B > OC				
Takamoto [65]	1980	2.0	30	22	B = C		B = C	B > C	B = C	B = C		B = C		B = C			B = C
Kanter [17]	1981	1.5	20	20	B = C								B > C			B > C	
					F > B								F > B			F > B	
Bing[a] [13]	1982	2.0	15	27	RC > C		RC = C	RC > C	RC = C	RC = C				RC > C	RC > C		
Magovern [18]	1982	1.5	30	20	B > C								B > C			B > C	B = C
				10	B = C								B = C			B = C	B = C
Swanson[b] [66]	1982			4	B = C								B = C			B < C	B < C
		2.0	30	12			B = C		B = C								
		3.0	30	20							B = C				B > C		
Nwaneri [67]	1983	2.0	20	22, 4	B = C		B = C	B > C	B = C	B = C							
Bing [29]	1984	2.0	15	27	RC > C		RC > C	RC > C	RC = C	RC = C	RC = C		RC > C	RC = C	RC > C		
Catinella [68]	1984	3.0	30	10	B = C	B = C	B > C		B > C					B = C			B > C
Catinella [69]	1984	3.0	30	13			B > C		B > C								
Feindel [70]	1984	4.5	30	27													B > C
		6.0	30	27													B > C
Novick[c] [19]	1985	3.0	20	10–15	B > C	B > C	B > C	B > C	B = C	B = C			B > C			B = C	B = C
		3.0	20	10–15	F > B	F = B	F = B	F = B	F = B	F = B			F = B			F = B	F > B
Heitmiller[d] [16]	1985	5.0	20	10	B10 = OC		B10 = OC		B10 = OC				B10 > OC			B10 = OC	B10 = OC
					B30 = OC		B30 = OC		B30 = OC				B30 > OC			B30 = OC	B30 = OC
Goldstein[e] [71]	1986	2.0	0	11	B = C		B = C		B = C								
Vinten-Johansen[f] [72]	1986	1.0	20	10	B > C		B = C		B = C								
Warner [23]	1987[23]	2.0	. . .[g]	14											B > C		
Rousou [73]	1988	2.0	15	10			B = OC	B = OC	B = OC	B = OC			B = OC	B = OC			
Illes[h] [30]	1989	2.0	20	11	B17 = OP	B17 = OP								B17 = OP			
					B8 = OP	B8 < OP								B8 = OP			
Coetzee [74]	1990	1.5	20	4	B < OC		B = OC		B = OC								

[a] Red cells added to crystalloid cardioplegia to give a hematocrit of 0.20. [b] Hypertrophic and normal hearts. [c] Hypertrophic hearts. [d] Red cells added to crystalloid cardioplegia to give a hematocrit of 0.10 (B10) and 0.30 (B30). [e] Coronary sinus cardioplegia infused continuously. [f] One hour of coronary ligation followed byone hour of cardioplegia with study of the ischemic segment. [g] Cardioplegia infused as needed to maintain myocardial temperature < 15°C. [h] Blood cardioplegia with hematocrit of 0.17 (B17) or 0.08 (B8) compared with 50% oxygenated plasma.

ATP = adenosine triphosphate; B = blood; C = crystalloid; CP = creatine phosphate; Dias = diastolic; F = Fluosol; Interval = interval between cardioplegic infusions; Isch = ischemia; Myo = myocardial; OC = oxygenated crystalloid; OP = oxygenated plasma; Rec = recovery; Sys = systolic; Temp = temperature; > significant difference favoring blood; = no significant difference; < significant difference favoring crystalloid.

2, 77]. The first of 12 randomized clinical trials of blood versus crystalloid cardioplegia appeared in 1980 (Table 2). The most comprehensive reports are from the University of Toronto [15, 86, 87]. Additionally, as in the experimental studies, there are multiple variables of composition of the solution, temperature, and parameters of infusion and many others that are not controlled.

The initial study evaluated intraoperative left ventricular function (cardiac output, left ventricular end-diastolic pressure, and maximum observed contractile element velocity) before and after bypass and found no difference after cardioplegia or between the groups [78].

Engleman and co-workers [79] found greater MVO_2 during arrest with blood and greater myocardial release of lactate with crystalloid cardioplegia. During reperfusion, there were no differences in MVO_2 and lactate uptake (or release) between the two groups. Release of the MB isoenzyme of creatine kinase (CK-MB) was the same, and no perioperative infarcts were noted.

Roberts and associates [80] studied 50 patients and found no difference between blood and crystalloid cardioplegia with regard to cardiac index (to 24 hours), left ventricular stroke work index, radionuclide ejection fraction (to 7 days), technetium-99m–pyrophosphate scans, or CK-MB release. Roberts and associates separately analyzed 16 of 50 patients with an ejection fraction less than 0.40 and found a significant improvement in ejection fraction at 1 and 7 days in the blood group. Additionally, they analyzed 25 of 50 patients with an ischemia time of more than 90 minutes and observed that the ejection fraction was better in the blood group at 7 days only.

The report by Singh and colleagues [81] focused on myocardial ultrastructure with the finding of mitochondrial swelling by morphometric analysis, intracellular edema, depletion of glycogen stores, and focal myofibrillary disorganization, as well as greater release of CK-MB, in patients receiving crystalloid cardioplegia. Similarly Chen and Lin reported better right atrial mitochondrial preservation with blood cardioplegia [84].

Buttner and colleagues [82] found that blood and crystalloid were similar with regard to ventricular function, lactate metabolism after reperfusion, and CK-MB release. They observed reduced ischemic MVO_2 with blood cardioplegia, which may have been related to the low temperature (4°C) of the infusate. They also reinfused cardioplegia every 30 to 40 minutes, which would be one reinfusion with a mean ischemia time of 47 minutes.

Iverson and colleagues [83] found that left ventricular stroke work index was depressed to 36 hours postoperatively in the crystalloid group and that CK-MB release was increased in this group. Codd and associates [85] reported that blood cardioplegia was associated with a reduced need for postoperative inotropic support and reduced CK-MB release.

Fremes and colleagues [15] have presented the most elegant clinical studies of cardioplegia. In comparing blood and crystalloid solutions in 90 patients having elective coronary grafting, they found a reduced rate of myocardial infarction ($p = 0.06$) and reduced maximum CK-MB release ($p < 0.02$) with blood cardioplegia. Sixty patients had more sensitive measurements of myocardial metabolism and function. With blood cardioplegia, coronary sinus blood flow was less with reperfusion, indicating less reactive hyperemia; myocardial lactate production was less during ischemia and early reperfusion; myocardial performance as determined by the left ventricular stroke work index–left ventricular end-diastolic volume index relation and systolic elastance (the systolic blood pressure–left ventricular end-systolic volume index relation) were better; and diastolic compliance (the left atrial pressure–left ventricular end-diastolic volume index relation) was similar to that for crystalloid cardioplegia. These 60 patients were carefully selected and at low risk for operation by the usual criteria. Despite this favorable clinical setting, blood cardioplegia was superior by sensitive endpoints, but little clinical improvement could be discerned. Ideally, patients at greater operative risk need to be studied by these same detailed methods, but the existence of compelling clinical situations frequently precludes investigative efforts such as these.

In a second study, the same group focused on ventricular function preoperatively, 3 to 5 hours postoperatively, and 5 months postoperatively with and without exercise [86]. They found that blood decreased ischemic injury (less elevation in CK-MB and aspartate aminotransferase levels), preserved ventricular performance (lower stroke work index at higher left ventricular end-diastolic volume index after crystalloid), and preserved systolic function (higher left ventricular end-systolic volume index at similar systolic blood pressure after crystalloid cardioplegia). Late postoperative function was not different between the two groups (blood and crystalloid) at rest or with exercise, but exercise function was better than preoperatively. This study is consistent with the results of their initial study in indicating that blood cardioplegia provides better perioperative myocardial preservation than does crystalloid cardioplegia, but this does not translate into better long-term ventricular function in patients having elective coronary bypass with an ejection fraction greater than 0.40.

A third study from Toronto [87] addressed right ventricular function in the perioperative interval and found better preservation of systolic function with crystalloid cardioplegia but equal preservation of right ventricular performance and diastolic function. Blood cardioplegia resulted in better left ventricular systolic and diastolic function as well as performance. Cardiac metabolic response to volume loading was similar in the two groups postoperatively with regard to coronary sinus flow, MVO_2, and lactate flux, which was also noted in the initial study [16]. The right ventricle was warmer than the left with both blood and crystalloid, and lower temperatures for both chambers ($p < 0.0001$) were achieved with crystalloid.

Adjuncts to Blood Cardioplegia

Beta Blockers

Ample experimental evidence indicates that β-adrenergic blockade reduces myocardial injury in models of hypoxia and ischemia [89–91]. When β-blockers were added to

Table 2. Randomized Clinical Studies of Blood and Crystalloid Cardioplegia

First Author	Year	No. of Patients	Isch Time (min)	Infusate Temp (°C)		Function		MVO_2		Lactate		ATP		Cor Flow	CK-MB	MI[a]	Ultra-structure
				B	C	Sys	Dias	Isch	Rec	Isch	Rec	Isch	Rec				
Shapira [78]	1980	24	53	10	4	B = C	B = C										
Engelman [79]	1981	27	65	9	9			B > C	B = C	B > C	B = C				B = C	B = C	
Roberts [80]	1982	50	70	8	8	B = C	B = C								B = C	B = C	
		16[b]				B > C											
		25[c]	>90			B > C											
Singh [81]	1982	40	68	20	16.5										B > C	B = C	B > C
Buttner[d] [82]	1984	60	47	4	4	B = C		B < C		B = C					B = C		
Fremes [15]	1984	90	63	11	5	B > C	B = C	B > C	B = C	B > C	B > C	B = C	B = C	B > C	B > C	B = C	
Iverson [83]	1984	207	58	9	5	B > C									B > C		
Chen[e] [84]	1985	20	73	15	4												
Codd [85]	1985	181	72	10	4										B > C	B = C	B = C
Mullen [86]	1986	47	64	11	5	B > C									B > C		
Mullen[f] [87]	1987	80	65	11	5	B < C	B = C		B = C		B = C			B = C	B > C		
Khuri[g] [88]	1988	28	100	9	4										B = C	B = C	

[a] New Q wave. [b] 16 patients with ejection fraction < 0.40 extracted from the total experience of 50 patients. [c] 25 patients with ischemia times > 90 minutes extracted from the total experience of 50 patients. [d] Reinfusion every 30 to 40 minutes. [e] Compared right atrial mitochondrial size, which increased significantly with crystalloid cardioplegia. [f] Compared right ventricular function and the effect of volume loading on cardiac metabolism. [g] Nonrandomized study of matched patients having aortic valve replacement with or without coronary bypass grafting with continuous monitoring of myocardial pH.

ATP = adenosine triphosphate; B = blood; C = crystalloid; CK-MB = MB isoenzyme of creatine kinase; Cor = coronary; Dias = diastolic; isch = ischemia; MI = myocardial infarction; MVO_2 = myocardial oxygen consumption; Rec = recovery; Sys = systolic; Temp = temperature; > significant difference favoring blood; = no significant difference; < significant difference favoring crystalloid.

experimental crystalloid cardioplegia, there was reduced metabolic activity during ischemia and improved ventricular compliance during reperfusion without depression of systolic function [92]. These effects could be obtained with a single dose of propranolol in the first bolus of cardioplegia. Clinically, 0.05 mg/kg of propranolol given systemically before aortic cross-clamping and blood cardioplegia was associated with a reduction in CK-MB release [93].

Calcium Entry Blockers

Extensive laboratory evaluation of calcium entry blockers has revealed that they alone may be as effective as potassium cardioplegia whether delivered in a vehicle based on crystalloid [94–97] or blood [98, 99]. Several investigators have found that a combination of hyperkalemia and calcium entry blockade may provide better myocardial protection than either alone when used with crystalloid [100–102] or blood cardioplegia [103]. These latter studies resulted in two randomized clinical trials in which diltiazem was added to the cardioplegic infusion. Christakis and associates [104] used crystalloid cardioplegia with 150 μg/kg diltiazem divided equally between the first and last bolus of cardioplegia. They found that diltiazem was associated with preservation of high-energy phosphates, improved postoperative myocardial metabolism, and reduced ischemic injury after elective coronary bypass. However, diltiazem was a potent negative inotrope and produced prolonged periods of electromechanical arrest with the requirement for atrioventricular sequential pacing to discontinue cardiopulmonary bypass in all patients. When diltiazem was added to blood potassium cardioplegia, there was increasing time to atrioventricular function as the dose of diltiazem was increased [105]. Diltiazem at a dose of 100 μg/kg (for each of three infusions) was associated with a reduction in peak CK-MB release. On the basis of these two reports, it is concluded that the risks of atrioventricular blockade and depressed ventricular function are of sufficient concern to outweigh the benefits achieved.

Verapamil (0.1 mg/kg up to 10 mg) given systemically before aortic cross-clamping and cold blood potassium cardioplegia was associated with lower CK-MB release compared with controls [106].

Local Anesthetics

Procaine has been added to crystalloid cardioplegia for membrane stabilization [107] or used as an isolated cardioplegia agent [108] but has not been used in blood cardioplegia. Hypersensitivity or allergy may occur with ester-linked agents such as procaine, whereas amide-linked local anesthetics such as lidocaine are singularly free of this complication [109]. Adding lidocaine (0.1 mg/mL) to blood cardioplegia was associated with spontaneous return of sinus rhythm in 74% of patients versus 22% of controls [110]. Left ventricular function was minimally depressed 30 minutes after reperfusion, but this effect was transient and of no clinical importance. Use of lidocaine in this way has been routine in our practice for 2 years.

Warm Induction Blood Cardioplegia

It was initially proposed that warm induction of blood cardioplegia (5 minutes of blood cardioplegia at 37°C) before multidose infusion of cold blood cardioplegia would increase the tolerance of energy-depleted hearts to subsequent aortic cross-clamping [20]. Warm induction was associated with greater MVO_2 during the initial 5 minutes of cardioplegia (cold, 8.1 mL/100 g; warm, 16.9 mL/100 g) and throughout the cold cardioplegic infusions (2 hours) (cold, 34 mL/100 g; warm, 40 mL/100 g). Creatine phosphate level rebounded above control with warm induction and remained near control in both groups, but the level of adenosine triphosphate increased only slightly and remained depressed in both groups. With cold induction glucose-6-phosphate content remained elevated, whereas glucose-6-phosphate levels fell with warm induction, suggesting better aerobic metabolism. These data support the concept of warm induction for hearts that are metabolically impaired through prior acute or ongoing ischemia or because of hypertrophy. Additionally, they suggest that, within limits, the absolute level of adenosine triphosphate is not critical with regard to left ventricular function, as good left ventricular function was observed with an adenosine triphosphate level of less than 50% of control.

Terminal Warm Blood Cardioplegia

The infusion of warm blood cardioplegia as a terminal "hot shot" to aid in myocardial recovery after an interval of cardioplegia was proposed in 1977 [111]. Initially, this was the same mix of blood cardioplegia as used for reinfusions, except for returning the temperature to 37°C. Later the blood cardioplegia was "enhanced" with glutamate [112] and subsequently glutamate and aspartate [113].

The only clinical study of terminal warm blood cardioplegia randomized 20 patients to receive cold blood cardioplegia with or without a "hot shot" [114]. With the "hot shot," myocardial metabolic recovery was improved (myocardial lactate consumption versus release after cross-clamp removal), high-energy phosphates were better preserved, the metabolic response to stress was normal, and diastolic function was preserved. This report has generated considerable interest among clinicians, and terminal warm cardioplegia with or without glutamate and aspartate is being used in many units.

Substrate Enhancement

Recovery of the myocardium from acute ischemic injury is aided with the Krebs cycle intermediates glutamate and aspartate [113]. These two amino acids can replenish Krebs cycle intermediates [115] and contribute to the malate-aspartate shuttle [116], which delivers reducing equivalents formed by the reduction of cytosolic nicotinamide adenine dinucleotide into the mitochondrion. Based on these experimental models, preoperative ischemic left ventricular failure has been treated with glutamate or aspartate, or both, using warm blood induction, cold blood cardioplegia, and terminal warm infusion [117–119]. This approach has enhanced the recovery of ventricular function and reduced the need for postoperative inotropic and mechanical ventricular support.

Subtle and transient impairment of left ventricular function and delayed myocardial metabolic recovery have been observed after elective coronary bypass grafting with apparently well-conducted cardioplegia [15, 114]. To determine whether substrate enhancement could ameliorate these limitations of clinical cardioplegia, patients having elective coronary bypass were randomized to receive warm blood induction and terminal warm blood cardioplegia supplemented with glutamate and aspartate in both infusions and cold blood cardioplegia without supplementation during the procedure whereas controls received standard cold blood cardioplegia without supplementation [120]. Myocardial lactate consumption was greater during reperfusion with supplementation and increased with pacing in the supplemented group but decreased in controls. Supplementation did not prevent the fall in adenosine triphosphate concentration, did not improve postoperative ventricular function, and had no influence on postoperative morbidity. Fifty-two patients having urgent coronary bypass for prolonged angina resistant to medical therapy were also randomized as above. Those receiving supplementation had a reduction in CK-MB released postoperatively (Teoh KH, Ivanov J, Weisel RD, Mickle DAG; unpublished results). The authors of this study concluded that the beneficial effects of glutamate and aspartate infused in this manner were too subtle to justify routine use, but in patients with energy-depleted hearts, the risks of urgent revascularization may be reduced. In patients having urgent revascularization (resistant angina, postinfarction angina, or catheterization complications) and randomized as above there was a reduction in CK-MB release in the supplemented group, and postoperative low output was reduced in those with left main stenosis or impaired left ventricular function (ejection fraction less than 0.40) [121].

Infusion of sodium lactate perioperatively in patients having myocardial revascularization with blood cardioplegia was associated with enhanced lactate uptake and oxidation during reperfusion [122]. Atrial pacing resulted in increased lactate uptake and oxidation, whereas the normal lactate group had lactate production. Systolic function (the relation between end-systolic pressure and volume) 3 hours postoperatively was better in the lactate group, and CK-MB release was less. Impaired metabolism of glucose and fatty acids after ischemia and cardioplegia [123, 124] logically led to this study of lactate as a preferred substrate.

Alternative Delivery of Blood Cardioplegia

Retrograde Blood Cardioplegia

Although retrograde coronary sinus perfusion for aortic valve operations was practiced as early as 1957 [125, 126], the technique of retrograde cardioplegia has become popular in the past decade [127] after laboratory demonstration of its usefulness [128–132].

Substantial clinical experience with retrograde coronary sinus cardioplegia has demonstrated its usefulness, particularly in operations on the aortic valve and root [133], in operations on the mitral valve, for coronary reoperations, and for acute ischemic syndromes (acute angioplasty failure). There are technical advantages of retrograde coronary sinus cardioplegia for valve operations in that conduct of the operation is usually not interrupted by the need for the surgeon to halt work on the valve and infuse antegrade cardioplegia. In coronary reoperations retrograde cardioplegia may limit embolization from diseased vein grafts and can deliver cardioplegia to myocardium supplied by patent internal thoracic artery grafts (which should be clamped). However, randomized clinical studies of antegrade versus retrograde coronary sinus blood cardioplegia have demonstrated no advantage of one technique over the other [134–136].

Retrograde atrial cardioplegia has also been used with good clinical results [137], but a randomized clinical study of antegrade and retrograde atrial blood cardioplegia has shown equivalent results with the two techniques [138]. Although there are concerns about preservation of right ventricular function with this technique [139], it can be argued that atrial cardioplegia perfuses veins draining directly into the right atrium, as well as veins draining into the proximal coronary sinus, which may be excluded from perfusion by virtue of the position of the balloon catheter in the coronary sinus.

Retrograde atrial cardioplegia tends to be more cumbersome, with the need for bicaval cannulation, caval snaring, pulmonary artery clamping, and distention of the right side of the heart that occurs with each infusion. Current techniques for retrograde coronary sinus cardioplegia include a two-stage venous cannula and closed transatrial coronary sinus cannulation with a balloon-tipped catheter having a lumen for distal pressure measurement.

Antegrade-Retrograde Blood Cardioplegia

It has been proposed that cardioplegia might be more effectively delivered in a combined fashion [140]. In support of this approach is the known heterogeneous delivery of cardioplegia by either route. Homogeneous delivery of antegrade cardioplegia is limited by chronic coronary stenoses or occlusions [140–145]. Retrograde cardioplegia has also been demonstrated to underperfuse the right ventricle and the interventricular septum in studies using india ink [146], low-viscosity resin [147], and radioactive microspheres [148]. Sequential use of both routes of administration should facilitate more homogeneous distribution of cardioplegia, although there are obvious situations (right coronary artery occlusion, left anterior descending coronary artery occlusion) where antegrade delivery would not greatly compensate (unless collaterals were highly developed) for the known deficiencies (right ventricle and posteroseptal portion of the left ventricle) of retrograde delivery, and other strategies of delivery may be required for optimal myocardial protection. When the combined technique is employed, it is appropriate to begin with the antegrade route to hasten the onset of diastolic arrest [135].

Continuous Blood Cardioplegia

Continuous infusion of cold blood cardioplegia has been used to manage patients with cardiac hypertrophy secondary to aortic valve disease [149] or patients at high risk for operation (postinfarction mitral insufficiency with cardiogenic shock) [150]. In a clinical study of myocardial pH, intermittent and continuous blood cardioplegia were compared in patients having valve replacement. Myocardial pH fell during cross-clamping (0.15 pH units) in the intermittent group and rose 0.17 units with continuous cardioplegia [88]. With reperfusion, pH did not decline in the intermittent group, but in the continuous group, pH fell during the initial 9.5 ± 2.1 minutes of reperfusion (0.31 units). There was no difference in the need for inotropic or mechanical support in the two groups or in release of CK-MB, and the postoperative courses were similar. Although continuous infusion was associated with maintenance of myocardial pH during cross-clamping, the fall in pH with reperfusion was similar to the low reperfusion pH of intermittent infusion and required 9.5 minutes to reach a nadir versus 3.5 minutes.

Summary and Conclusions

This brief review has attempted to trace the evolution of blood cardioplegia and to compare it with crystalloid cardioplegia. Adequate delivery of oxygen to the myocyte at the temperatures employed for clinical cardioplegia (5° to 20°C) can be accomplished by physically dissolved oxygen in fully oxygenated blood or crystalloid cardioplegia without the need for hemoglobin transport of oxygen. Red cells, through their contained hemoglobin and carbonic anhydrase, provide a highly effective buffer system that can be partially attained by the addition of the amino acid histidine to crystalloid cardioplegia. Although small numbers of red cells (hematocrit of 0.02) [44] will greatly enhance capillary flow, some data suggest that more complete and uniform flow distribution is achieved with a higher hematocrit. The importance of free radical–mediated injury and scavenging of free radicals in clinical cardioplegia has not been defined, but there are important mechanisms present in red cells and plasma for preventing free radical generation and for neutralization of free radicals. Plasma provides oncotic pressure for blood cardioplegia, which is probably not important as myocardial edema has not been a significant occurrence with crystalloid cardioplegia. The systemic hemodilution associated with large volumes of crystalloid cardioplegia, when not recovered, has occasionally resulted in problematic systemic hemodilution and edema. The routine use of terminal warm cardioplegia is an adjunctive method of apparent value; the advantages of warm induction and substrate enhancement have not been clearly established, but they may have a role in acute or subacute ischemia or for the chronically energy-depleted myocardium. Retrograde delivery of cardioplegia provides some mechanistic advantages but has inadequacies of distribution, so that optimum delivery may be provided by a combination of both routes (antegrade/retrograde). Continuous cold blood cardioplegia has had limited application, whereas continuous warm blood cardioplegia is an even newer technique, and both may complicate the conduct of the operation.

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CORRESPONDENCE

Are We Losing Out in the Care of Non–Small Cell Lung Cancer Patients?

To the Editor:

Lung cancer is difficult to cure because it is usually already advanced when discovered. Non–small cell lung cancer discovered early responds to resective operation, but more advanced disease usually requires multimodality therapy. Radiotherapy and operation are the two modalities that are by themselves curative. Each has its strong points. Operation is best in early disease confined to the lung. Radiotherapy, on the other hand, is best for microscopic and macroscopic spread. The area of the spread, if not sterilized, is shrunken so that it can be more easily removed. Thus, radiotherapy and operation *in that order* complement each other. Trials, 30 years ago, of preoperative irradiation failed to show a survival advantage over operation alone, and its theoretical advantage has not been seriously considered in more modern times.

One would think that communication and collaboration between the purveyors of the two curative modalities would occur on a frequent basis, and that radiotherapists and surgeons would be best of friends. This is far from the case. Why can't the radiotherapist let surgeons know about modern treatment—treatment optimally consisting of continuous-course small fractions to therapeutic dose levels using molded fields and computerized dosimetry? Why can't they explain how much better this is than 30 years ago? Why can't the surgeons acknowledge their gains in preoperative and postoperative care and intraoperative techniques (use of staplers, for example)? Why can't we learn to operate after radiotherapy with a reasonable morbidity and mortality? Why can't the radiotherapist chide the surgeon about sending him or her patients to treat with gross disease left behind? Is this the surgeon who denies that debulking is good therapy but who is applying glorified "dubulking" in most advanced lung cases, as up to one half of recurrences are local ones? Is this the same surgeon who refers all patients for postoperative radiotherapy where he or she has *left* gross disease behind or believes he or she *might have left any* local spread behind? Is this the surgeon who, when reporting results, fails to acknowledge the place postoperative radiotherapy might have played? Why don't the radiotherapists more often ask the surgeon at least occasionally to refer a virgin (surgically unmolested) case for therapy? Perhaps they are too polite. Or perhaps they realize that they will one day treat the patient regardless of *when* the surgeon operates.

In superior sulcus tumors, why do surgeons shackle the radiotherapist to split-course therapy with operation sandwiched between courses? At least one radiotherapist [1] recommends initial thoracotomy in *all* possible resectable cases of superior sulcus tumor and *then* referral for continuous course postoperative radiotherapy. Why can't the surgeons and radiologists start to speak to each other and work together as a team to re-run the 20- to 30-year-old preoperative radiotherapy trials? During the seemingly interminable time it takes to organize and carry out such trials, should the many locally advanced patients not in the trials be given the benefit of the combined team approach? Perhaps the resident who has never operated on a previously irradiated patient can get to know the satisfaction of resecting a previously unresectable patient who now has a better chance for a cure. Isn't it time for the surgeon to update his or her thinking and learn to operate after modern therapeutic dose radiotherapy? The chemotherapists are urging such a preoperative chemotherapy approach. Some authors, namely Kirschner [2], Neptune [3], Stern [4], and Patterson and associates [5], have written to a certain extent on this topic. Why haven't they been more often heeded and copied? Is it because surgeons are happy just to operate first even if the chance for cure is minimal?

I know a pulmonologist who claims to be "surgically minded" and who refers a patient to operation even if he or she only has a "1 in 1000" chance because that is the patient's "only chance." Wouldn't he be better off referring to an enlightened radiotherapist who, after therapeutic dose radiotherapy, in turn might refer that patient to an enlightened, modern surgeon who then might, sure enough, resect with a *better* chance for a cure? Today, a patient is *diagnosed* medically without being seen by the surgeon, is *staged* without the surgeon, and is *referred* without a surgical consult to *radiotherapy* for "some palliation." Is this happening because our medical colleagues have given up on us? Given up on our "operate now at all costs" approach? Isn't there a better chance of our being involved in the decision making, direct patient contact and rapport, and guided optimistic approach when we work as a team with our buddies—the radiotherapists? Perhaps the team approach might help educate our pessimistic colleagues and allow some of the locally advanced non–small cell lung cancer patients a better chance for cure. Incidentally, some radiotherapists include in their radiotherapy for cure category disease confined to the hemithorax including ipsilateral supraclavicular or contralateral hilar nodes! Imagine the surgeon operating on such a patient for curative resection of residual disease after a good response to radiotherapy! It might happen.

I think surgeons would welcome the chance to regain a place in the care of cancer patients and help save some of the large group of locally advanced patients who are now falling between the cracks. A small increase in cure of such salvage cases would make a real dent in the present miserable outcome in advanced non–small cell lung cancer. Isn't it strange how rare present-day trials are that combine in logical sequence the two modalities that, by themselves, are curative? They have been replaced by trials of modalities that, by themselves, are *non*curative—chemotherapy and immunotherapy. Someday after better drugs are found, they may have earned a place in non–small cell lung cancer therapy, but not yet.

Are we up to this challenge? I believe we are, but it takes a rearrangement of our motivation and thinking to collaborate with the radiotherapists—our great and good buddies.

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Tetralogy of Fallot

To the Editor:

Snir and his colleagues [1] are to be congratulated on their truly spectacular results for the so-called absent pulmonary valve syndrome in tetralogy of Fallot. I am intrigued, however, by their

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observation concerning "the ventricular septal defect that is usually located in the outlet septum." This is the more intriguing because one of the authors (M.J.E.) had collaborated with us in another study recently published in *The Annals* that, as far as I was aware, had clarified the limited nature of the outlet septum in this lesion and emphasized its malalignment with the remainder of the muscular septum [2]. Have the Great Ormond Street group uncovered still newer insights into the anatomy of the outflow tracts in tetralogy?

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Reply

To the Editor:

We have read Dr Anderson's query about the ventricular septal defect in patients with tetralogy and absent pulmonary valve syndrome. What we wished to emphasize was the fact that in the majority of cases this was a "defect with exclusively muscular boundaries opening in to the outlet component of the right ventricle." This quote is from Dr Anderson's report on the surgical anatomy of ventricular septal defect. We do hope that this will dispel Dr Anderson's worries.

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Spontaneous Esophageal Rupture

To the Editor:

I read with interest the recent article by McNamee and associates [1], which describes a technique in which a Salem sump is introduced into the periesophageal tissues through the esophageal tear for drainage and irrigation of the contaminated mediastinum. Instead of using the nasal route, McNamee and associates introduced the sump through a pharyngostomy. This technique was initially described by Cetrullo and DiNino in 1977 [2] and more recently by Kanschin and Pogodina in 1983 [3]. In both reports the irrigation catheter was introduced by the transnasal route. However, the basic concept of irrigating the periesophageal area with a catheter going through the tear in the esophageal wall is described in these two reports.

Three cases of instrumental perforation recognized during endoscopy were also treated by McNamee and associates using the same technique with good results. When instrumental perforation occurs, the patient is prepared with an empty stomach for endoscopy.

However, the pathology is different in spontaneous rupture, which usually occurs in a patient with a full stomach. The pressures required to rupture the esophagus have been demonstrated to be greater than 5 psi [4], and when the esophagus ruptures under this high pressure a true explosion in the posterior mediastinum occurs with large areas of the mediastinum flooded with chemical substances, enzymes, food, and bacteria.

For these patients with life-threatening mediastinitis after spontaneous rupture not amenable to direct repair or after a failed repair, my colleagues and I [5] continue to advocate an irrigation technique described in 1986. This technique uses a Levine tube positioned inside the esophagus proximal to the esophageal tear and a chest tube positioned outside the esophagus. Saline solution is used for continuous irrigation through the Levine tube. The chest tube is connected to suction, establishing in this way a continuous irrigation of the mediastinum. Good results have been consistently obtained with this technique.

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Reply

To the Editor:

I thank Dr Santos for drawing our attention to the report by Drs Cetrullo and DiNino in 1977, who essentially were using the same principles as our technique except that we opted to introduce the suction catheter through a pharyngoscopy, believing that this would be more comfortable for the patient in the long term. It serves to emphasize that however assiduous one is in searching the literature, important contributions can be missed, and that what we thought was new is only new in one small respect and the basic concept is not original. Nevertheless I am pleased that we have reported our experience and that this was accepted by *The Annals of Thoracic Surgery* for publication, for it does serve to remind our colleagues of this important conservative approach to the difficult problem of the esophagus whose integrity has been breached.

One must treat each case on its merits, but I cannot but believe that with some of our cases, had we been more aggressive, there might not have been such a successful outcome. I also believe that morbidity and the time in hospital were reduced by creating an internal sinus rather than external fistula. It is for others to judge, but I commend it as a possibility to be considered in the various situations that we have described in our report.

I agree entirely with Dr Santos about the acute rupture and that our method of internal drainage is unlikely to be rewarded with success. I congratulate him on his technique of pleural irrigation, which should be considered in the presence of gross mediastinal sepsis. Our personal preference is for early ruptures to be repaired directly, but in patients in whom the rupture is more than 24 hours old, as is very often the case, we have opted for cervical esophagostomy, subtotal esophageal resection, and gastrostomy to be followed at a later date by reconstituting swallowing with a gastric pull-up. Clearly there are patients who at the time of presentation are not fit to withstand such a major operation, and Dr Santos' alternative could be life saving and buy time until definitive treatment is more appropriate.

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Retrograde Disc Escape in a Harken Mitral Valve Prosthesis 21 Years After Implantation

To the Editor:

The Harken discoid mitral valve, introduced in 1967 [1] and available in three sizes (small, medium, and large), consisted of a barium-impregnated silicone rubber disc inside a highly polished titanium cage, with two metallic struts crossing each other at 90-degree angles. Scarce data are published on mechanical dysfunctions of this valve or its long-term performance. We have seen a rare type of dysfunction very late (21 years) after implantation.

A 54-year-old woman received a Harken medium-size mitral valve in 1969 for severe mitral regurgitation early after attempted redo mitral commissurotomy. In 1981 she had a stroke (left hemiparesis) followed by complete neurological recovery. She remained in New York Heart Association class I or II up to May 1990 when sudden respiratory distress was noted. On admission the patient was in pulmonary edema and no prosthetic sounds were heard; she was intubated, and mechanical ventilation and inotropic support were started. Echocardiography suggested mitral valve thrombosis and massive tricuspid regurgitation. Thrombus lysis with anisoylated plasminogen streptokinase activator complex was attempted and followed by some clinical improvement; the physician caring for the patient reported reappearance of prosthetic valve sounds. Later on, the clinical status of the patient worsened and high fever appeared. A new color echocardiogram and Doppler study suggested mild mitral and moderate tricuspid regurgitation. Hours later giant V waves were noted in the capillary wedge pressure and a new echocardiogram showed massive mitral regurgitation.

At operation the left atrium was partially covered by mural thrombus with some areas of calcification, and the disc of the prosthesis was found freely floating in the atrium. On closer examination the disc had a nick in one of the borders, but no thrombus or any other macroscopic abnormality was detected. The cage of the prosthesis was well incorporated into the annulus, without any signs of thrombosis, leak, or infection; no wear of the seat was identified. The cage was explanted and a 29-mm St. Jude Medical valve prosthesis was implanted with standard techniques. Continuous hemodialysis for established renal failure was performed during bypass. Respiratory, hepatic, and renal failure requiring hemodialysis, as well as severe neurological dysfunction, complicated the first postoperative days, but the patient subsequently improved and was finally discharged in good condition. Six months after operation she was leading an active life, again in functional class II.

Discoid valve prostheses, mainly used in the late 1960s and early 1970s, showed vulnerability to catastrophic failure and high rates of thromboembolism as their most important weaknesses. Retrograde disc escape into the left atrium is a rare complication of discoid valves, previously reported [2] in a patient with a Cross-Jones mitral valve prosthesis. The echocardiographic sequence of events in our patient, suggesting impaction of the disc eventually leading to retrograde disc escape, seems rather unusual. Although valve thrombosis may be more frequent than disc variance in patients with discoid valve prostheses [3], we now believe that urgent operation instead of thrombolysis should be performed in a patient with signs of obstruction of a Harken valve prostheses if the diagnosis of disc variance cannot be ruled out.

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Vagal Schwannoma

To the Editor:

We read with interest the article by Heitmiller and colleagues [1] detailing their experience with a patient who had a benign schwannoma of the right intrathoracic vagus nerve, and their review of the literature which showed this to be a rare neurogenic tumor of the thorax. Recently, a 38-year-old man came to our attention for evaluation and treatment of a 4.0-cm mass in the left posterior mediastinum adjacent to the aortic knob, which was detected on routine chest roentgenogram (Fig 1). A history of blunt chest

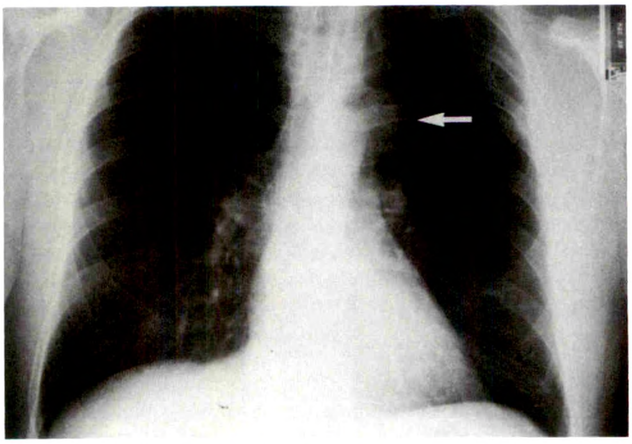

Fig 1. Standard posteroanterior chest roentgenogram with an abnormal 4-cm density adjacent to the aortic knob.

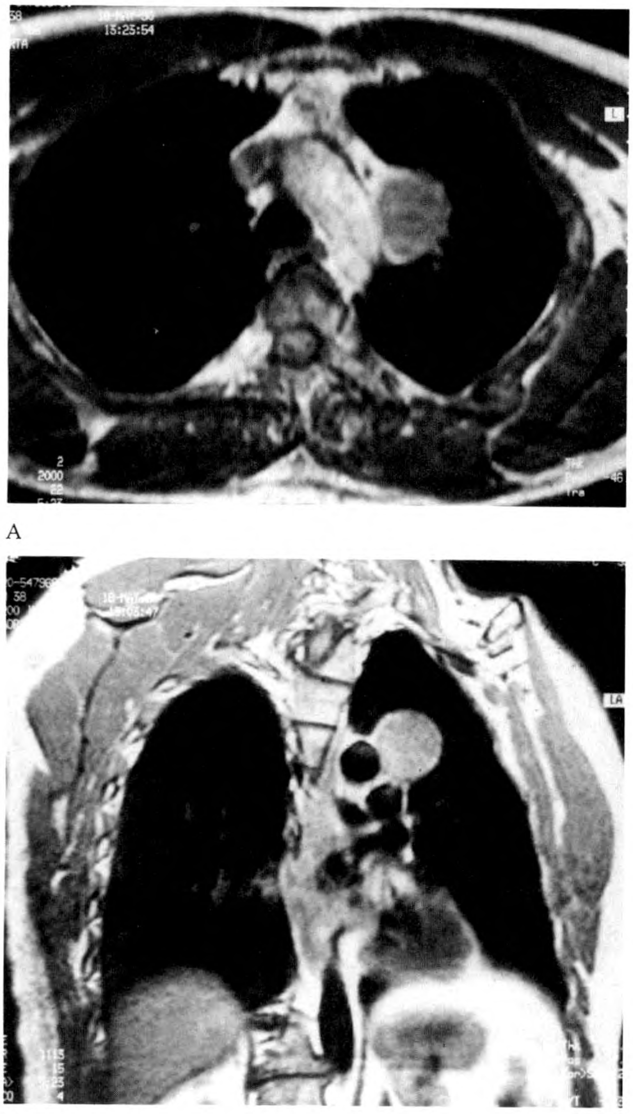

Fig 2. Magnetic resonance images demonstrating a well-delineated mass adjacent to the aortic arch, with a discrete tissue plane between the lesion and the aorta.

trauma 1 year earlier aroused suspicion that this might be a pseudoaneurysm of the descending aorta, which could not be excluded by contrasted computed tomographic scan of the chest. A magnetic resonance imaging scan clearly demonstrated the mass to be distinct from the aorta. The mass did not contain signal compatible with extracellular methemoglobin or hemosiderin, excluding a vascular origin (Fig 2). The patient was explored through a left posterolateral thoracotomy, where a firm, 4.0 × 4.0 × 3.5-cm mass was found. The mass originated from the vagus nerve just distal to the recurrent laryngeal nerve. The recurrent laryngeal nerve was dissected free, and the mass was excised along with a segment of the vagus nerve. Pathologically, the tumor was a benign schwannoma. The patient's recovery was uneventful.

This case was interesting to us for two reasons: (1) the rarity of a schwannoma taking origin from the intrathoracic vagus nerve and (2) the location of the tumor adjacent to the aortic knob in conjunction with a history of chest trauma that aroused suspicion that the mass was a pseudoaneurysm, which could not be excluded by computed tomographic scan. A magnetic resonance imaging scan, however, clearly showed the mass to be distinct and separate from the aorta and, therefore, not of vascular origin.

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Reference

1. Heitmiller RF, Labs JD, Lipsetl PA. Vagal schwannoma. Ann Thorac Surg 1990;50:811–3.

Reply

To the Editor:

Recently there have been several reports on intrathoracic vagal schwannomas, which have focused attention on these unusual tumors [1, 2]. The case presented by Drs Walker and Maguire represents another example and underscores the principal characteristics of these tumors, which are as follows: (1) they are rare, (2) the tumors are invariably discovered on routine chest roentgenograms and produce no symptoms, (3) tumors may occur anywhere along the intrathoracic vagus nerve, and depending on their size and location, may radiographically mimic other intrathoracic pathology, (4) thoracotomy is necessary for definitive diagnosis and treatment, (5) the vast majority of these tumors are benign, and (6) complete resection is curative.

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References

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THE SOCIETY OF THORACIC SURGEONS

Twenty-eighth Annual Meeting February 3–5, 1992 Walt Disney World Dolphin Hotel, Lake Buena Vista (Orlando), Florida

The Twenty-eighth Annual Meeting of The Society of Thoracic Surgeons will be held at the Walt Disney World Dolphin Hotel on February 3–5, 1992, preceded by the Twenty-fifth Postgraduate Program on Sunday, February 2. The meeting is open to all duly qualified physicians. Both members and nonmembers are urged to register in advance.

The 1991 Program Committee is very pleased with the response to the abstract call, counting 358 abstracts submitted. In addition to the quantity, the high quality of the abstracts made possible the development of a program that represents all aspects of adult and pediatric cardiac surgery and general thoracic surgery. The utilization of breakout sessions continued to be very successful at the last Annual Meeting. On Monday afternoon, there will be parallel research-oriented sessions entitled "General Thoracic Research," "Cardiac Research," and "Transplantation/Congenital Research" running simultaneously from 1:30 to 2:45.

On Tuesday afternoon, all sessions will run from 1:30 to 5:00 PM. Session I will include "Transplantation" followed by "General Thoracic" segments. Session II will include "Valvular Surgery" followed by "Congenital Surgery," and Session III comprises the "Acquired Heart Disease I and II" segments.

The popular Tuesday morning Problem Case Presentations will be scheduled opposite the new Technical "How I Do It" Sessions. The Wednesday morning Clinical Workshops will also continue.

The Committee had a significant increase in the number of video tapes submitted for the Annual Movie Night, making it possible to develop an excellent program representative of all aspects of the specialty. The movie night session will be in the Walt Disney World Dolphin Hotel.

At 11:15 AM on Tuesday, Mark V. Braimbridge of The Rayne Institute, St. Thomas Hospital, London, England, will present the second Herbert Sloan Lecture entitled "The International Cardiothoracic Surgeon."

Registration and Fees

An advance registration form with hotel reservation information and details regarding spouses' activities will be mailed to Society members. Nonmembers should write to the Secretary, Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611.

Members are not required to pay a registration fee for the Scientific Program. Residents and fellows may register without payment of a registration fee upon presentation of a letter from their chief of service. The fee for other physicians is $250, except guest speakers, authors, coauthors, and invited discussants. Nurses and paramedical personnel may register upon payment of a $40 fee and presentation of a letter of introduction from a Society member.

The Postgraduate Program is sponsored by the Committee on Postgraduate Education of The Society. Registration fee, which includes lunch, for the Postgraduate Program is $65 for all physicians, residents, and fellows.

There will be no mail-in registration for the concurrent Problem Case Presentations or the Technical "How I Do It" Sessions on Tuesday, February 4, or the Clinical Workshops on Wednesday, February 5. Attendance will be by ticket only, and each seminar is limited to 40 physicians. Tickets can be purchased at The Society's registration desk beginning Saturday, February 1, at 1:30 PM. A continental breakfast is included.

Presentation and Publication

Papers presented at the Scientific Session must be submitted to the Secretary at the time of presentation, or may be submitted to the Editor prior to the meeting for possible earlier publication.

Speakers for the Scientific Sessions should limit their presentations to 10 minutes. They are requested to meet with the projectionist in the preview room prior to the opening of their session to prepare their slides for presentation.

Those who wish to discuss a paper may write the Secretary prior to the meeting. Discussants from the floor should submit their names to the Moderator at the podium prior to the opening of the session during which the paper is to be presented. Discussants from the floor are limited to 2 minutes; primary discussants are limited to 3 minutes.

Discussants will receive a copy of their remarks shortly after the Annual Meeting. Any delay in the return of the corrected discussion to the Editor will hold up publication of the paper. Timely response of all authors and discussants is, therefore, urged since delays are unfair to those who promptly return their material for publication.

Accreditation

The Society of Thoracic Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. One credit hour in Category I of the Physician's Recognition Award of the American Medical Association may be claimed for each hour of participation by the individual physician. The Postgraduate Program is authorized for eight hours of Category I CME credit.

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TWENTY-FIFTH POSTGRADUATE PROGRAM
THE SOCIETY OF THORACIC SURGEONS

Sunday, February 2, 1992
Walt Disney World Dolphin Hotel, Lake Buena Vista (Orlando), Florida

8:00–12:00 **Morning Session**

8:00 Introduction to the 1992 Postgraduate Course
Cary W. Akins, Boston, MA, Co-Chairman, Postgraduate Committee

I. Mini Symposium: Reoperative Myocardial Revascularization

8:15 Indications for and Timing of Coronary Reoperations
Ellis L. Jones, Atlanta, GA

8:30 Choice of Bypass Conduits for Coronary Reoperation
Noel L. Mills, New Orleans, LA

8:45 Myocardial Preservation for Coronary Reoperation
Richard D. Weisel, Toronto, Ontario, Canada

9:00 Should Old Patent Grafts Be Preserved?
Hartzell V. Schaff*, Rochester, MN

II. Mini Symposium: Problems in Thoracic Aortic Surgery

9:15 Ascending Aortic Dissection
Nicholas T. Kouchoukos, St. Louis, MO

9:30 Spinal Cord Preservation in Descending Aortic Surgery
Joseph N. Cunningham, Jr, Brooklyn, NY

9:45 Traumatic Disruption of the Thoracic Aorta
Alan D. Hilgenberg, Boston, MA

10:00 Recurrent Coarctation of the Aorta
John W. Brown, Indianapolis, IN

10:15–11:00 Coffee Break: Visit Exhibits

11:00 **III. C.C.C.E.T.S. Basic Science Lecture****
Introduction
George C. Kaiser, St. Louis, MO, Chairman, Coordinating Committee for Continuing Education in Thoracic Surgery

Optimal Transfusion Practice in 1992
Harold A. Oberman*, The University of Michigan, University Hospital, Ann Arbor, MI

12:00–1:30 Luncheon

Luncheon Address: Military Preparations for Managing Thoracic and Cardiovascular Trauma in International Conflicts
David J. Cohen, San Antonio, TX

1:30–4:45 **Afternoon Session**

Introduction
Robert J. Ginsberg, New York, NY, Co-Chairman, Postgraduate Committee

IV. Mini Symposium: Esophageal Reflux

1:30 Indications for Antireflux Procedures
Robert J. Ginsberg, New York, NY

1:40 Belsey Repair—Indications, Contraindications, Technique, and Late Results
David B. Skinner, New York, NY

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* By Invitation
** Open Session

1:55 Nissen Repair—Indications, Contraindications, Technique, and Late Results
Tom R. DeMeester, Los Angeles, CA

2:10 Hill Repair—Indications, Contraindications, Technique, and Late Results
Donald E. Low*, Seattle, WA

2:25 Collis Gastroplasty—Indications, Contraindications, Technique, and Late Results
F. Griffith Pearson, Toronto, Ontario, Canada

2:40 Panel Discussion

3:00–3:45 Coffee Break: Visit Exhibits

V. State of the Art Lectures

3:45 Conservative Mitral Valve Surgery
Delos M. Cosgrove III, Cleveland, OH

4:00 Conservative Aortic Valve Surgery
Carlos G. Duran, Riyadh, Saudi Arabia

4:15 Thoracoscopy—Indications and Technique
Ralph J. Lewis, New Brunswick, NJ

PROGRAM
TWENTY-EIGHTH ANNUAL MEETING
THE SOCIETY OF THORACIC SURGEONS

February 3–5, 1992

February 3, 1992 (Monday)
Morning

8:00 Business Meeting (members only)

8:05 Opening Remarks for General Scientific Session

8:15–11:15 **Scientific Session**

1. **J. Maxwell Chamberlain Memorial Paper**

   Fontan's Procedure for Hypoplastic Left Heart Syndrome
   William I. Norwood, Jr, Marshall L. Jacobs, and John D. Murphy, Philadelphia, PA

2. Aprotinin Therapy for Reoperative Myocardial Revascularization: A Randomized, Double-Blind, Placebo-Controlled Study
   Delos M. Cosgrove III, Blaine Heric, Bruce W. Lytle, Paul C. Taylor, Roberto Novoa, Leonard A. R. Golding, Robert W. Stewart, Patrick M. McCarthy, and Floyd D. Loop, Cleveland, OH

3. Barrett's Esophagus With High-Grade Dysplasia: An Indication for Esophagectomy?
   Manual Pera, Victor F. Trastek, Herschel A. Carpenter, Mark S. Allen, Claude Deschamps, and Peter C. Pairolero, Rochester, MN

4. Composite Valve Graft Replacement of the Proximal Aorta: Comparison of Techniques in 348 patients
   Lars G. Svensson, E. Stanley Crawford, Kenneth R. Hess, Joseph S. Coselli, and Hazim J. Safi, Houston, TX

5. Management of Thyroid Carcinoma Invading the Airway
   Hon-Chi Suen, Hermes C. Grillo, Douglas J. Mathisen, and John C. Wain, Jr, Boston, MA

6. Pediatric Lung Transplantation for Pulmonary Hypertension and Congenital Heart Disease
   Thomas L. Spray, George B. Mallory, Charles E. Canter, Charles B. Huddleston, and Larry R. Kaiser, St. Louis, MO

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* By Invitation

7. The Radial Artery for Coronary Bypass Operations: Revival of an Old Conduit
Christophe Acar, Victor A. Jebara, Michele Portoghèse, Patrice Dervanian, Miguel De Souza, Juan C. Chachques, Jean-Noël Fabiani, Alain Deloche, and Alain Carpentier, Paris, France

11:15–12:00 **Presidential Address**
If Not the Art, Why the Science?
Robert W. Jamplis

Afternoon

Parallel Scientific Sessions
Session I

1:30–2:45 **General Thoracic Research**
Moderators: Victor F. Trastek, Rochester, MN, and John C. Wain, Jr, Boston, MA

8. Optimal Pleurodesis: A Comparison Study
Michael Bresticker, Junichi Oba, Joseph LoCicero III, and Rodney Greene, Chicago, IL, and Boston, MA

9. The Present Role of Thoracoscopy in the Diagnosis and Treatment of Diseases of the Chest
Michael Mack, Ronald Aronoff, Tea Acuff, Mark Douthit, Richard Bowman, and William H. Ryan, Dallas, TX

10. Acute Rejection of Pulmonary Allografts: Incidence, Severity, and Effect of Induction With Rabbit Antithymocyte Globulin
Bartley P. Griffith, John M. Armitage, Robert L. Kormos, Gary C. Marrone, Irvin Paradis, James Dauber, Samuel A. Yousem, and Robert L. Hardesty, Pittsburgh, PA

11. Early Cellular Events in the Lung Allograft
Riad Adoumie, Cyril Serrick, and Hani Shennib, Montreal, Quebec, Canada, and Marseilles, France

12. Predictive Criteria for the Need of Extracorporeal Circulation in Single-Lung Transplantation
Stephan W. Hirt, Axel Haverich, Thorsten Wahlers, Jochen Schafers, Aiman Alken, and Hans G. Borst, Hannover, Germany

Session II

1:30–2:45 **Cardiac Research**
Moderators: Richard D. Weisel, Toronto, Ontario, Canada, and Peter K. Smith, Durham, NC

13. Right Heart Assist With Skeletal Muscle Ventricles: Up to 16 Weeks' Experience in Circulation
Hiroshi Niinami, Timothy L. Hooper, Renato Ruggiero, Robert L. Hammond, and Larry W. Stephenson, Detroit, MI

14. A Quantitative Electron Microscopic Analysis of Injury to Leaflet Fibroblasts Associated With Variable Warm Ischemic Time in Human Cryopreserved Valve Conduits
Donald Crescenzo, Stephen Hilbert, Robert Messier, Jr, Patrick Domkowski, Mary Kay Barrick, Perry Lange, Ali Analouei, Robert B. Wallace, and Richard A. Hopkins, Washington, DC

15. Leukocyte Depletion in a Neonatal Model of Cardiac Surgery
Ian Wilson, Joseph M. DiNatale, William E. Curtis, A. Marc Gillinov, Duke E. Cameron, and Timothy J. Gardner, Baltimore, MD

16. Bioassay of Endothelium-Derived Relaxing Factor From Internal Mammary Arteries: Implications for Early and Late Bypass Patency
Paul J. Pearson, Paulo R. B. Evora, and Hartzell V. Schaff, Rochester, MN

17. Expression of Adhesion Molecules by In Vivo Fresh and Cryopreserved Aortic Valve Allografts
Michael S. Mulligan, Thomas T. Tsai, James M. Kneebone, Peter A. Ward, and Flavian M. Lupinetti, Ann Arbor, MI

Session III
1:30–2:45 **Transplantation/Congenital Research**
Moderators: Adnan Cobanoglu, Portland, OR, and Jeffrey M. Pearl, Los Angeles, CA

18. Inhibition of Contracture by Butanedione Monoxime Extends Myocardial Cold Ischemic Tolerance
James C. Stringham, Karen L. Paulsen, James H. Southard, Barry L. Fields, and Folkert O. Belzer, Madison, WI

19. The Use of Epicardial Electrocardiograms for Detecting Cardiac Allograft Rejection
Eric D. Irwin, Richard Bianco, Rose Clack, David Slovut, Raouf Nakhleh, R. Morton Bolman III, and Sara Shumway, Minneapolis, MN

20. University of Wisconsin Solution Induces Endothelial Functional Damage During Long-Term Storage in the Isolated Rat Heart
Raymond Cartier, Charleen Hollmann, Josie Buluran, and Yves Leclerc, Montreal, Quebec, Canada

21. Cerebral Blood Flow and Metabolism in Hypothermic Circulatory Arrest
Craig K. Mezrow, Ali M. Sadeghi, Alejandro Gandsas, Howard Shiang, Dale Levy, Robert Green, Ian R. Holzman, and Randall B. Griepp, New York, NY

22. Optimal Strategies for Cerebral Protection During Neonatal Heart Repair
James Mault, Shigeaki Ohtake, Mary Lilly, Jeffrey Heinle, William J. Greeley, and Ross M. Ungerleider, Durham, NC

3:30–5:00 Business Meeting (members only)

Evening

8:00–10:00 **Surgical Motion Pictures**
Moderators: Delos M. Cosgrove III, Cleveland, OH, and Douglas J. Mathisen, Boston, MA

1. Aortic Operation for Chronic Type A Dissection, Act II: Elephant Trunk Replacement of the Proximal Descending Aorta
Hans G. Borst and Joachim Laas, Hannover, Germany

2. Restoring Normal Tracheal Architecture Without Resection in Diffuse Tracheal Malacia With Expiratory Collapse
George B. Haasler, Jonathan Hammond, Akira Funahashi, David Rosenzweig, Marilyn Corlew, and Karin Madsen, Milwaukee, WI

3. Aortic Root Replacement With a Cryopreserved Homograft in a Child
Dominique Metras and Bernard Kreitmann, Marseille, France

4. Sleeve Pneumonectomy With Combined Resection of Superior Vena Cava for Lung Cancer
Ryosuke Tsuchiya, Haruhiko Kondo, Tomoyuki Goya, Tsuguo Naruke, and Keiichi Suemasu, Tokyo, Japan

5. Patch Angioplasty for Isolated Left Main Coronary Artery Stenosis
Nicholas P. Rossi, Iowa City, IA

6. Panoramic View of Thoracoscopic Procedures
Stephen R. Hazelrigg, Paul E. Seifert, and James E. Auer, Milwaukee, WI

7. Radical Aortic Root Enlargement
Adnan Cobanoglu, Portland, OR

8. En Bloc Resection of a Mediastinal Malignant Fibrous Histiocytoma With Aortic Arch Reconstruction
Joe B. Putnam, Michael S. Sweeney, N. E. J. Papodapolous, E. M. Milissinos, and J. P. Williams, Houston, TX

9. The Maze Procedure for Atrial Fibrillation: Surgical Technique
Patrick M. McCarthy and James L. Cox, Cleveland, OH, and St. Louis, MO

February 4, 1992 (Tuesday)
Morning

7:00–8:00 **Breakfast Sessions**

No advance registration. Attendance by ticket only and limited to 40 per seminar. Tickets can be purchased at registration desk beginning Saturday, February 1, at 1:30 PM. Price of ticket covers attendance at seminar and continental breakfast. These sessions run concurrently.

Problem Case Presentations (see pp 1381–2 for program)

Technical Sessions—How I Do It (see p 1382 for program)

8:15–11:15 **Scientific Session**

23. Treatment of AIDS-Related Bronchopleural Fistula by Pleurectomy
Bernard K. Crawford, Aubrey C. Galloway, Arthur D. Boyd, and Frank C. Spencer, New York, NY

24. Video Thoracoscopic Wedge Resesctions of the Lung
Daniel L. Miller, Mark S. Allen, Victor F. Trastek, Claude Deschamps, and Peter C. Pairolero, Rochester, MN

25. Intraaortic Balloon Pump: Patterns of Usage and Outcome in Cardiac Surgical Patients
Lawrence L. Creswell, James L. Cox, Thomas B. Ferguson, Sr, Nicholas T. Kouchoukos, Thomas L. Spray, Michael K. Pasque, T. Bruce Ferguson, Jr, Thomas H. Wareing, Charles B. Huddleston, and Michael Rosenbloom, St. Louis, MO

26. Postcardiotomy Centrifugal Mechanical Ventricular Support
Leonard A. R. Golding, Ray Crouch, Robert W. Stewart, Roberto Novoa, Patrick M. McCarthy, Paul C. Taylor, Floyd D. Loop, and Delos M. Cosgrove III, Cleveland, OH

27. In Situ Reconstruction for Mycotic Aneurysm of the Thoracic Aorta
Miralem Pasic, Andreas Laske, Thierry Carrel, Erwin Bauer, Ludwig von Segesser, and Marko I. Turina, Zurich, Switzerland

Invited Speaker

100 Consecutive Imaged Thoracoscopic Surgical Procedures
Ralph J. Lewis, New Brunswick, NJ

28. Factors Affecting Mitral Valve Replacement in 317 Survivors After Mitral Valve Reconstruction
Javier Fernandez, Glenn W. Laub, Mark S. Adkins, William A. Anderson, Chao Chen, Kathryn J. Hirschfeld, Bridget Bailey, and Lynn B. McGrath, Browns Mills, NJ

11:15–12:00 **The Herbert Sloan Lecture**

The International Cardiothoracic Surgeon
Mark V. Braimbridge, London, England

Afternoon

1:30–5:00 **Parallel Scientific Sessions**
Session I

1:30–2:45 **Transplantation**
Moderators: Walter H. Merrill, Nashville, TN, and Patrick M. McCarthy, Cleveland, OH

29. Cardiac Retransplantation: A Viable Option?
Shreekanth V. Karwande, Dale G. Renlund, Wayne E. Richenbacher, Donald B. Doty, William A. Gay, Jr, and Utah Cardiac Transplant Program, Salt Lake City, UT

30. The Clinical Trial of FK 506 Immunosuppression in Adult Cardiac Transplantation
John M. Armitage, Robert L. Kormos, John Fung, Thomas E. Starzl, and Bartley P. Griffith, Pittsburgh, PA

31. Successful Lung Transplantation in the Pediatric Population
Michel Noirclerc, Jean Camboulives, Laurent Viard, Marie-Ange Silicani, Monique Badier, Jean François Dumon, Bernard Kreitmann, Dominique Metras, David S. Mulder, Christo I. Tchervenkov, Jean François Morin, Benoit DeVarennes, Robert Levy, and Hani Shennib, Marseille, France, and Montreal, Quebec, Canada

32. Mediastinitis in Heart Transplantation
Shreekanth V. Karwande, Dale G. Renlund, Stephanie L. Olsen, Wayne E. Richenbacher, Roger C. Millar, William A. Gay, Jr, and Utah Cardiac Transplant Program, Salt Lake City, UT

33. Incidence and Recurrence of Cytomegalovirus Infection of the Upper Gastrointestinal Tract in Cardiac Transplant Patients
Francisco A. Arabia, Luis J. Rosado, Casey L. Huston, Gulshan K. Sethi, and Jack G. Copeland III, Tucson, AZ

3:30–5:00 **General Thoracic**
Moderators: Richard F. Heitmiller, Baltimore, MD, and Mark K. Ferguson, Chicago, IL

34. **Thoracic Surgery Directors Association Resident Research Award Paper**
Intrathecal Perfusion of an Oxygenated Perfluorocarbon Emulsion Prevents Paraplegia After Extended Normothermic Aortic Cross-Clamping
Robert E. Maughan, Chittur Mohan, Ira M. Nathan, Joseph N. Cunningham, Jr, Enrico Ascer, Peter Damiani, and Corrado P. Marini, Brooklyn, NY

35. Thoracoscopic Resection of 52 Pulmonary Lesions
Rodney J. Landreneau, Stephen R. Hazelrigg, Peter F. Ferson, Joel A. Johnson, Weerchai Nawarawong, Theresa Boley, David B. Herlan, and Robert D. Dowling, Pittsburgh, PA

36. Clinical Manifestation of Mediastinal Fibrosis and Histoplasmosis
Douglas J. Mathisen and Hermes C. Grillo, Boston, MA

37. Primary Bony and Cartilaginous Sarcomas of Chest Wall: Results of Therapy
Michael E. Burt, Michael Fulton, Susan Dunlop, Martin Karpeh, Manjit S. Bains, Nael Martini, Patricia M. McCormack, Valerie W. Rusch, and Robert J. Ginsberg, New York, NY

38. Long-Term Effect of Total Fundoplication on the Myotomized Esophagus
Ph. Topart, C. Deschamps, and Andre Duranceau, Montreal, Quebec, Canada

39. Symptomatic and Functional Assessment of Failed Antireflux Procedures
Jean-Marie Collard and Tom R. DeMeester, Los Angeles, CA

1:30–5:00 **Session II**

1:30–2:45 **Valvular Surgery**
Moderators: D. Craig Miller, Stanford, CA, and Stephen B. Colvin, New York, NY

40. Valvular Disease in the Elderly: Influence on Surgical Results
Elizabeth A. Davis, Timothy J. Gardner, A. Marc Gillinov, William A. Baumgartner, Duke E. Cameron, Vincent L. Gott, Levi Watkins, Jr, and Bruce A. Reitz, Baltimore, MD

41. Stentless Porcine and Pericardial Bioprostheses in Aortic Position
Riccardo Casabona, Ruggero De Paulis, Giuseppe Zattera, Michele di Summa, Mario Vrandecich, and Mario Morea, Torino, Italy, and Belo Horizonte, Brazil

42. Lunulae Hypertrophy and Aortic Insufficiency
Nadiv Shapira, Javier Fernandez, Kathryn J. Hirschfeld, Antonio J. Serra, Kathleen W. McNicholas, and Gerald M. Lemole, Wilmington, DE

43. Clinical and Hemodynamic Performance of the Hancock II Bioprosthesis
Tirone E. David, Susan Armstrong, and Zhao Sun, Toronto, Ontario, Canada

44. Left Atrial Isolation Associated With Mitral Valve Operations
Angelo Graffigna, Gaetano Minzioni, Francesco Pagani, and Mario Viganó, Pavia, Italy

3:30–5:00 **Congenital Surgery**
Moderators: John W. Hammon, Jr, Winston-Salem, NC, and Marshall L. Jacobs, Philadelphia, PA

45. Coarctation of the Aorta in Infants: Does the Aortic Arch Grow After Repair?
John L. Myers, Brian McConnel, and John A. Waldhausen, Hershey, PA

46. Optimum Treatment of Discrete Subaortic Stenosis
Flavian M. Lupinetti, Ara K. Pridjian, Louise B. Callow, and Edward L. Bove, Ann Arbor, MI

47. Advantages of Repair of Atrioventricular Canal Defect With Two-Patch Technique and Tricuspidization of the Mitral Valve
Zahid Amin, Rajesh Sharma, Hashmat Ashraf, and Sambamurthy Subramanian, Miami, FL

48. Damus-Kaye-Stansel Procedure With Cavopulmonary Connection in Infants With Single Ventricle and Subaortic Obstruction
Charles B. Huddleston and Thomas L. Spray, St. Louis, MO

49. Anomalous Left Coronary Artery From the Pulmonary Artery: Early and Late Results After Isolated Aortic Reimplantation
Pascal R. Vouhé, Daniel Tamisier, Daniel Sidi, Francoise Vernant, Philippe Mauriat, Philippe Pouard, and Francine Leca, Paris, France

50. Cardiopulmonary Failure After Congenital Heart Operation: Results of Treatment With Extracorporeal Membrane Oxygenation
Stanley Ziomek, James E. Harrell, Jr, J. W. Fasules, Sherry Faulkner, Carl Chipman, Michele Moss, Elizabeth Frazier, and Stephen VanDevanter, Little Rock, AR

1:30–5:00 **Session III**

1:30–2:45 **Acquired Heart Disease I**
Moderators: Fred A. Crawford, Jr, Charleston, SC, and Gus J. Vlahakes, Boston, MA

51. Does a Posterior Aneurysm Increase the Risk of Endocardial Resection?
Irving L. Kron, Pierre Theodore, Terry L. Flanagan, John A. Kern, David E. Haines, Michael J. Barber, and John P. DiMarco, Charlottesville, VA

52. Prolonged Open Sternotomy and Delayed Sternal Closure After Cardiac Operations
Anthony P. Furnary, James A. Magovern, Kathleen A. Simpson, and George J. Magovern, Sr, Pittsburgh, PA

53. The Systemic Effects of Normothermic Versus Hypothermic Cardiopulmonary Bypass
George T. Christakis, Jean Paul Koch, Kathy Deemar, Bernard S. Goldman, and Erluo Chen, Toronto, Ontario, Canada

54. Subacute Rupture of the Left Ventricle After Myocardial Infarction: Repair With a Sutureless Technique
Alejandro Aris, José M. Mesa, José M. Padró, Jorge Silvestre, José Luis Larrea, José M. Caralps, and Francisco Cerrón, Barcelona and Madrid, Spain

55. Subcutaneous Use of Erythropoietin in Heart Operations
Yasunori Watanabe, Katsuo Fuse, Yoshihiro Naruse, Toshiya Kobayashi, Shin Yamamoto, and Hiroaki Konishi, Tokyo, Japan

3:30–5:00 **Acquired Heart Disease II**
Moderators: Sidney Levitsky, Boston, MA, and Edward D. Verrier, Seattle, WA

56. Warm Aerobic Blood Cardioplegia: Superior Protection During Revascularization for Acute Myocardial Ischemia
W. Morris Brown, John L. Jay, John P. Gott, Alice H. Huang, Pan-Chih, Lynne M. A. Dorsey, Sara L. Katzmark, William S. Horsley, and Robert A. Guyton, Atlanta, GA

57. Superior Myocardial Protection by Normothermic Aerobic Arrest Over Ischemic Arrest for High-Risk Patients
Gregory Louis Kay, Atsushi Aoki, Pablo Zubiate, Curtis Prejean, Joseph Ruggio, and Jerome Harold Kay, Los Angeles, CA

58. Normothermic Continuous Retrograde Blood Cardioplegia: Is Aortic Cross-Clamping Still Synonymous With Myocardial Ischemia?
Philippe Menasché, Jacqueline Peynet, Bernard Touchot, Mohamed Aziz, Sam Haydar, Ghislaine Perez, Gérard Bloch, and Armand Piwnica, Paris, France

59. Continuous Warm Blood Retrograde Cardioplegia: Maintenance of Myocardial Homeostasis and Elimination of Myocardial Ischemia in Humans
Steven R. Gundry, Nan Wang, David Bannon, Robert E. Vigesaa, Clifford Eke, Simon Pain, and Leonard L. Bailey, Loma Linda, CA

60. Competitive Flow From a Fully Patent Coronary Artery Does Not Limit Acute Mammary Artery Graft Flow
Paul A. Spence, Robert M. Lust, Richard S. Zeri, Stanley R. Jolly, Prabodh M. Mehta, Masaki Otaki, You Su Sun, and W. Randolph Chitwood, Greenville, NC

61. Cryopreserved Allograft Veins as Alternative Coronary Artery Bypass Conduits—Early Phase Results
Glenn W. Laub, S. Muralidharan, Robert Clancy, Jay Eldredge, Chao Chen, Mark S. Adkins, Javier Fernandez, William A. Anderson, and Lynn B. McGrath, Browns Mills, NJ

7:15 **Reception for Members and Guests**

February 5, 1992 (Wednesday)
Morning

7:00–8:00 **Breakfast Sessions**

No advance registration. Attendance by ticket only and limited to 40 per seminar. Tickets can be purchased at registration desk beginning Saturday, February 1, at 1:30 PM. Price of ticket covers attendance at seminar and continental breakfast. These sessions run concurrently.

Clinical Workshops (see p 1382 for program)

8:15–12:00 **Scientific Session**

62. Anterior Mediastinal Tracheostomy With and Without Cervical Exenteration
Mark B. Orringer, Ann Arbor, MI

63. Survival After Resection in Stage II Non–Small Cell Lung Cancer
Nael Martini, Michael E. Burt, Manjit S. Bains, Patricia M. McCormack, Valerie W. Rusch, and Robert J. Ginsberg, New York, NY

64. Postpneumonectomy Syndrome: Surgical Management and Results
Hermes C. Grillo, Jo-Anne O. Shepard, and Douglas J. Mathisen, Boston, MA

65. Repair of Total Anomalous Pulmonary Venous Return in Neonates and Young Infants: Results in the Current Era
Adnan Cobanoglu, Portland, OR

66. Anatomical Correction of Tetralogy of Fallot: Ten or More Years' Follow-Up
Gaetano Azzolina, Massa, Italy

67. Effect of Coronary Bypass Grafting on Long-Term Survival After Postinfarction Ventricular Septal Defect Repair
Derek D. Muehrcke, Willard M. Daggett, Jr, Mortimer J. Buckley, Cary W. Akins, Alan D. Hilgenberg, and W. Gerald Austen, Boston, MA

68. Early and Late Cardiac and Neurologic Results After Combined Carotid and Coronary Revascularization: The Preferred Approach for the Severe Vasculopath
Robert J. Rizzo, Anthony D. Whittemore, Gregory S. Couper, Magruder C. Donaldson, Sary F. Aranki, John J. Collins, Jr, John A. Mannick, and Lawrence H. Cohn, Boston, MA

69. Late Results With Operation in Patients With Ventricular Tachycardia and Poor Left Ventricular Function
Lynda Mickleborough, Shunichi Mizuno, Eugene Downar, and Gordon Gray, Toronto, Ontario, Canada

70. VAD-Supported Myocardial Revascularization: The Benefits of Avoiding Pump Oxygenation and Cardiac Arrest in Operations on Dilapidated Hearts
Michael S. Sweeney and O. Howard Frazier, Houston, TX

71. Coronary Artery Bypass Without Cardiopulmonary Bypass
Albert J. Pfister, Jorge M. Garcia, Luis A. Mispireta, Paul J. Corso, Anjum G. Qazi, Steven W. Boyce, Thomas R. Coughlin, and Patricia Gurny, Washington, DC, and Takoma Park, MD

February 4, 1992 (Tuesday morning)

7:00–8:00 PROBLEM CASE PRESENTATIONS
(5 Simultaneous Sessions)
Session Committee: Adnan Cobanoglu, Portland, OR, and John W. Hammon, Jr, Winston-Salem, NC

SEMINAR I
PULMONARY
Discussion Leader: Thomas W. Rice, Cleveland, OH

1. Chronic Pulmonary Torsion
   Paul F. Waters, Los Angeles, CA
2. Giant Pleural Fibroma Associated With Congestive Heart Failure
   Stephen Wallenhaupt and John W. Hammon, Jr, Winston-Salem, NC
3. Uncontrollable Chylothorax
   Donald D. Thomas, Colin Bailey, and Zwi Steiger, Detroit, MI

SEMINAR II
THORACIC TRAUMA
Discussion Leader: Kenneth L. Mattox, Houston, TX

4. Tracheoesophageal Fistula After Primary Repair of Tracheal Gunshot Wound
   James N. Heller, Paul J. P. Bolanowski, Robert J. Wilcott, and James S. Donahoo, Newark, NJ
5. Repair of Muscular Ventricular Septal Defect Secondary to Extrapericardial Trauma
   Richard J. Novick, Allan H. Menkis, G. Jablonsky, and F. Neil McKenzie, London, Ontario, Canada
6. Traumatic Main Bronchus Rupture Complicated by Pulmonary Coccidiomycosis
   Felix D. Battistella, David M. Follette, and Mervin B. O'Neil, Sacramento, CA

SEMINAR III
CONGENITAL
Discussion Leader: Richard A. Jonas, Boston, MA

7. Critical Pulmonic Stenosis in a Neonate
   Duke E. Cameron, Baltimore, MD
8. Acute Occlusion of Left Main Coronary Artery After Arterial Switch Repair
   Robert L. Fortune and Peter Baron, Phoenix, AZ
9. Management of Ectopia Cordis of the Thoracoabdominal Type
   Joseph J. Amato, Ralph J. Galdieri, David M. Lemonick, Hendrick B. Barner, and F. Z. Bierman, New Hyde Park, NY

SEMINAR IV
ESOPHAGUS
Discussion Leader: Herbert E. Cohn, Philadelphia, PA

10. Achalasia After Chemotherapy and Radiotherapy for Adenocarcinoma of the Esophagus
    Victor A. Ferraris, William R. Berry, Robert R. Klingman, and John Balint, Albany, NY
11. Anastomotic Stricture After Transhiatal Esophagectomy
    Samuel Edwin Carroll, London, Ontario, Canada
12. Management of Gastrobronchial Fistula 2 Years After Surgical Correction of Benign Esophagobronchial Fistula Secondary to Hiatal Hernia, Reflux Esophagitis, and Stricture
    Elliot Senderoff, Ambrish P. Mathur, Kedambady P. Sheka, and David Bryk, Brooklyn, NY

SEMINAR V
ADULT CARDIAC
Discussion Leader: Stanley K. Brockman, Philadelphia, PA

13. Intraoperative Aortoventricular Separation After Aortic Valve Replacement
    Tomas D. Martin, M. W. Connoly, and Joseph M. Craver, Atlanta, GA
14. Dynamic Left Ventricular Outflow Tract Obstruction After In Situ Mitral Valve Replacement
    Thomas F. Flavin, Richard E. Kerber, Allyn B. Mark, and Douglas M. Behrendt, Iowa City, IA

15. Repair of Left Ventricular to Right Atrial Fistula After Mitral Valve Re-Replacement Using Right Thoracotomy and Circulatory Arrest
Verdi J. DiSesa, Philadelphia, PA

February 4, 1992 (Tuesday morning)

7:00–8:00 TECHNICAL SESSIONS—HOW I DO IT
(5 Simultaneous Sessions)
Session Committee: Delos M. Cosgrove III, Cleveland, OH, and Douglas M. Mathisen, Boston, MA

SEMINAR I
THOROCOSCOPIC SURGERY
Mark S. Allen, Rochester, MN

SEMINAR II
LUNG TRANSPLANTATION—HARVESTING, PRESERVATION, IMPLANTATION
G. Alexander Patterson, St. Louis, MO

SEMINAR III
CONGENITAL ARTERIAL SWITCH
Jan M. Quaegebeur, New York, NY

SEMINAR IV
PULMONARY THROMBOENDARTERECTOMY
Pat O. Daily, San Diego, CA

SEMINAR V
TECHNICAL ASPECTS OF AORTIC VALVE REPAIR
Carlos G. Duran, Riyadh, Saudi Arabia

February 5, 1992 (Wednesday morning)

7:00–8:00 CLINICAL WORKSHOPS
(8 Simultaneous Sessions)
Session Committee: Timothy J. Gardner, Baltimore, MD, and Victor F. Trastek, Rochester, MN

WORKSHOP I
ACID IMPLANTATION
Levi Watkins, Jr, Baltimore, MD

WORKSHOP II
INTRAOPERATIVE ECHOCARDIOGRAPHY
Henry M. Spotnitz, New York, NY

WORKSHOP III
CARDIOMYOPLASTY
James A. Magovern, Pittsburgh, PA

WORKSHOP IV
CHOICE OF ARTERIAL CONDUITS
Hendrick B. Barner, New Hyde Park, NY

WORKSHOP V
MODIFICATIONS OF THE FORTAN PROCEDURE
Hillel Laks, Los Angeles, CA

WORKSHOP VI
BARRETT'S DISEASE OF THE ESOPHAGUS
Victor F. Trastek, Rochester, MN

WORKSHOP VII
NEW APPROACHES TO MANAGEMENT OT BULLOUS EMPHYSEMA
Bill Nelems, Vancouver, British Columbia, Canada

WORKSHOP VIII
NEW DEVELOPMENTS IN CODING FOR REIMBURSEMENT
Sidney Levitsky, Boston, MA, and Jeanne Fitzgerald, Ann Arbor, MI

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Volume 52 (July–December 1991)

KEY TO ABBREVIATIONS USED FOR ARTICLE CLASSIFICATION

* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence

CT = Collective Review
CV = Current Review
DI = Discussion
ED = Editorial
HW = How to Do It
IC = Invited Commentary

KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

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* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence
CT = Collective Review
CV = Current Review
ED = Editorial
HW = How to Do It
KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

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Thermodilution catheter-induced endobronchial hemorrhage with pulmonary hypertension (Ravichandran, Kelly, & others) 1991;52:1208,CP

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Vagal schwannoma involving esophagus (Vaghei & Yost) 1991;52:1334–6,CR

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Emergency esophagogastrectomy for treatment of hydrochloric acid injury (Horváth, Oláh, & others) 1991;52:98–101*

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Successful management of aortoesophageal fistula due to thoracic aortic aneurysm (von Oppell, de Groot, & others) 1991;52:1168–70,CR

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Cardiopulmonary bypass and thyroid function: a "euthyroid sick syndrome" (Holland, Brown, & others) 1991;52:46–50*

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Fatty acids suppress recovery of heart function after hypothermic perfusion (Mjøs, Ichihara, & others) 1991;52:965–70*

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Fibrin sealant improves surgical results of type A acute aortic dissections (Séguin, Frapier, & others) 1991;52:745–9*

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DNA ploidy patterns of tumors diagnosed as metachronous or recurrent lung cancers (Ichinose, Hara, & others) 1991;52:469–73*

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Furosemide

Diuresis in hemodynamically compromised patients (Honnekeri & Tendolkar) 1991;52:585,CP; Reply (Magovern) 1991;52:585,CP

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Gastric emptying after gastric pull-up (Casson, Powe, & others) 1991;52:1207–8,CP

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Use of the inferior epigastric artery as a free graft for myocardial revascularization (Barner, Naunheim, & others) 1991;52:429–37*

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Successful repair of a submitral left ventricular infected pseudoaneurysm (Zisbrod, Manjoney, & others) 1991;52:304–5,CR

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Treatment of cardiogenic shock with the Hemopump left ventricular assist device (Wampler, Frazier, & others) 1991;52:506–13*

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Bidirectional cavopulmonary anastomosis using vertical vein for right isomerism, pulmonary atresia, and TAPVR (Okita, Miki, & others) 1991;52:544–6,CR

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Acute postinfarction septal rupture: long-term results (Loisance, Lordez, & others) 1991;52:474–8*
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- Aprotinin used in emergency coronary operation after streptokinase treatment (Efstratiadis, Munsch, & others) 1991;52:1320–1,CR
- Preoperative hemostatic activity and excessive bleeding after cardiopulmonary bypass (Ratnatunga, Rees, & others) 1991;52:250–7*
- Thermodilution catheter-induced endobronchial hemorrhage with pulmonary hypertension (Ravichandran, Kelly, & others) 1991;52:1208,CP

Hemostasis
- Preoperative hemostatic activity and excessive bleeding after cardiopulmonary bypass (Ratnatunga, Rees, & others) 1991;52:250–7*

Hemostatometry
- Preoperative hemostatic activity and excessive bleeding after cardiopulmonary bypass (Ratnatunga, Rees, & others) 1991;52:250–7*

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- Peritoneopericardial diaphragmatic hernia discovered at coronary bypass operation (Kessler, Pett, & Wernly) 1991;52:562–3,CR

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- Surgical management of esophageal reflux and hiatal hernia (Hiebert) 1991;52:159–60,CL

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- Tuberculosis, the Adirondacks, and coming of age for thoracic surgery (Meyer) 1991;52:881–5,CL

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- Prevention of retrovirus infection after injury with contaminated instruments: an experimental study (Robicsek, Duncan, & others) 1991;52:74–7*

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- Cardiac retractor for coronary bypass operations (Rousou, Engelman, & others) 1991;52:877–8,HW
- Cosmetic approach for placement of the automatic implantable cardioverter-defibrillator in young women (Curiale, Rosenfeld, & Elefteriades) 1991;52:1340–1,HW
- Dacron mesh wrapping to support the aneurysmally dilated or friable ascending aorta (Milgalter & Laks) 1991;52:874–6,HW

  Doppler miniprobe to measure arterial graft flow in coronary artery bypass grafting (Takayama, Suma, & others) 1991; 52:322–4,HW
  Endarterectomy, vein patch, and mammary bypass of the anterior descending artery (Ladowski, Schatzlein, & others) 1991;52:1187–9,HW
  Improved forceps for pulmonary embolectomy (von Segesser, Bauer, & others) 1991;52:1185–6,HW
  Integrated pump switch for antegrade/retrograde cardioplegia (Loop, Foster, & others) 1991;52:320–1,HW
  Modified Robicsek technique for complicated sternal closure (Tavilla, van Son, & others) 1991;52:1179–80,HW
  Modified technique of internal mammary artery harvest (John, Edmondson, & Rees) 1991;52:157–8,HW
  Myocardium-sparing cannulation technique for left ventricular assist device support (Duncan, Baldwin, & others) 1991; 52:565–6,HW
  A new balanced operation for complex gastroesophageal reflux disease (Landreneau, Marshall, & others) 1991;52:325–7,HW
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  Preoperative duplex scan assessment of the inferior epigastric artery as a coronary bypass conduit (Milgalter, Laks, & others) 1991;52:567–8,HW
  Repair of the torn, friable right atrium after decannulation (St. Cyr, Fullerton, & Harken) 1991;52:1344–5,HW
  Replacement of the aortic valve with a pulmonary autograft: the switch operation (Ross) 1991;52:1346–50,HW
  Resternotomy in patients with valved conduits adherent to the sternum (DeLeon, Ilbawi, & others) 1991;52:569–71,HW
  Simplified placement of retrograde cardioplegic catheter (Bahn) 1991;52:879–80,HW
  Subclavian vein-to-right atrial bypass for symptomatic venous hypertension (Duncan, Baldwin, & others) 1991;52:1342–3,HW
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  Emergency esophagogastrectomy for treatment of hydrochloric acid injury (Horváth, Oláh, & others) 1991;52:98–101*

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  Comparative study of the hydrodynamic function of the CarboMedics valve (Butterfield, Fisher, & others) 1991;52:815–20*

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  Effect of pH shifts induced by oxygenating crystalloid cardioplegic solutions (Oppell, King, & others) 1991;52:903–7*

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  Subclavian vein-to-right atrial bypass for symptomatic venous hypertension (Duncan, Baldwin, & others) 1991;52:1342–3,HW
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  Myocardial protection in the hypertrophied right ventricle (Cusimano, Ashe, & others) 1991;52:934–8*

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  Perfusion without systemic heparinization for rewarming accidental hypothermia (von Segesser, Garcia, & Turina) 1991; 52:560–1,CR

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  Cardiopulmonary bypass, profound hypothermia, and circulatory arrest for neurosurgery (Williams, Rainer, & others) 1991;52:1069–75*
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  Fatty acids suppress recovery of heart function after hypothermic perfusion (Mjøs, Ichihara, & others) 1991;52:965–70*
  Not all neonatal hearts are equally protected from ischemic damage during hypothermia (Wittnich, Maitland, & others) 1991;52:1000–4*

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  Triiodothyronine-enhanced left ventricular function after ischemic injury (Dyke, Yeh, & others) 1991;52:14–9*

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  Cardiac transplantation with corticosteroid-free immunosuppression: long-term results (Lee, Pierce, & others) 1991;52:211–8*

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- Surgical management of resistant mycobacterial tuberculosis and other mycobacterial pulmonary infections (Pomerantz, Madsen, & others) 1991;52:1008–12*

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- Not all neonatal hearts are equally protected from ischemic damage during hypothermia (Wittnich, Maitland, & others) 1991;52:1000–4*
- Recovery of oxygen utilization efficiency after global myocardial ischemia (Furukawa, Kreiner, & others) 1991;52:1063–8*
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  Diaphragm paralysis following cardiac surgery: role of phrenic nerve cold injury (Efthimiou, Butler, & others) 1991;52:1005–8*
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To provide a place for ALL thoracic surgeons to relate experiences which will help other practicing thoracic surgeons give better patient care.
Herbert Sloan, MD

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EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

■ Twenty-eighth Annual Meeting of The Society of Thoracic Surgeons, Orlando, Florida—February 3–5, 1992

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

■ Second International Lung Transplant Symposium—Update 1991, Laguna Niguel, California—February 12–14, 1992

This symposium is sponsored by Cedars-Sinai Medical Center. For information on this meeting, contact Department of Continuing Medical Education, Cedars-Sinai Medical Center, at (213) 855-5547.

■ First International Symposium on Diseases of the Aorta, Tokyo, Japan—February 24–26, 1992

For information on this meeting, contact Japan Convention Services, Inc, Nippon Press Center Building, 2-1, 2-chome, Uchisaiwai-cho, Chiyoda-ku, Tokyo 100, Japan.

■ Pathophysiology & Techniques of Cardiopulmonary Bypass, San Diego, California—February 27–March 1, 1992

This meeting is the Twelfth Annual San Diego Cardiothoracic Surgery Symposium. For information on this meeting, contact the Cardiothoracic Research & Education Foundation, PO Box 23220, San Diego, CA 92193; or telephone (619) 541-1444 (facsimile: (619) 541-1447).

■ American College of Surgeons, Orlando, Florida—March 22–25, 1992

For information on this meeting, contact American College of Surgeons, 55 Erie St, Chicago, IL 60611; or telephone (312) 664-4050.

■ General Thoracic Surgery, Las Vegas, Nevada—April 2–3, 1992

This postgraduate course is sponsored by the American College of Chest Physicians and the University of Nevada School of Medicine. For information, contact ACCP, Division of Education, 3300 Dundee Rd, Northbrook, IL 60062; or telephone (708) 498-1400 (facsimile: (708) 498-5460).

■ Twelfth Annual Meeting of the International Society for Heart Transplantation, San Diego, California—April 2–4, 1992

For information on this meeting, contact ISHT, 435 N Michigan Ave, Suite 1717, Chicago, IL 60611; or telephone (312) 644-0828.

■ One Hundred Twelfth Annual Meeting of the American Surgical Association, Palm Desert, California—April 6–8, 1992

For information on this meeting, contact American Surgical Association, Department of Surgery, CB 7245, University of North Carolina, 136 Burnett-Womack Building, Chapel Hill, NC 27599; or telephone (919) 966-4320 (facsimile: (919) 966-6009).

■ Forty-first Annual Meeting of the American College of Cardiology, Dallas, Texas—April 12–16, 1992

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

■ Seventy-second Annual Meeting of the American Association for Thoracic Surgery, Los Angeles, California—April 27–29, 1992

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Thirty-eighth Annual Meeting of the American Society for Artificial Internal Organs, Nashville, Tennessee—May 7–9, 1992

For information on this meeting, contact ASAIO Registration, 2200 North Federal Highway, Suite 201, Boca Raton, FL 33431-7710.

■ Fourteenth World Congress of The International Society for Heart Research, Kobe, Japan—May 10–14, 1992

For information on this meeting, contact Makoto Nagano, MD, Secretariat, XIV World Congress of ISHR, Department of Internal Medicine, Jikei University Medical School, Aoto Hospital, 6-41-2 Aoto, Katsushika-ku, Tokyo 125, Japan.

■ The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Chicago, Illinois—June 8–10, 1992

For information on this meeting, contact the Association offices, 13

Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Cardiostim 92, Nice, France—June 17–20, 1992

For information on this meeting, contact Dr J. Mugica, Centre Chirurgical Val d'Or, 16, rue Pasteur, 92211 St-Cloud Cedex, France; or telephone (33) 1.46.02.70.72 (facsimile: (33) 1.46.02.05.09).

■ Eighteenth Annual Meeting of The Western Thoracic Surgical Association, Kauai, Hawaii—June 25–27, 1992

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

■ Sixth Annual Meeting of The European Association for Cardio-Thoracic Surgery, Geneva, Switzerland—September 14–16, 1992

For information on this meeting, contact Symporg S.A., 108, route de-Frontenex, CH-1208 Geneva, Switzerland; or telephone 41-22 786 37 44 (facsimile: 41-22 786 40 80).

■ Fifty-eighth Annual Scientific Assembly, American College of Chest Physicians, Chicago, Illinois—October 26–30, 1992

For information on this meeting, contact American College of Chest Physicians, 3300 Dundee Rd, Northbrook, IL 60062; or telephone (708) 498-1400 (facsimile: (708) 498-5460).

■ Thirty-ninth Annual Meeting of the Southern Thoracic Surgical Association, Wesley Chapel (near Tampa), Florida—November 5–7, 1992

For information on this meeting, contact Southern Thoracic Surgical Association, 401 N Michigan Ave, Chicago, IL 60611-4267; or telephone (312) 644-6610 (facsimile: (312) 321-6869).

■ Sixty-fifth Scientific Session of the American Heart Association, New Orleans, Louisiana—November 16–19, 1992

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 373-6300 (facsimile: (214) 373-3406).

CLASSIFIED ADVERTISEMENTS

Classified advertisements for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Art Carlucci, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010 (telephone: (212) 633-3806; facsimile: (212) 633-3820). All advertisements are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.50 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $18.00 per insertion for box number advertisements.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Advertisement orders should be for a maximum of 6 months; advertisements may be renewed for up to 6 months at a time. Advertisement copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery,* Desk Editorial, Classified Advertisements, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3853). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the advertisement. To respond to an anonymous advertisement, write to Box Number, *The Annals of Thoracic Surgery,* Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Possible academic affiliation. Please respond with CV.

Please respond to Box 404. 404I/C

Cardiothoracic and vascular surgeon, board eligible or certified, to join 2-man private practice in Ft. Lauderdale, Florida. Send CV.

Please respond to Box 542. 542L/E

Cardiovascular and thoracic surgeon, BE/BC, wanted to join an established expanding private practice in the Northeast with an academic affiliation. Must have experience in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership.

Please respond to Box 554. 554G/L

Cardiovascular surgeon, board certified or board eligible in thoracic surgery. Large surgical practice in western Pennsylvania. Excellent compensation and benefits. Please send CV and references.

Please respond to Box 555. 555G/L

University of Arizona. Expanding university program creates exciting opportunities for new faculty, both academic (tenure) and clinical tracks. Applicants must be interested in academic career. Interested particularly in cardiac, obstetric, ICU, and pediatric subspecialties. An AA/EOE.

Contact Burnell R. Brown, Jr, MD, PhD, Department of Anesthesiology, Arizona Health Sciences Center, Tucson, AZ 85724. Closing date: 03/31/92 or until position is filled. 560G/L

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join 2-man surgical group in the Southeast. 1–2 years of experience preferred. Please respond with curriculum vitae.

Please respond to Box 562. 562G/L

Cardiothoracic surgeon, recent graduate or no more than 2 years out of training, to join established group in a major Texas city. Practice includes adult and some pediatric cardiac, electrophysiologic, thoracic, and peripheral vascular surgery. Please respond with CV.

Please respond to Box 564. 564G/L

BC/BE cardiovascular and thoracic surgeon wanted to join busy two-member group in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308 [(305) 561-9781 after 9:00 PM]. 570H/A

Cardiovascular and thoracic surgeon to join established 3-man private practice in mid-South. Practice includes thoracic, cardiac, and vascular surgery. Must be board certified or eligible in thoracic and cardiovascular surgery. Prefer candidate who has recently completed his/her training. Please respond with curriculum vitae.

Please respond to Box 580. 580H/A

Cardiothoracic surgeon—metropolitan Kansas City area: An exceptional opportunity exists for BC/BE cardiothoracic surgeon in a large, expanding, independent multispecialty group. The practice is conveniently located close to hospitals, affordable luxury housing, and nationally recognized school systems. There is a strong, diversified mix of patients with an excellent referral base. A very attractive incentive package is available including a guaranteed income. Malpractice, marketing, and relocation assistance is also available.

Please send curriculum vitae to John Storm, 51 N 12th St, Kansas City, KS 66102; (913) 281-7775, FAX (913) 281-8494. 591I/L

Cardiovascular/thoracic surgeon: A progressive major regional multispecialty group in Texas Gulf Coast area is seeking a BC/BE cardiovascular /thoracic surgeon. Applicant should be technically strong in all phases of adult cardiac surgery. Cardiology department has three cardiologists. Practice is located in subtropical climate in an upward bound economic environment. Guaranteed salary with bonus plan. Shareholder status available 12–18 months. Malpractice insurance, interview, and relocation expenses included in benefits package. Location is attractive to those who enjoy outdoor sports such as fishing, golfing, and sailing. Look into our great team!

Contact Amanda Fuhro, Valley Diagnostic Medical and Surgical Clinic, PA, 2200 Haine Dr, Harlingen, TX 78550; (512) 421-5199. 592I/L

Thoracic-peripheral vascular surgeon: BC/BE to join busy 32-physician multispecialty group practice in central Florida. Competitive salary incentive and benefit package with early partnership. Five-hundred-bed hospital located less than ½ mile from office. Attractive central Florida lake country community is abundant in water sports, fishing, hunting, and outdoor sports. One and one-half hours to either coast.

Please respond to Box 600. 600J/A

Cardiothoracic surgeon, BE/BC, to join busy 1-man private practice in mid-Michigan. Excellent opportunity. Salary leading to partnership. Please send CV and references.

Please respond to Box 601. 601J/L

Thoracic and vascular surgeon, BE/BC, to join active (noncardiac) thoracic and vascular practice in Pittsburgh, PA. Please respond with curriculum vitae and details of experience.

Please respond to Box 602. 602J/L

Chairman, Cardiothoracic Surgery—excellent opportunity to lead cardiothoracic department for 400+-bed tertiary care teaching center located in western New York. Excellent program affiliated with prestigious medical school. Qualified candidate should possess academic credentials and should have interest in developing clinical research.

Please contact Jeanne Dowrick, Fulton, Longshore & Associates, Inc, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 604J/L

Cardiovascular and thoracic surgeon, BC/BE in thoracic surgery, to join active cardiac, thoracic, and vascular practice with academic affiliation in large Midwest city. Transplant experience desired. Reply with CV.

Please respond to Box 608. 608J/A

Cardiovascular and thoracic surgeon, board certified or board eligible, to join a rapidly expanding single hospital practice in the upper Midwest. Salary leading to partnership. Please respond with curriculum vitae.

Please respond to Box 609. 609J/C

Cardiothoracic surgeon—Northern California hospital is seeking an experienced full time cardiothoracic surgeon to direct and develop its new cardiac surgery program. Requirements include board eligibility or board certification and a minimum of 3 years' experience. This 250-bed, full-service facility anticipates 150 to 200 cases the first year.

Please respond to Box 610. 610J/C

Private practice opportunity in New York. Adult and pediatric cardiac surgery only. Postgraduate training in pediatrics a plus but will seriously consider all qualified applicants. Please respond with CV.

Please respond to Box 611. 611J/C

Cardiovascular/thoracic surgeon: Opportunity available for young surgeon to join two-man private practice doing cardiac, thoracic, pacemaker, and major vascular cases in Michigan. Early partnership. Excellent community and working conditions. Send CV and operative experience.

Please respond to Box 612. 612J/L

Cardiovascular thoracic surgeon—Board certified/board eligible and recently trained to join an established, busy, dynamic, and expanding private practice in northwest Indiana. The sphere of practice is approximately 30 minutes from downtown Chicago. Guaranteed excellent starting salary with progressive future growth and comprehensive corporate fringe benefit program.

Write including CV and details of experience and capabilities to PO Box 3066, Munster, IN 46321. 615J/L

Thoracic/vascular surgeon—Needed to join and expand active four-man group in desirable community in Ohio. (Emphasis on peripheral vascular, pulmonary, esophageal surgery and pacemakers.) Excellent opportunity for well-trained, highly motivated individual. Medical school affiliation possible. Please send curriculum vitae.

Please respond to Box 617. 617J/L

Assistant surgeon: Cardiac, thoracic, or general surgeon with cardiac experience to join well-established four-man group in the midwest. Ideal for physician who desires low-stress practice. Generous salary and fringe benefits. Please send curriculum vitae.

Please respond to Box 618. 618J/L

Cardiothoracic surgeon: Wanted to join a busy, well-established practice located in the midwest. Prefer recent graduate, must be board certified or eligible, medical school affiliation possible. Respond with curriculum vitae.

Please respond to Box 619. 619J/L

Excellent practice opportunity for well-mannered, technically accomplished CV surgeon in California wine country. Growing 2-surgeon practice in beautiful semirural area close to San Francisco—great family environment. Prefer young surgeon with 2–5 years' experience, ABTS board certified.

Please respond to Box 621. 621J/C

Thoracic surgeon needed to participate in growth of thriving cardiothoracic surgical practice located in greater New York metropolitan area. Excellent opportunity for well-trained, highly motivated surgeon who is board certified or eligible. Please submit CV.

Please respond to Box 629. 629J/L

General thoracic surgeon: The University of Rochester Division of Cardiovascular Surgery is seeking applicants for a full-time academic position in general thoracic surgery. Applicants must be BC/BE in cardiothoracic surgery. He or she will join the thoracic surgery section and must be devoted to the ongoing advancement of thoracic surgical clinical practice and academic scholarship. This position offers the individual committed to general thoracic surgery the opportunity to participate in the continued growth of academic thoracic surgical section in a comfortable and genuinely supportive environment. UR is an EEO/AA employer and represents an excellent environment for academic development.

Please respond and send curriculum vitae to Richard H. Feins, MD, Strong Memorial Hospital, 601 Elmwood Ave, Box Surgery, Rochester, NY 14642. 631K/A

Cardiovascular and thoracic surgeon: Position available for a cardiovascular and thoracic surgeon to join a 3-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association features competitive salary, excellent fringe benefits, and a goal of early partnership. Please send curriculum vitae.

Please respond to Box 634. 634K/A

Assistant Professor, Cardiac Surgery: The Section of Cardiac Surgery of UMDNJ-Robert Wood Johnson Medical School is seeking a highly qualified individual as an addition to the faculty at the level of Assistant Professor. This individual must have a keen interest in pursuing an academic career and must have demonstrated expertise in research.

Please send CV to Alan J. Spotnitz, MD, Department of Surgery, University of Medicine & Dentistry of New Jersey-Robert Wood Johnson Medical School, One Robert Wood Johnson Place, CN 19, New Brunswick, NJ 08903-0019. The UMDNJ is an affirmative action/equal employment opportunity employer, m/f/h/v, and a member of the University Health System of New Jersey. 637K/L

Cardiothoracic surgeon needed for expanding program in a great city on a great lake. BC/BE required. Respond with CV and references.

Please respond to Box 638. 638L

Cardiothoracic surgeon, board certified/eligible, wanted to join established practice in northwest Indiana (very near Chicago). Excellent salary. Please respond with curriculum vitae.

Please respond to Box 639. 639L/B

Assistant cardiac surgeon for an expanding program in its third year in the Tri-State area of Ohio, Kentucky, and West Virginia. The selected individual will be in charge of first assist in the operating room and share the responsibility of assessing the preoperative and postoperative cardiac surgical patient. The average load is 200 patients per year and increasing. The necessary qualifications include a valid WV license and board eligibility in thoracic surgery. Working experience in cardiac surgery preferred.

Please inquire at (304) 525-1414 or send CV to Tri-State Cardiac Surgery, Inc, 2828 First Ave, Suite 100, Huntington, WV 25702. 640L/B

Cardiac surgeon: recently trained, board eligible or certified cardiac surgeon interested in joining an expanding practice in the Tri-State area of Ohio, Kentucky, and West Virginia.

Please inquire at (304) 525-1414 or send CV and letters of recommendation to Tri-State Cardiac Surgery, Inc, 2828 First Ave, Suite 100, Huntington, WV 25702. 641L/B

Cardiovascular surgeon: excellent practice opportunity for recently trained BC/BE cardiovascular surgeon with initiative. Work to focus on cardiac surgery, but vascular coverage necessary. Midwest location. Salary/full partnership negotiable. Please send CV.

Please respond to Box 642. 642L/B

Cardiothoracic and vascular surgeon, board certified/board eligible, to join a 3-man practice in the Midwest. Salary leading to full partnership. Please send curriculum vitae.

Please respond to Box 643. 643L/B

Cardiothoracic surgeon, BE/BC, wanted to join established practice in mid-Michigan group of two. Ninety percent of practice is adult surgery and ten percent is general thoracic surgery. Respond with CV and names of references.

Please respond to Box 644. 644L/E

Busy cardiovascular group in the southeastern United States in a large metropolitan area seeks a new or recent graduate with comprehensive cardiovascular surgery experience. Salary leading to partnership. Please respond with curriculum vitae.

Please respond to Box 645. 645L

The Division of Thoracic and Cardiovascular Surgery at the University of New Mexico School of Medicine in Albuquerque is seeking applications for a full-time faculty position at the Assistant Professor level for July 1992. We are looking for an individual with excellent surgical skills, strong interest in research and teaching. The candidate must be board eligible or certified. We offer a competitive salary. This is an excellent opportunity for an individual interested in academic achievements as well as a good clinical experience. Please forward curriculum vitae and three letters of recommendation to Jorge A. Wernly, MD, Chief, Division of Thoracic and Cardiovascular Surgery, University of New Mexico Medical Center-ACC2, Albuquerque, NM 87131. An affirmative action/equal opportunity employer. 646L/C

Cardiothoracic surgeon urgently needed for reorganizing active practice in southern California. Superb working and living conditions. Excellent package leading to rapid partnership.

Please respond to Box 647. 647L/B

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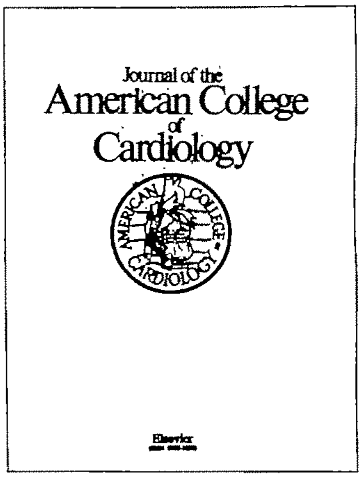

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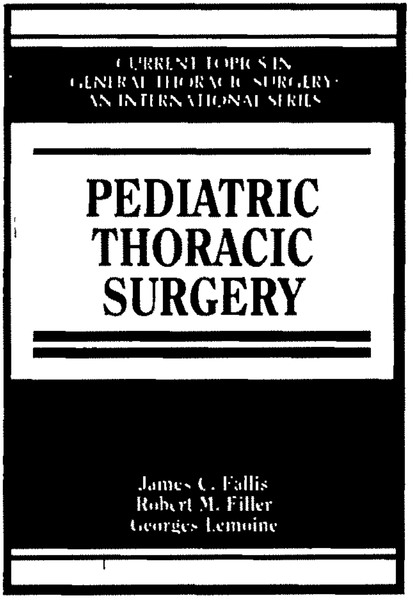
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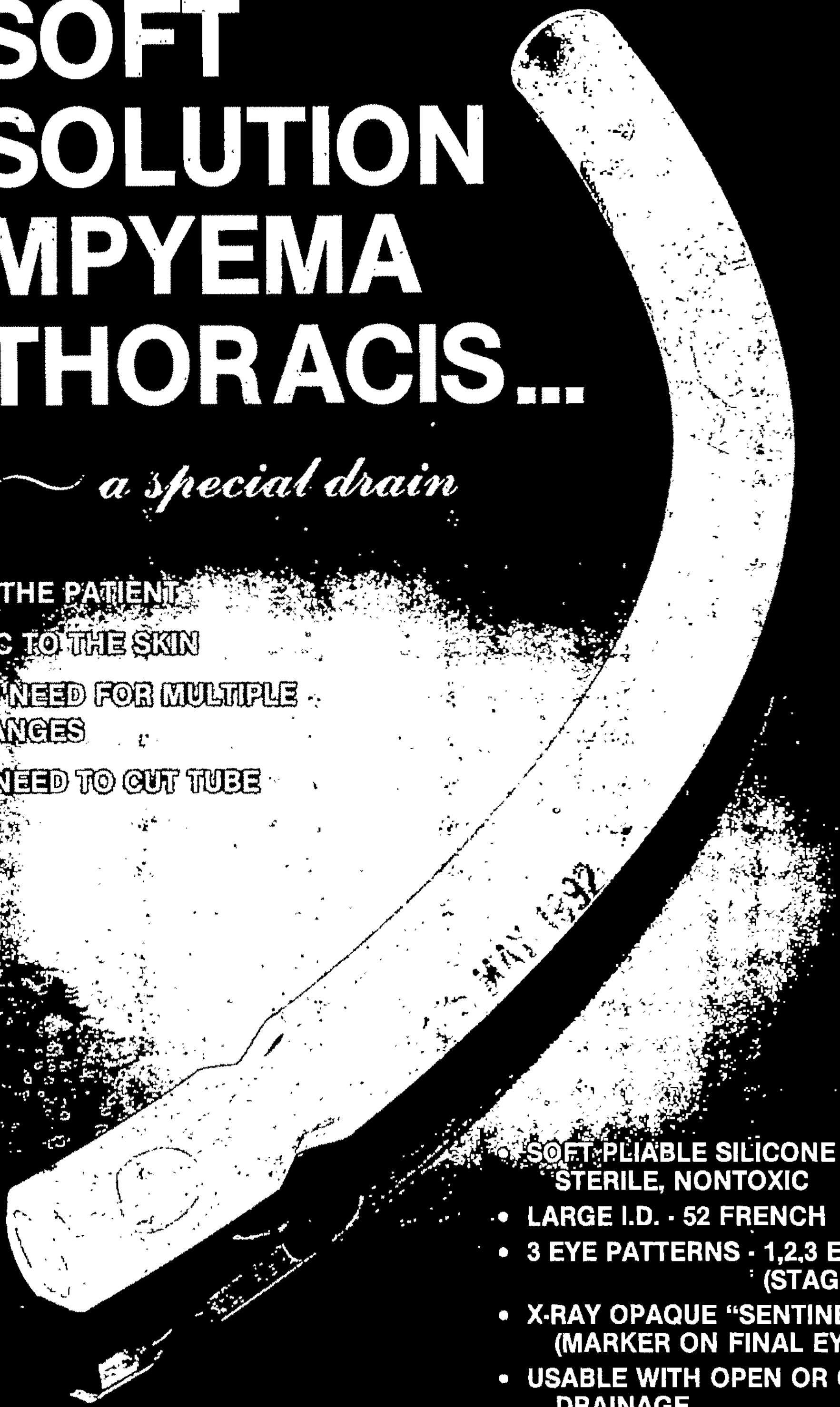

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